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THE

PRINCIPLES AND PRACTICE

OF

MEDICINE.

CHARLES HILTON FAGGE, M.D.

THE

PRINCIPLES AND PRACTICE

OF

MEDICINE.

BY THE LATE

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INCLUDING

A SECTION ON CUTANEOUS DISEASES,

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CHAPTERS ON CARDIAC DISEASES,

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AND

COMPLETE INDEXES,

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VOLUME I.

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PREFACE.

Soon after Dr. Fagge's lamented death, I was asked by his widow to complete and edit the Treatise on Medicine, which occupied his best energies for more than twelve years, and on which he was at work up to the last. I was also told that my late friend and colleague had himself expressed a desire that, if he should be unable to finish his book, its completion should be entrusted to me.

A request thus enforced could scarcely be refused, and, in accordance with the wish of the publishers, the manuscript was put into my hands about eighteen months ago. I was the more encouraged to undertake what I foresaw would be a difficult and laborious task, by my honored friend Dr. Wilks, who, with characteristic kindness, not only collected and looked through the scattered parcels of manuscript and ascertained what progress the author had made, but offered to help in the work by supplying the most important chapter of Diseases of the Heart—that on Affections of the Valves. The brief but masterly account which Dr. Wilks has given of this difficult branch of clinical medicine makes, in my judgment, one of the most valuable chapters of the book.

I was also so happy as to secure the intelligent and untiring help of Dr. Carrington, Assistant to Guy's Hospital, in seeing these sheets through the press. He has taken the greatest possible interest in its progress, and I have been continually indebted to his knowledge and care in verifying references and correcting errors. To him, also, is due the credit of the full and, I hope, useful indices.

The whole of the vast field of internal pathology and therapeutics had been traversed, and the account of it now before the reader had been written—much of it twice written—by Dr. Fagge, with two exceptions. He had left to the last the subject of Cardiac Diseases, to which he had

made such valuable contributions in the "*Guy's Hospital Reports*" and in Dr. Reynolds' "System of Medicine," and that of Diseases of the Skin, with which also he was perfectly familiar.

The part of the former subject which was unfinished when the pen dropped from his hand is, as I have said, well supplied by the friendly help of his old teacher, Dr. Wilks. The section on Cutaneous Diseases had not been begun, and this I have added, to the best of my ability, much regretting, for my own sake as well as for the profession generally, that we have not the results of my late colleague's large experience, sound judgment, and wide knowledge in what was once his favorite field of work—or rather one of them—for his interest in every branch of medicine was constant and almost universal.

Unfortunately, we could find no table of contents or other indication of the plan and arrangement intended by the author. Here and there occur indications of the sequence of subjects proposed, and at the beginning of some of the sections the order of the topics included under it is stated. But the general arrangement of sections and the sequence of most of the subordinate groups had to be determined without such help. In so doing I have aimed at convenience rather than symmetry, and have been more guided by the clinical than the pathological relations of the several subjects. I am not sure whether the author would have placed Syphilis among the specific diseases or not. I should myself have done so, and have put Tubercle beside it. But the latter subject must clearly, in this work, be placed close to Inflammation, and the former, therefore, holds an intermediate position. The order of the specific Fevers, the place assigned to such affections as Rheumatism and Rickets, and the grouping of the complicated series of Nervous diseases, must be always difficult to determine. In drawing up the contents of each chapter, the head-lines of the pages, and the titles of the subcapitular sections, I have, while following the lines indicated by the author, endeavored to make the order easy to follow and also to facilitate reference.

In revising the language employed, though I have been anxious to make as few alterations as possible, there is, I suppose, scarcely a paragraph left untouched. But the changes were almost entirely verbal, and consisted chiefly in breaking up long sentences with relative clauses, and in omitting frequently recurrent adverbs, the corrections which commonly suggest themselves in proof sheets. I have, however, left the conversational style, which it would have been impossible, if desirable, to change without more exten-

sive alteration than an editor is justified in making; and those who knew the writer will pardon an occasional colloquialism, if, as I hope, they will sometimes while they read seem to hear his low, rapid voice, and see his bright, intelligent glance, and note his characteristic attitude of suspense, his quiet smile of doubt, or his eager nod of assent.

Here and there throughout the work I have inserted or completed a reference which seemed likely to be useful. Whenever this was not suggested by the text, I have marked the addition by square brackets. In the first volume, almost all of which had, I believe, been revised by the author, the corrections and additions have been few. In the second, I have been obliged not only to add references to some of the most important papers which have appeared since the manuscript was written, but also here and there to supply foot-notes or paragraphs. I can only hope that the justification for each of these may be apparent to the reader. Every addition beyond mere references or obvious corrections is carefully distinguished as being made by the editor; and I have not thought it my duty, in editing so recent a work, to make any comment on the few points in which I might differ from the author.

Both Dr. Carrington and myself have taken much pains to secure accuracy in references and in names; but I fear that, notwithstanding all our care, we shall stand in need of the occasional indulgence of the reader. I should be much indebted to any one who would be so kind as to inform me, through the publishers, of misquotations or wrongly cited names. I hope that the references to hospital Reports, weekly Journals, and Transactions of societies, whether English or foreign, will be sufficient for the student; in most cases I have thought it enough to indicate the year or the volume where a glance at the contents would identify the article referred to. In the case of authors whose systematic writings are again and again quoted, as Watson, Walshe, Murchison, Virchow, Cohnheim, Trousseau, Charcot, and the writers in Reynolds' "System" and Ziemssen's "Cyclopædia," I have, in order to avoid repeated references after the first, put against the name in the index of authors the title of the work or works cited *passim* in the text.

I feel that some apology is due for the frequent quotation from the "*Guy's Hospital Reports*" and from other writings of those connected with that Medical School. I have repeatedly omitted the name where the omission did not sacrifice perspicuity. But in truth one of the great merits of this book is that, while paying, I trust, due respect to the many admirable publications of the last twenty years, both in this country and abroad, it is

essentially based upon experience gathered in the wards, the dead-house, and the out-patient rooms of one of the great London hospitals. If some of the eminent physicians connected with the sister schools of medicine in London, Edinburgh, or Dublin could be induced, like Dr. Bristowe and Dr. Frederick Roberts, to write a systematic treatise on medicine, and if they would put before the profession the invaluable traditions of pathological experience and of practical use which each great school possesses, I am persuaded that we should all wish them to write as frankly and unrestrainedly as did the author of these volumes.

P. H. PYE-SMITH.

HARLEY STREET;
September, 1885.

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THE PRINCIPLES AND PRACTICE OF MEDICINE.

MANY attempts have been made to frame a satisfactory definition of *disease;* but hitherto no better result has been arrived at than that it is the opposite of *health*, a derangement of structure or of function in some part of the body or in the whole of it, causing pain or discomfort, or tending to shorten life.

The art of medicine, however, is concerned, not with disease but with *diseases.* And it is of the first importance that we should form correct opinions upon what constitutes *a disease,* and upon what principle diseases should be defined and named.

From an early period in the present century, the utmost zeal and perseverance have been devoted to the search for morbid changes in the various organs and tissues of the dead body, and to tracing the relations between such changes after death and states of ill health during life. Numerous lesions have been thus discovered, the occurrence of which was altogether unknown to the physicians of former ages; while many of them have been found to betray their presence by signs which skilled observers can recognize without difficulty, even when the patient himself draws no attention to them, or is unaware that he is ill. On the other hand, there is a not inconsiderable number of disorders which force themselves into notice by the pain or discomfort they occasion, and which may in some cases actually destroy life; and yet after death all the organs and tissues seem to have their structure unaltered, even when they are examined with the highest powers of the microscope and by every chemical test that can be devised. At first sight it appears as though the distinction thus suggested must be a very important and fundamental one, between *organic* or *structural* diseases attended with recognizable morbid changes and *functional* diseases in which no such lesions can be found.

By many thinkers, however, the occurrence of functional diseases is altogether denied. They are of opinion that if our inquiries could be carried far enough we should discover some morbid changes to account for every disorder or complaint, even the slightest. They say that they "cannot conceive" how any of the vital processes should fail to be carried on naturally so long as every part of the body retains an absolutely normal structure; and they almost seem to suppose that no further argument is needed to establish their view. But, as is well known, the history of philosophy is full of lessons which show how fallacious it is to assume that things are impossible because when submitted to the scrutiny of our faculties they seem to be inconceivable. The question is not as to whether an effect can arise without a cause, but as to whether the cause must necessarily be of one particular kind because we know of no other kinds.

However, physiology suggests an analogy that may not be altogether without its application to pathology: the case of muscular fatigue. This was formerly supposed to be solely a result of the consumption of contractile material in the performance of work by the muscles. It is now known to be partly due to the accumulation in the tissues of those waste products which arise out of the contractile material so consumed. But the nerves and nerve-centres are also liable to exhaustion; and it seems not unlikely that the *neuroses*—as functional nerve disorders are called—may depend upon similar conditions. No doubt each of the factors which are concerned in the production of muscular fatigue can be conceived as within the range of our mental vision; the one appearing as a minute structural change, the other as a change of chemical composition. But it must still be important to maintain the distinction between diseases arising in such ways and those in which conspicuous lesions are present.

Unfortunately, however, the direction in which medical thought has tended during the last thirty years has been such as to lead writers to ignore functional diseases as far as possible, even apart from theoretical doubts as to their possible existence. Those who have been termed *par excellence* "pathologists"—whose studies have been devoted to the investigation of morbid changes in the dead body—have, from the time of Rokitansky, been disposed to maintain that such changes are really the diseases in the cases in which they are present, and that their effects, which are generally the most obvious phenomena in the living patient and which used themselves to be called diseases, are only "symptoms." Thus, whereas paralysis and dropsy, and jaundice, were once conspicuous members of the nosology, they are now almost banished from it. Sometimes, as in the case of apoplexy, the very name has been taken from a clinical condition, and transferred to the lesion which, according to modern investigations, is more or less constantly present. And to account for many of the slighter complaints to which mankind are liable, hypothetical lesions—hyperæmia and anæmia of a variety of organs—have been lavishly created on speculative grounds.

It seems to me that all this is as incorrect as it is inconvenient. The two principal objects of the art, and even of the science, of medicine are to prevent or to cure disease; and for those who alone wish to study medicine, the part of each disease which is of real importance is that which can be recognized during life. It is time to admit frankly that some of the reasons which have generally been assigned for the cultivation of morbid anatomy can no longer be justified. It used to be said that no one could properly treat a patient without understanding the nature of his disease; and when the microscope was first brought to bear upon pathology, it was in the hope that improved methods of cure would directly result from the discoveries that might thus be made, one line of practice being applicable if a morbid change was inflammatory, another if it were degenerative, and so on.

Now, within the last few years there has been much discussion as to the nature of tubercle, as to the part played by inflammation in pulmonary phthisis, and as to the degenerative or inflammatory character of arterial atheroma. But will any one maintain that our treatment of the patients in whom these several affections occur has been based to any appreciable extent upon our theoretical views with regard to them? It rather seems to me that while writers have been insisting on the dogma that pathology is the true basis of therapeutics, practitioners have been too much disposed to fall back upon an unexpressed notion that structural changes of whatever kind are but little likely to be interrupted in their progress by the drugs which we can administer, or by any other curative measures which we can adopt.

With the dogma itself I should, indeed, have little fault to find, if by "pathology" were to be understood, not merely the morbid anatomy of a disease, but the best attainable knowledge of its origin, course, and termination. In the latter sense the term has of late been seldom employed in England, but so I shall always endeavor to use it in the present work.

Morbid anatomy, however, may, in one sense, be said to be the true foundation upon which nearly the whole art of medicine, including therapeutics, should, as far as possible, be built. But the reason for this is a very different one from that suggested in the previous paragraphs. It is that until we have traced a disease to a definite organic lesion we can seldom be sure that cases which seem to us to be identical, may not, in fact, differ from one another in their nature; and that upon their real identity depends all certainty of knowledge as to their prevention or cure. Probably no structural change has ever been discovered without leading to a further subdivision of the corresponding disease, in consequence of there being cases more or less similar, in which the change in question is absent. Even in an affection so restricted in its scope as "bulbar paralysis" this result has not failed to follow, since the discovery was made that it frequently depends upon a primary atrophy of certain nerve nuclei.

The old writers on medicine have often been reproached for looking on diseases as entities rather than as morbid processes of which the living structures are the seats. But what can be more of an entity than a tissue affected with an organic lesion? And how could one go further in giving to a disease a substantive existence than by confining it to that which can be seen and handled, while one regards as its "attributes" or "symptoms" all its attendant vital phenomena? It seems to me that Dr. Reynolds and Dr. Bristowe are right when they maintain, in the introductory chapters of their respective works, that the real disease is not merely the organic lesion, but a "complex," or "sum total, of morbid changes in both function and structure."

I hold, therefore, that in constructing a Nosology, the main thing to be attended to is that each disease should represent a homogeneous group of cases, resembling one another in their principal features. It is not necessary that all diseases should be constituted alike; we have in *scabies* one of which the fundamental character is the presence of a special cause; in *aneurism* one in which it is a lesion; in *migraine* one in which it is a series of morbid sensations and disorders of function. Whenever the whole of a group of cases can be traced to a single structural change, this should doubtless be regarded as the most essential element in the definition of the corresponding disease, and it should, if possible, furnish the name. But if the effect of several lesions are identical, so that they are clinically undistinguishable from one another, it is often better to keep them united under a common designation. Such an arrangement may be regarded as merely provisional, but it is one which is likely to be needed for very many years to come. And for my own part, I should generally give to these comprehensive diseases the most prominent places in the list, and I should assign altogether subordinate positions to the affections included in them, notwithstanding that the latter are alone known to the morbid anatomist. It is, however, often necessary that the two sets of names should stand side by side.

The process by which we give a name to a case, or by which we determine to what disease it belongs, is one of analysis; and there is nothing surprising in the fact that in some cases we can carry this process further than in others. To say that a patient has "hepatic ascites" is far more

accurate than to speak of him as affected with "cirrhosis of the liver," if, after all, we are not certain that the lesion may not be peri-hepatitis, or cancer, or gummata. To define a cerebral case as one of "hemiplegia" is much better than to call it "softening of the brain," if the lesion is really but little less likely to be a clot of effused blood, or a tumor, or an abscess. And yet there are other cases in which we can be sure that cirrhosis of the liver or that softening of the brain is present; and it is obviously better to name such cases according to their respective lesions. It is just as though a chemist were sometimes unable to discern the ultimate elements of the substances submitted to him for analysis, and had to be contented with determining their proximate principles; and there is this further force in the comparison, that, for practical purposes, it is often far more important to know the "proximate" than the "ultimate" diagnosis.

Above all, I wish to protest against the statement so commonly made, that it is "incorrect to call hemiplegia, or ascites, or any like condition, a disease, *because* it is really only a symptom." I have already endeavored to point out that the phenomena which are commonly spoken of as symptoms are parts of the disease to which they belong, no less than the lesion, or the specific cause, or whatever else is taken as its main characteristic. It is true that, when they afford the means by which we infer the existence of a hidden morbid change, they almost necessarily bear to it the relation of effects to a cause, so that one then naturally comes to look upon them as subordinate, and of secondary importance; but this only shows that the term symptom is, in fact, a relative one. So long as we are unable, either during life or after death, to discover any cause for a set of clinical phenomena, they certainly are not symptoms, for they indicate nothing. There can be no symptom until the disease of which it is a symptom is discovered. As the process of analyzing a case, or a group of cases, advances, that which we call the disease is rightly changed at every step; and each "disease" becomes in its turn a "symptom" of the disease which succeeds it. I suppose that itching was once hardly distinguished from having the itch; afterwards, when the characters of the eruption of scabies were made out, itching was looked on as a symptom, and the eruption was the disease; now that the *sarcoptes* has been discovered, the eruption is itself reduced to being a symptom. If, in an important class of cases, the analysis is always arrested at a certain point, it is absurd to say that the ultimate fact in those cases, which cannot be made to reveal anything further as to their action, is "only a symptom." So far from hesitating to classify clinical phenomena as diseases, when we are unable to trace them to their causes, I only wish that we had a more complete supply of suitable names for them. It is very probable, as Dr. Moxon has argued in the "Guy's Hospital Reports" for 1870, that a good "clinical nomenclature" would be quickly followed by an immense advance in our knowledge of such conditions.

Moreover, even when we do understand the whole pathology of a case, and when the phenomena which characterize it clinically are fairly to be called symptoms, the distinction between them and the anatomical changes is often very uncertain and arbitrary. Thus, in acute tuberculosis, if with the ophthalmoscope one discovers a tubercle in the choroid membrane of the patient's eye, one is very likely to think of it as a symptom; but in reality the disease itself consists of nothing but a multitude of precisely similar tubercles. So, again, the enlarged liver and spleen of obstructive disease of the heart, the osseous nodes of syphilis, the swollen joints of acute rheumatism, each belong, in strictness, to the pathological anatomy of these several complaints. Nor, if we would be logical, can we stop even here; the same thing must be said of all cutaneous eruptions, and of all affections of mucous membranes, down to the rose-rash of enteric fever, or the scanty

sputum of spasmodic asthma. In that part of "Ziemssen's Cyclopædia" which is devoted to the exanthemata, the appearances seen on the skin are, in fact, described in some instances (but not in all) apart from the "symptoms," and under the head of "anatomical changes." But, for my own part, I prefer to employ the former term in its natural sense, even though I should be driven to admit that such a use of it is conventional. On the other hand, in the case of a functional disorder, to speak of the clinical phenomena as symptoms is really to reduce the disorder itself to an abstraction.

Thus, it appears to me that all the morbid changes, whether of structure or of function, which are recognizable during life, may properly be called symptoms, notwithstanding the logical objections to this proceeding.

Symptoms, so defined, naturally divide themselves into two distinct classes. Some of them are merely "subjective," or, in other words, they are appreciable by no one but the patient, so that our knowledge of them rests solely on his statements. Others are "objective;" that is, they can be directly observed by other persons. Indeed, they can be investigated, even though the patient himself is unconscious; or, being in the possession of his senses, he may be the only person who cannot perceive them.

Now, subjective symptoms are open to many sources of fallacy. They can often be feigned by one who desires to deceive, pretending to be ill when he is not really so. Moreover, and this is of far greater importance, their severity depends almost entirely upon the nervous susceptibility of the individual in whom they occur. A hearty laboring man will take no notice whatever of a pain which would drive a delicate lady to her bed for several weeks. In the former case, one must be as careful not to overlook the occurrence of disease, as in the latter case not to assume its presence without sufficient grounds. And yet it is essential that great and constant attention should be paid to subjective symptoms, for they often yield information which can be obtained in no other way.

The physician himself elicits objective symptoms by examining the patient's body with fingers, eyes and ears. But a few years ago he could, in this direction, do little more than feel the pulse and look at the tongue. Then came the discoveries of percussion and of auscultation, which gave to the diagnosis of diseases a precision that no one before had dreamt of. It was natural that those who introduced the new methods should endeavor to mark their value by giving a special name to the indications afforded by them. And thus it became usual to speak of "physical signs" as opposed to ordinary "symptoms." In reality, however, the distinction is untenable. As Dr. Reynolds remarks, heat of skin, an eruption on its surface, the wasting of a muscle, a change in any one of the secretions, are each of them "physical signs," no less than a cardiac murmur or a dull percussion-note. Equally unimportant is the division of symptoms into "general" and "local," upon which some writers have laid stress. And lastly, it is misleading to pick out a particular symptom as "pathognomonic," or as bearing so close a relation to the disease, that whereas all the rest of the symptoms afford only presumptive indications of its presence, this one proves it absolutely.

Our knowledge of the *causes* of diseases, or of their *ætiology*, is, as a rule, exceedingly fragmentary and imperfect. It may fairly be doubted whether, in the whole domain of pathology, any cause is to be found which invariably and under all circumstances produces a given effect. But in many instances there is little difficulty in perceiving that an illness, of whatever kind, has been preceded by some definite change in the outward

circumstances of the patient, or in the action of some vital organ. He may have been chilled by exposure to cold, he may have eaten unwholesome food, he may have been over-excited or over-fatigued. Either of these conditions is then called the "*exciting cause*" of the disease. But, on further consideration, it appears that people often go through weather no less inclement, indulge their appetites in precisely similar ways, have their mental and bodily powers strained to quite as great an extent, without being attacked by the same complaint, and, indeed, without their health being in any degree impaired. We now have no alternative but to assume that some other cause or causes has been in operation. This may act in either of two ways; it may combine with the exciting cause to produce the disease in the individual who falls ill, or its influence may be exerted upon those who remain well, counteracting and nullifying the operation of the exciting cause. If, for example, it is found that a complaint occurs only in persons at a certain time of life, this may either depend upon that particular age being favorable to its development, or upon other ages being unfavorable. Probably it sometimes happens that the one explanation is correct, sometimes that the other is; or both may apply in the same case. But in practice we generally assume that all causes of this kind have a positive instead of a negative action; and we are, therefore, accustomed to group them together under the name of "*predisposing causes.*"

It is, however, important to note that the conditions which are commonly included among the predisposing causes of diseases, do not always operate in either of the ways just mentioned. Reverting to the case already put, of a complaint affecting chiefly or solely persons at a particular age, we may, on consideration, find that this is merely due to the fact that those who are at that age are more exposed than others to the action of the exciting cause. The simplest illustration is, perhaps, afforded by the preponderance of certain ages among those who meet with injuries of various kinds. Thus, cutaneous scalds and burns are particularly frequent among young children; and scalds of the throat from drinking hot water out of the mouth of a kettle scarcely occur in persons who are grown up. On the other hand, young adults are much more apt to get their limbs broken than very old men. Such differences arise, not from a predisposing influence exerted by one period of life, nor even from a counteracting influence on the part of another period of life, but merely from the ignorance of infants as to the effects of heat, and from the fact that men at their prime are obliged to work under conditions which expose them to accidents. So, again, if general paralysis and locomotor ataxy, and many other diseases of the nervous centres, are more frequent in males than in females, the reason may be, not that one sex rather than the other predisposes to their occurrence, but that more men than women come under the operation of the exciting causes of these complaints, which are believed to be chiefly overwork, sexual exhaustion, overstrain of the mental faculties, and the like. All this is simple enough when we know what the exciting cause of a disease is; but if we are altogether ignorant of it, as is very often the case, our interpretation of the so-called predisposing causes must obviously be in the highest degree speculative and uncertain. And perhaps the most unsatisfactory of them all are those which are commonly included under the term "idiosyncrasy." It is well known, for example, that in certain persons nettlerash is invariably produced by strawberries, and that in other persons the odor of cats excites asthma. In such cases the exciting cause is obvious, and it is impossible to deny the title of predisposing causes to the conditions, whatever they may be, which lead to its operation in a particular individual and not in others. But as to the nature of these conditions we are altogether ignorant.

At first sight one might think it advisable to confine the term "predisposing cause" to those conditions which alone have logical claims to be so designated. Virchow many years ago made an attempt to introduce such a limitation, defining the "predisposition of an organ to disease as dependent upon a certain slight deviation from its normal structure, which causes its constituent parts to be too loosely held together." And a similar statement is upheld by Wagner. But a very little consideration will show that we shall scarcely ever be able to speak of predisposing causes at all if we adopt such a definition. For my own part, however, I am so sensible of the inaccuracy of the common use of this term that I shall, as far as possible, avoid making use of it in the present work.

It is in reference to the prevention of diseases that a knowledge of their ætiology is primarily of practical use, but indirectly it not infrequently has an important bearing upon their cure. I have already shown that the process of analyzing the clinical phenomena (or symptoms) of a case, which constitutes the diagnosis of it, is often arrested at a point short of our being able to trace it to a definite anatomical lesion; and, not infrequently, we are unable even to carry that process far enough to reach any solid basis on which to institute a sound treatment. Every circumstance in regard to possible exciting or predisposing causes of the patient's illness may then be of the greatest value in helping us to arrive at its correct interpretation.

But before we presume to attempt to modify the progress of a disease by medicinal or other treatment we have something to do beyond observing its symptoms, searching into its causes, and determining its place in the nosology. We have to ascertain what is its natural course when undisturbed by our art, so that we may know as certainly as possible whether the remedies which we use are really effectual or otherwise. It is this study that enables us to give what is termed a *prognosis* of the case to the patient himself, or at least to some near relative, a part of our duty which may be of the highest importance, and in the performance of which our credit and reputation are often at stake. In some cases, indeed, the prognosis is of such a character as almost to do away with the necessity of our rendering any further services to our patient. We may be able to declare that the disease will quickly and safely pass off, without tendency to relapse, and without risk of its leaving any ill effects behind it. A case in point is perhaps afforded by an attack of shingles in a child. Or we may have to say that the complaint is one which must inevitably prove rapidly fatal, in spite of every effort that can be made to arrest it. Such a verdict, for instance, must be given in many cases of apoplexy, and in hydrophobia. But, as a rule, our prognosis is applicable, not so much to the natural and undisturbed course of the disorder, as to the course which we believe it to be likely to take under the most judicious treatment that we can devise for the benefit of the patient.

One of the ways in which a disease may end is in *death*. Now, in the mode of dying, and in the phenomena which attend upon the last minutes or hours of life, there are certain varieties which require to be mentioned, and for the discussion of which the present is, perhaps, the most convenient occasion.

It was an ancient doctrine that different organs might in different cases be the *atria mortis*, which phrase may perhaps be fairly translated "the portals of death." But in modern times Bichat first endeavored to distinguish three modes of dying, at least so far as sudden deaths were concerned, beginning respectively by interruption of the circulation, of the respiration, or of the action of the brain. His views were based upon the

contrast which he drew between the "organic life" and the "animal life;" the heart and the lungs were regarded by him as bearing the same relation to the former as the brain to the latter. Now, it has long been recognized that the third of Bichat's modes of death—at least in the way in which he understood it—may be resolved into the other two. And yet, although writers point out that apoplectic and other comatose patients often die by "asphyxia," and perhaps sometimes by "syncope," they still continue to include *coma* among the modes of dying. But it seems to me that the logical way of dealing with the question is to describe as forms of death only those conditions which lead directly to the extinction of life, and which are themselves incapable of further analysis. Now, during sleep, and under other circumstances as well, the movements of the chest walls and those of the heart constitute the two great signs of life, all obvious indications of cerebral activity being in abeyance. We may, therefore, I think, reasonably suppose that if there are any fundamental differences in the phenomena which usher in death, the key to them should be found in the different order in which these two sets of movements successively cease. But, in Dr. Foster's "Text-book of Physiology," I find that little or no encouragement is given to the notion that any such distinctions can be drawn. Of the "respiratory centre" in the medulla oblongata, upon which the maintenance of the respiration depends, he says that "when this spot is excised or injured, breathing at once ceases, and since the inhibitory vagus centre is generally at the same time stimulated, and the heart's beat arrested, death ensues almost instantaneously." Hence this portion of the nervous system was styled by Flourens the "vital knot" or ganglion of life—*nœud vital*. Dr. Foster's statement appears to be derived directly from the results of experiments on animals, rather than from observations upon the human subject. But there can be no doubt that an equally complete and sudden extinction of all the vital actions occurs in persons who are killed by lightning, and in at least some of those who are shot dead, or who succumb to other violent and extensive injuries. Again, I am quite disposed to believe that in many cases in which the action of the heart is suddenly arrested, as, for example, where there is disease of that organ, or of the aorta, the respiratory centre is at the same instant paralyzed, so that both sets of movements cease together. In all probability the same thing occurs in some of those very rare instances in which a large quantity of blood effused into the interior of the cranial cavity destroys life with almost absolute suddenness.

But, whatever may be the case in the physiological laboratory, it is certain that in the wards of a hospital some cases occur in which the respiration stops while the heart is still beating; while there are others in which the cardiac pulsations are arrested before the thoracic muscles and the diaphragm cease to draw air into the lungs. Thus we can clearly recognize two principal modes of dying which are fundamentally distinct from one another. Each of the vital movements in question, however, is dependent upon the influence of nervous ganglia: the respiratory (as we have already seen), upon that of a centre in the medulla oblongata; the circulatory, upon that of a series of minute ganglia situated at the base of the heart. Thus, it is obviously possible for death by arrest of the breathing to be directly due to a morbid condition of the respiratory centre just referred to. At first sight, we seem now to be reintroducing Bichat's third form of death—that by interruption of the action of the brain. But a little consideration will show that this is not really the case. For the conditions under which a *sudden* paralysis of the respiratory centre occurs are not those of a cessation of the animal before that of the organic life, as Bichat supposed; nor does such paralysis, in fact, appear to bear a much closer relation to diseases of the brain than to those of the lungs and air passages. A *gradual* exhaus-

tion of the respiratory centre seems to be a necessary part of the process by which life is extinguished in all other cases in which the breathing ceases before the heart stops.

I. *Dying by the Lungs.*—This may, as I have just hinted, occur in two different ways; one in which the centre in the medulla oblongata is suddenly paralyzed, and another in which its vital activity becomes gradually, though perhaps rapidly, exhausted. The difference appears to me to be a most important one, since there is a complete contrast in the attendant phenomena.

(1) Death by stoppage of the breathing, dependent upon *sudden paralysis of the respiratory centre*, is often absolutely quiet. No doubt, if the person were sitting or standing up he would fall, and this would attract notice to the change in him. But, when lying in bed, he may draw his last breath without making the slightest noise, or moving a limb; and this, notwithstanding that he was awake and perfectly conscious a few moments before.

On two occasions, in visiting patients in consultation, I have found them in the act of dying in this way when I first entered the room. In neither case had the relatives the slightest idea that anything was amiss, although they were standing by the side of the bed or at its foot. What at once arrested our attention was, in each instance, the exceedingly flickering and feeble state of the pulse; and a careful scrutiny soon showed us that there were no respiratory movements. One circumstance which seems very remarkable, and the explanation of which is not apparent to me, is that there was no obvious lividity, or change in color of the face. We had hardly time to give a warning of what was impending before the pulse became imperceptible, and the beats of the heart very soon afterward ceased. There was no autopsy in either case; but the accounts which I received of the previous symptoms seemed to leave little doubt as to their nature. The first patient was the wife of a tradesman who had been suffering for a considerable time from severe pain in the head, and from other symptoms of cerebral tumor; she had been morose, and unwilling to be disturbed, so that her taking no notice of the visit of a stranger did not appear surprising. The other patient, an old lady, had been ill with bronchitis for some weeks, but not so as to excite much anxiety; she was supposed to be asleep, having said that she would like to rest for a quarter of an hour before the time appointed for the consultation; but she was absolutely unconscious, and the strongest liquor ammoniæ had not the slightest effect when brought to her nostrils.

I believe that these instances illustrate the two chief conditions under which sudden paralysis of the respiratory centre is met with in medical practice. Within the last few years several cases of tumor, or of abscess of the brain, have terminated in this way, suddenly and quite unexpectedly, at Guy's Hospital. I know of at least one instance in which the same thing would have happened during an epileptic fit, had it not been for the diligent maintenance of artificial respiration. And in cerebral hemorrhage I believe that this mode of death is not infrequent. On the other hand, it seems to occur in various diseases of the lungs and air passages. Three years ago, one of our students, a strong, healthy young man, devoted to football, was admitted into the Clinical Ward laboring under a severe attack of asthma, to which complaint he was subject. A few hours later, the house physician, who was sleeping in an adjoining room, was hastily summoned to him on account of an alarming failure of the breathing. The respirations became more and more shallow, and at last they ceased altogether; he fell forward in a state of insensibility, and remained unconscious for several minutes. Artificial respiration was at once resorted to; this was kept up

for a little while, and the patient recovered. I have no doubt that he would have died if no medical man had been at hand. It seems to me likely that when death occurs suddenly, from obstruction to the entrance of air into the larynx, it is due, not (as is generally supposed) to syncope, but to paralysis of the respiratory centre. It is well known, for example, that persons choked by the impaction of food in the fauces not infrequently die without any of the symptoms which would ordinarily be expected from such an accident; so that no suspicion of it arises until an autopsy is made. I am not sure whether those cases of pleurisy, or of phthisis, in which sudden death takes place should or should not be explained in the same way. A chronic disease, especially if it causes much wasting, may fairly be supposed to impair the nutrition of the heart, and in other ways to interfere with the regular maintenance of its movements. The question is one upon which direct observations are needed, as to whether the pulse or the breathing is the first to stop; or whether they both cease together. It is, of course, well known that many poisons kill by paralyzing the centre in the medulla oblongata: this is one of the chief ways in which death is apt to occur during the administration of chloroform and other anæsthetics.

(2) Death by stoppage of the breathing, dependent upon gradual (though often very rapid) exhaustion of the respiratory centre, has long been known under the name of *asphyxia*. Etymologically, indeed, this is incorrect, for the term properly means "pulselessness," whereas (as I have already explained) the heart goes on beating to the very last; but at the present day no misunderstanding can arise from the use of "asphyxia" in the usual sense, which has not only common acceptance, but the authority of many generations of writers, in its favor. Some years ago an attempt was made to substitute for it the name of *apnœa*. This, however, has never been widely adopted, and its employment is now to be discouraged as much as possible, since physiologists employ it to denote an exactly opposite condition, that which consists in an excessive supply of oxygen to the blood, whereby respiratory movements are for the time rendered unnecessary. We may, therefore, continue to describe as asphyxia the state which generally arises when a person is strangled, or smothered, or drowned, or when he is choked by a solid substance impacted in his throat. The primary effect of such an accident is, that the face and other parts of the surface assume a livid or purple color, in consequence of the altered condition of the blood in the systemic arteries, which becomes far darker and contains much more carbonic acid than the venous blood of health. The respiratory movements at once become more forcible and more frequent. Dr. Michael Foster sets forth how, in experiments on animals, it is very soon observed that the expiratory efforts are exaggerated out of all proportion to the inspiratory, and how they quickly pass into violent convulsions. During this period (which is found to occupy a little more than a minute), the blood pressure in the arteries rises very greatly and the cardiac pulsations are increased in frequency. At the end of it the breathing suddenly becomes deep and slow, the inspiratory movements being now the more marked of the two. The arterial pressure rapidly falls again, and the beats of the heart are reduced in number, although they still remain forcible. Gradually the rhythm of the respiratory movements ceases to be regular; long pauses occur, each separated by a series of quickly following inspirations, and each in its turn seeming as though it would be final. The inspirations themselves become more and more shallow, they have a gasping character, being accompanied by contractions of accessory muscles, especially of the face; at length the gasps spread into a convulsive stretching of the whole body; and with extended limbs, and a straightened trunk, with the head thrown back, the mouth widely open, the face drawn, and the nostrils dilated, the

last breath is taken in. In sudden asphyxia all this occurs within four or five minutes. The heart continues to beat for some seconds longer, its strokes at last rapidly failing in frequency and strength.

In experiments on animals in which it is possible to determine the state of the heart's chambers at the moment of death, they are seen to be all gorged with blood, the left as well as the right. If in ordinary autopsies in the human subject the left auricle and ventricle are found comparatively empty, while the right are distended, it is because the former unload themselves during the setting-in of rigor mortis. To Dr. Foster's account of the phenomena observed during life under such circumstances—which I have followed almost word for word—I may add the fact (in clinical practice a very important one) that when there is an actual obstruction to the entrance of air into the lungs the more yielding parts of the chest walls recede at every inspiration; thus the supra-sternal, supra-clavicular, and lower intercostal spaces, the præcordial region, even the inferior part of the sternum, and the lower ribs, may appear to be violently sucked inward. And after death the pleura may be found spotted with ecchymoses, which appear to result from the corresponding traction upon the sub-serous capillaries.

But asphyxia is by no means limited to cases in which a mechanical obstruction exists; it occurs in a scarcely less typical form when an animal or a human being is kept in a limited area into which no fresh air can enter, as, for instance, in the Black Hole of Calcutta. And a more or less chronic variety of it is seen in croup and in diseases of the larynx and trachea, as well as in bronchitis and other affections of the lungs, in which the air passages become blocked by accumulated secretions or by extravasated blood. The lividity of the skin and mucous membranes, and the recession of the yielding parts of the chest walls may be well marked in such cases. Convulsions are perhaps less commonly observed; but death ultimately occurs in precisely the same way, with a gradually increasing shallowness of the inspiratory movements. Again, in the case of apoplexy and of other cerebral diseases, asphyxia is often obviously the cause of death; fluid accumulates in the bronchial tubes, and at every breath the air bubbles through it. But under such circumstances, we can seldom be sure that a gradual paralysis of the respiratory centre (from the direct pressure of intracranial effusion) may not help in bringing about the cessation of the breathing, even if it be not the sole cause of it.

The timely performance of artificial respiration is sometimes effectual in saving the lives of persons in the act of dying by either of the two forms of stoppage of the breathing. Nor is it difficult to see what conditions are generally necessary to the success of this procedure. In cases of sudden paralysis of the respiratory centre, little can be expected from it, when the cause of the paralysis is an intracranial lesion. I think, however, that I remember one instance in which a patient with a cerebral tumor was in this way snatched from the grave for a time, although only to succumb to a recurrence of the same symptoms a few days later. Where the breathing is suddenly arrested in the course of an epileptic fit, or of an asthmatic seizure, artificial respiration appears to offer a good chance, as is shown by the case which I have narrated. And during the administration of chloroform it has often been as successful as could possibly be desired. In asphyxia, on the other hand, artificial respiration is useless unless the cause of the asphyxia can first be removed. If a man has been hanged or strangled, the throat must first be relieved from pressure; if he has been choked, one must first clear out the fauces and the entrance of the larynx, or open the trachea, so as to admit the entrance of air. If he has been drowned, the fluids in the bronchial tubes must first be allowed to drain away.

Thus in bronchitis and in other thoracic diseases, in which asphyxia gradually advances to a fatal termination, artificial respiration is seldom of any use.

Various methods of performing artificial respiration have been recommended by different observers. They all consist in the repetition, at regular intervals, about sixteen times in the minute, of alternate movements, by one of which air is driven out of the chest, while by the other it is sucked in. The oldest plan of all is simply to compress the thoracic walls and then to relax the pressure, the patient lying on his back. In 1856 Dr. Marshall Hall introduced what he termed the "postural" method. He first placed the body face downward, when its weight caused flattening of the chest and abdomen, and therefore expiration; afterwards he turned it over upon its side, when the elasticity of the ribs caused them to rise, and so led to a slight inspiration. One supposed advantage of the prone position was that the tongue fell forward against the teeth rather than backward over the entrance of the larynx; but as this was at the time when air was escaping from the air passages, and not when it was entering, the advantage was probably small. A drawback, obviously, was that only one side of the chest was likely to expand. Dr. Marshall Hall's method has therefore been generally abandoned in favor of one which was suggested by Dr. Henry Silvester, of Clapham, in the year 1857. According to his plan the patient is placed on his back with the shoulders raised and supported on a folded article of dress. The feet are fastened down or held by an assistant. The mouth and nostrils are then wiped, if necessary. The tongue is drawn forward, and kept projecting between the lips. One way of doing this is gently to raise the lower jaw so that the teeth hold the tongue forward. The operator now kneels at the patient's head, grasps his arms just above the elbows, and carries them upward and a little forward until they nearly meet, holding them there for two seconds; this action enlarges the chest, the ribs being drawn up by the pectoral and other muscles. Next he turns down the arms and presses them gently but firmly against the sides of the body. This diminishes the capacity of the chest, especially if the operator's hands are made to exert further pressure upon it. The Royal Humane Society soon began to recommend Dr. Silvester's plan, and it has since been generally adopted. But in 1877 Dr. Howard, of New York, proposed a third method, an account of which may be found in the *Lancet* for that year. His first step is to tear off the patient's clothing to the waist, and make of it a large, solid bolster. He next turns the body face downward, so that this bolster comes under the epigastrium. He then presses heavily upon the back for a few seconds, two or three times, so as to empty, as far as possible, both the lungs and the stomach. Next, the body is replaced on its back, the bolster beneath it making the epigastrium and the costal cartilages the highest points, while the shoulders and the occiput rest upon the ground at a lower level. The patient's wrists are now seized, crossed behind the head, and fixed there. His tongue is drawn forward with the thumb and forefinger (protected by the corner of a handkerchief), and it is held out of the right corner of the mouth. This part of the procedure may be confided to an assistant. The operator himself kneels astride the patient's hips. He places his two hands one on each side of the chest, the thumbs close to the xiphoid cartilage, the fingers over the lower intercostal spaces. He then slowly and steadily throws his whole weight forward until his face nearly touches that of the patient, and afterwards, by a final push, springs suddenly back into an erect but still kneeling posture. This procedure he repeats eight or ten times a minute. A point which Dr. Howard claims for his "direct method" (as he terms it) is that the entrance of the larynx is kept open by the backward

curvature of the patient's neck. There seems to be no doubt that it is far less fatiguing to the operator than Dr. Silvester's plan, and this is a very important consideration, inasmuch as artificial respiration has often to be persevered with for three or four hours at a time. It is surprising what a long interval will elapse, even in cases which recover, before a single spontaneous respiratory effort is discernible. Of course, where any such effort, however slight, is perceived great care should be taken not to let the artificial movements clash with it.

Whatever mode of artificial respiration may be adopted there is, of course, no reason why it should not be supplemented by the employment of warmth and friction to the limbs. The nostrils may also be excited by ammonia, and cold water may be dashed upon the face at intervals; but in cases that are really critical very little is to be hoped for from such means.

If a Faradic apparatus be at hand it may be often used with advantage. One mode of applying it is simply to stimulate the cutaneous nerves, placing, for example, one pole inside the cheek and another over the xiphoid cartilage, and allowing the current to pass without regard to the rhythm of the respiratory movements. I suppose, however, that this would probably fail if the respiratory centre were exhausted. Even then success may sometimes be attained by stimulating the phrenic nerves directly. The most exact method of doing this will be described elsewhere. But, for the purpose now in view, it is sufficient to use a large wetted sponge for each pole, and to put one on each side of the neck just outside the sterno-mastoid muscle. This brings some of the shoulder muscles also into play as well as the diaphragm, but that is no disadvantage. The essential thing is to stop and to reapply the current at regular intervals; so that each contraction may be followed by a pause, during which an assistant may compress the chest and the abdomen, so as to drive as much air as possible out of the lungs.

II. *Death by failure of the heart's action* is attended with different symptoms in different cases, according to the circumstances in which it occurs.

By some writers, including no less an authority than Sir Thomas Watson, the term *syncope* is employed as a general appellation for all forms of death beginning at the heart. And probably this is the best use to which the word can be put, though many instances might be found of its being taken with a narrower meaning, one nearly equivalent to fainting, of which an essential feature is stoppage of the organ in diastole.

(1) In some cases death by the heart is absolutely sudden, the patient falling flat upon the ground or sinking back in his chair. The respiration may perhaps cease at the same instant as the beats of the heart, or a few deep gasps occur, and then a kind of shiver runs through the frame, pallor spreads over the surface, and all is over. It is often impossible in such cases to determine whether paralysis of the heart or spasm is the cause of the cessation of its movements. If an autopsy is made, its chambers may be found either relaxed or contracted, either empty or more or less full of blood. But it is difficult to state how far its state may be modified by post-mortem contraction of the ventricular walls. If they are flabby, while the muscles generally are in a state of rigor mortis, it seems fair to conclude at the time when life became extinct the heart stopped in a condition of diastole. On the other hand, stoppage in systole is not necessarily due to spasm; it may be the result of pressure upon the outside of the heart, as when the pericardial sac becomes filled with blood by rupture of the aorta. An appearance which is, so far as I know, never seen when death begins at the heart, is an overloading of the right cavities with blood while the left are empty, as in cases of asphyxia.

The causes of sudden cessation of the cardiac movements are various. There may be disease of the heart itself, or of its valves, or of the base of the aorta; and in many cases of this kind it is probable that lesions of the nervous ganglia situated near the base of the organ might be discovered if carefully sought for. Or the functions of the heart may be arrested by affections of distant parts of the body. Upon such cases a flood of light has been thrown by modern physiological researches. It has been found that in animals stimulation of one of the *nervi vagi*, if sufficiently powerful, almost instantly arrests the cardiac beats; a similar effect follows irritation of a spot in the medulla oblongata, which, therefore, is called the "cardio-inhibitory centre;" and, lastly, it can be produced as a reflex effect by a violent impression upon peripheral nerves (as by suddenly crushing the foot), by comparatively slight irritation of the mesenteric nerves (in the frog, by sharply striking the exposed intestine with the handle of a scalpel), or even by gentle stimulation of them when peritonitis has first been set up.

In human pathology, all these varieties of inhibition seem to occur more or less frequently. That of which in medical practice we know least is perhaps direct inhibition by stimulation of the vagus. Czermak, however, was able, at will, to stop the beating of his own heart, by pressing the trunk of the pneumogastric nerve against a small osseous tumor in the neck. In cases of aneurism of the aorta it is not uncommon for sudden death to occur without the autopsy revealing a rupture of the sac or any other definite change; in such cases the fibres of the left vagus nerve are commonly spread out over and inseparably blended with the walls of the aneurism; and it seems very likely that this ought to be regarded as the cause of the arrest of the cardiac pulsations. Inhibition of the heart by stimulation of the centre in the medulla oblongata probably occurs when fainting is caused by emotion. Whether intracranial diseases ever destroy life in this way is perhaps doubtful; at least I am not aware of any observations in which it has been shown that the heart's beat ceased before the respiratory movements. On the other hand, death by reflex inhibition appears to be of frequent occurrence. There can be little doubt that the "fatal shock" which accompanies severe injuries of the limbs or of any part of the body is of this nature. But what is perhaps most striking is that abdominal diseases are specially apt to be attended with sudden death, just as we have seen that in experiments on animals the cardiac beats are so easily arrested by impressions on the mesenteric nerves. It is not necessary to appeal to the oft-cited cases in which persons have been killed instantaneously by blows on the epigastrium. In the collapse of acute peritonitis life is often suddenly extinguished, as I shall hereafter have occasion to point out. Whether diseases of the lung ever produce sudden death in the same manner I do not know.

(2) In other cases death by failure of the heart's action is more gradual; minutes or even hours pass before life becomes finally extinct. The symptoms may then vary according to circumstances, so that two or three different forms require description.

Sometimes the fatal termination occurs by fainting, for which the ancient designations were *deliquium animi* and *lipothymia*. The patient experiences distressing sensations of giddiness, nausea, and sinking at the epigastrium. He turns cold and pale, and perhaps breaks out in a clammy sweat. His sight becomes dim, and everything may appear black to him. He hears rushing noises in the ears. More or less rapidly he becomes completely insensible. His pulse is rapid and very weak, and it soon ceases to be felt at the wrist, though it may still for a time be counted in the carotids. The heart's impulse grows more and more feeble, until it may be no longer perceptible. Still, however, one may be able to hear with the stethoscope that

the organ continues to beat, though the sounds are very feeble, and the second one is audible only at the base. The respiration, in the meantime, becomes infrequent, irregular, and shallow. The pupils are dilated. Sometimes there is an involuntary discharge of urine and of fæces. When the heart's beats cease altogether, it may be considered that death has actually occurred. At the autopsy its walls are found relaxed, and its chambers may be more or less full of blood.

(3) Somewhat different in its phenomena is that form of death which is due to *hemorrhage*. There is then a waxy pallor of the face and lips, of the hands and finger-nails, and, indeed, of the surface of the body generally, which is more extreme than that accompanying mere fainting. Consciousness is, perhaps, more gradually lost, though much depends upon the rapidity of the bleeding. Delirium is often present, and in many cases epileptiform convulsions are developed. Sometimes the patient lapses from time to time into a state of insensibility, regaining consciousness in the intervals; it is then generally said that "fainting fits" occur. They are especially apt to arise if the patient attempts to sit up, and at the same time the pulse at the wrist becomes much more feeble than before, or is even temporarily imperceptible. After death from hemorrhage the heart is found contracted and empty.

The physician sees this form of death in cases of hæmatemesis or hæmoptysis, or ruptured aneurism, as well as in others. Sometimes when the giving way of a large vessel is the direct cause of the cessation of the heart's action, there is no external bleeding at all; the blood is extravasated into some large space within the body, such as the peritoneal cavity, or one of the pleural sacs, or even the alimentary canal. But I am not sure that these can be cited as examples of death by hemorrhage, for the symptoms seem to be seldom altogether typical.

(4) In marked contrast with the forms of death hitherto described, although also beginning with failure of the heart, is that which is called a state of *collapse*. This form, like the others, is attended with an extreme feebleness of the heart's action, the pulse at the wrist becoming imperceptible, and the cardiac sounds being heard with difficulty, even through the stethoscope. The surface of the body, and especially of the hands and feet, becomes cold and deathlike. The features are sunken, the eyes retracted in their sockets, and the orbits surrounded by deep brown rings. Yet the patient is often entirely free from all subjective sensations of giddiness, nausea, or faintness. He may still have a fair amount of muscular power, may get out of bed, and even walk for some distance. His consciousness is often retained up to the last moment; death, when it does occur, being absolutely sudden.

I am not aware that any satisfactory explanation has been given of the remarkable differences between the two conditions of fainting and collapse. The retention of the functions of the brain and even of the muscles, in the latter state, would seem to imply that these organs continue to receive a certain amount of arterial blood. Is it possible that the feeble stream which still flows from the heart, until its beats finally cease, is mainly or even entirely directed to the deeper parts, by an adjustment of the vasomotor apparatus, while the flow through the various superficial arteries is cut off?

Either fainting or collapse may result from affections of the abdominal organs, and are then probably due to reflex inhibition from the solar plexus, as in the cases to which I have already alluded, of sudden death from blows upon the epigastrium. But either of them may also be due to other causes; collapse, for example, occurs in certain cases of diphtheria, and it may also be produced by pulmonary embolism. If the analogy with the inhibitory

phenomena observed in the physiological laboratory can be looked upon as tolerably complete, it is an interesting question whether in any circumstances the supervention of collapse or fainting can be prevented by the injection of atropine, which in animals is to completely annul the normal inhibitory action of the vagus.

In many cases of death by cardiac failure life is extinct before aid can reach the patient. But sometimes the administration of a full dose of brandy or of ether by the mouth is effectual in arresting the danger; and the hypodermic injection of these remedies, when the syringe is at hand, is still more likely to be successful. Our house physicians frequently adopt this practice, and in many cases with marked success, in stimulating the action of the heart, although the result is too often temporary. Another method, advanced by Dr. J. C. Reid ("*Brit. Med. Journ.*," 1880, Vol. ii, p. 1014), is that of pouring hot, but not scalding, water over the præcordial region. He cites a case in point that occurred in an old man of seventy, who was thus restored from a condition of apparent death, and who lived for many years afterward. It does not appear very clear whether galvanism can be used with advantage in such circumstances. Ziemssen recently found ("*Deutsch. Arch.*," xxx, 1881) in a patient whose chest wall was deficient, so that the heart was covered only by the skin, that the heart's beats could be accelerated by powerful currents; Erb recommends that in galvanizing the heart large electrodes should be used, one being applied over the surface of the organ, the other over the dorsal vertebræ; a current of the highest intensity should then be passed, its direction being reversed seventy or eighty times in the minute. Previous observers, as cited by Walshe, had found that with strong currents there was a risk of inhibiting the cardiac contractions instead of stimulating them. And although it was admitted that, if the irritability of the organ were lowered, a powerful current, which, under ordinary circumstances, might have arrested its action, would, perhaps, have the effect of reviving, there yet seems to be need of caution, so that it would be advisable first to make trial of a feeble current, and afterward to increase its strength. But in the cases now under consideration there is seldom time for such a method of proceeding.

III. If now we turn our attention to the phenomena which accompany the *gradual* fading away of existence under the influence of some wasting disease, and endeavor to analyze them, we find that although this also is commonly spoken of as death by *asthenia*, we can hardly identify satisfactorily the characters of that mode of dying, any more than we can do so in a person who succumbs to mere old age. It is to be observed, however, that very few cases occur in which such diseases really run on to what appears to be their natural termination. If we take, for example, a series of cases of cancer of the œsophagus, or of any non-vital organ, and if we critically investigate them, we find that in a very large majority of them it is some accidental complication which actually destroys life. Still there are some few exceptions to this rule, and in these exceptions all the vital functions are brought to so low an ebb before any one of them is absolutely arrested, that to determine the order in which their ultimate extinction takes place is superfluous, even if it be not impossible. Slowly, and week by week, the sick man becomes more and more indifferent to those about him. His memory, his power of attention, all his mental faculties, gradually fail him. He passes days and nights alike in a dreamy dose or stupor. His special senses one by one disappear; smell and taste seem to go first; then he loses his sight, so that everything appears dark to him; last of all he becomes unable to hear. The sensibility of the skin is often greatly diminished; flies crawl unnoticed over the face and hands, so that these parts must be covered with gauze; and the conjunctivæ may lose their sensibility to impres-

sions to such an extent that the dull, lustreless corneæ become dry between the motionless, half-opened eyelids. The pupils are generally contracted, but before death they are said to become dilated. The eyes can no longer be brought to bear upon an object, but remain fixed, with their axes parallel. The muscular weakness is extreme; the body sinks down in the bed, the head cannot be raised from the pillow, the limbs lie powerless in any position into which they may happen to fall. The organic functions are reduced to the lowest possible point. Wagner, to whom I am indebted for several of the details above mentioned, speaks of fluids as falling down into the stomach through the paralyzed œsophagus with a splashing noise; but for many days before death it often happens that nothing whatever is swallowed, only from time to time a feather moistened with a little weak brandy-and-water is introduced a little way between the lips. The evacuations, both of urine and fæces, are passed into the sheets without exciting any consciousness. The temperature of the body usually falls, until it reaches several degrees below normal, the hands and the feet become obviously cold; sometimes, when the stupor is not very marked, and the advance of death not very slow, the patient himself feels the cold creeping up from his limbs toward his trunk. The respiration becomes infrequent, irregular, and shallow. Toward the last the movements of the chest walls may be so slight that it is scarcely possible to perceive them; one way of ascertaining whether they still go on, is to hold before the mouth a looking-glass, the cold surface of which becomes dimmed by the moisture of the breath. But sometimes the expiratory efforts are deep and sighing; they may then be separated from one another by intervals of a minute, or even longer, so that those who are watching by the bedside keep expecting each one of them in turn to be the last. Long before this it has become impossible to detect the pulse at the wrist, which for many days may have been small and rapid and flickering. At length, even with the stethoscope, no sign of the beating of the heart can be discovered, and then, often not without some hesitation, one pronounces the verdict that all is over.

In most cases, there is no difficulty in determining the exact moment at which death occurs. But sometimes it cannot be fixed with certainty, and there are some altogether exceptional instances (though I have never myself met with one) in which, for hours, or even for days, it remains uncertain whether life is extinct or merely suspended. I believe that the only sign of death which is both certain to manifest itself in the course of a few days, and also absolutely conclusive and infallible, is the occurrence of putrefaction, which is generally first indicated by discoloration of the surface of the abdomen. And in any case admitting of doubt, the coffin should not be closed until this has shown itself. But rigor mortis, chilling of the body, the cadaveric odor, the loss of transparency or flattening of the cornea, livid staining of the dependent parts of the body—each of these signs, when present, and when developed in the usual way after suspension of all obvious vital phenomena, may be taken as proofs that life is really at an end. I do not think that there is any foundation for the strong fear which many persons entertain of being buried alive, after supposed death. The cases really requiring caution are some very few instances of persons found in the streets, or losing consciousness unexpectedly and in unusual circumstances. Dr. Walshe says, that the only serious difficulty lies in diagnosing what he terms "prolonged syncopal trance," but of this affection he gives no detailed description.

We still have an imperfect knowledge of the nature of many of the morbid changes to which the human body is liable; and no doubt this

explains the fact that the current classifications of the different forms of hypertrophy, atrophy and degeneration are unsatisfactory, and to a great extent artificial. Since most of these affections are limited to particular structures, they will be best discussed hereafter, when we come to them in dealing with the diseases of the several organs. But there are other pathological processes, which are met with almost everywhere, and which almost form the basis of the whole science of medicine. Such are Inflammation, Tuberculosis, and certain kinds of New Growth. To these I must necessarily devote preliminary chapters; but it will be convenient to deal first with the closely allied subjects of Contagion and Pyrexia.

CONTAGION.

SPECIFIC DISEASES—CONTAGION AND MIASM—THEORY OF INFECTION—CONTAGIUM VIVUM—MICROZYMES—THEIR ORIGIN AND LIFE—IMMUNITY FROM CONTAGION—QUARANTINE—PROTECTIVE INOCULATION—MODES OF TRANSFERENCE—MIASMATA AND CONTAGIA—THEORY AND PRACTICE OF DISINFECTION.

Probably there is no medical man who does not, from time to time, apply the epithet *specific* to certain diseases, yet, as is the case with so many other terms that are habitually employed, there is no little difficulty in defining its meaning, and in determining what diseases should be classed as specific, and on what grounds. Most writers seem to be of opinion that their characters and their course differ from those of diseases which, by way of contrast, may be called "simple." Thus, Sir James Paget lays stress on the statement that "each specific disease constantly observes a certain phase or construction in its morbid processes." According to Liebermeister, the fundamental point is that among specific diseases the affection produced by a morbific cause is always of a particular kind. He instances the fact that a chill may in one person give rise to a common cold, in another to a bronchial catarrh, in a third to diarrhœa, in a fourth to toothache, and in yet other persons to a variety of "rheumatic" complaints; whereas, he says, the poison of syphilis produces only syphilis, that of measles only measles, that of smallpox only smallpox, and so forth. But I think that we shall hereafter find that there is no disease whatever of which the phenomena do not in certain cases vary so widely as to compel us to base our belief in their specificity entirely upon our knowledge of their etiology. And it seems to me that the only logical way is to frame our conceptions in accordance with this fact, unless, indeed, we give up the use of the epithet altogether.

I would, therefore, propose to define as "specific," *those diseases which have a definite cause, arising from without but acting within the body, distinct from the causes of all other diseases.*

The clause "acting within the body" is introduced for the purpose of excluding wounds and injuries and external cold, which set up "simple" or "common" inflammation. It may be also taken as excluding injuries inflicted upon the mucous surface of the alimentary tract, whether by caustic poisons or intestinal worms, since this is, for physiological purposes, outside the body. But lead poisoning and trichiniasis should certainly be regarded as specific diseases, and so, I think, should the affections caused by animal or vegetable parasites which penetrate into the substance of the skin, such as the *Sarcoptes scabiei* and the fungus of ringworm.

The difference of opinion as to the infective character of tubercular affections involves the question of their being specific or otherwise. Malignant and other tumors are, I think, certainly not specific. In reference to them and to some other diseases, a useful test of specificity is that of the existence of transitional forms, connecting one affection with another. Between simple morbid processes transitional forms are very frequent; in the case of specific diseases I do not think that they ever occur. It is true that Dr. Braxton Hicks brought before the Obstetrical Society, in 1870, a large series of observations, showing that when parturient women are exposed to the poison of scarlet fever, the resulting disease is often not attended with either rash or obvious sore

throat, and has rather the characters of a common puerperal septicæmia. But, as he himself pointed out, it may be that the scarlatinal virus disturbs the processes which go on after parturition in the bruised and ecchymosed uterus, and causes local inflammatory changes, and therefore that these, rather than the scarlatinal virus itself, give origin to the septicæmia. This view has since been powerfully supported by Mr. Howse, in the "Guy's Hospital Reports" for 1879; he finds that when scarlatina affects a surgical case after operation, septicæmic symptoms are not developed if antiseptic treatment is being carried out, and if the occurrence of putrefaction in the wound is prevented.

I have defined the causes of specific diseases as arising from without, although they act within the body. Now, there are two (or perhaps three) groups of specific diseases, of which the etiological relations require to be carefully studied. They are as follows:—

(1) *Miasmatic Diseases.*—These are caused by a morbific agency, which is derived from the soil, or from the water, or from the air of a place, altogether independently of the occurrence of illness of a similar kind in any other individual. Ague is the chief of them, but another miasmatic disease is influenza.

(2) *Contagious Diseases.*—Each of these maladies owes its origin, in most cases, if not in all, to a *virus* derived from a person already suffering from it, or sometimes from one of the lower animals. Among these are the exanthemata, typhus, the plague, mumps, whooping cough and glanders.

(3) *Miasmatic-contagious Diseases.*—This group, which is acknowledged by all German observers, is little, if at all, recognized in England; it therefore needs to be discussed somewhat fully. The epithet "miasmatic-contagious" was, I believe, first proposed by Henle in 1840. He, however, only had in his mind the circumstance that smallpox, scarlet fever, typhus and other maladies often seem, at first, to break out epidemically, as though they might have been caused by a miasma, whereas, afterwards the individual cases afford clear evidence of contagion. The question whether the exanthemata or any other contagious diseases do, as a matter of fact, ever arise independently of previous cases, is one with which I shall hereafter have to deal. But Liebermeister and other recent writers have assigned quite a fresh meaning to the term "miasmatic-contagious," and have limited its application to a special class of maladies. The typical examples are enteric fever and cholera, diseases which are undoubtedly spread by human intercourse, and which are usually, if not always, traceable to previous cases, but which are, nevertheless, believed not to be directly communicable from one sick person to another. Liebermeister's conception with regard to them is, briefly, that patients give off a contagion which is inoperative until it has passed through further stages of development outside the human body, which may be taken as converting it into a kind of *miasm*.

It is often convenient to speak of contagious and miasmatic-contagious diseases, under the common name of *infective diseases*, from which it is understood that miasmatic diseases are excluded, and also those other specific diseases which are due to an ordinary animal or vegetable parasite, or to a mineral or vegetable poison. And the process by which contagia act, may be designated "infection" with sufficient accuracy for all practical purposes, although this term is also applicable to inflammation and to other simple pathological processes.

Theory of Infection.—Within the last few years the subject of contagion in general has been profoundly studied. Contagia often appear as fluids; this is the case, for example, with the vaccine lymph, with the contents of smallpox vesicles or pustules, with the nasal discharge of measles, and with the faucial secretion of diphtheria. Chauveau, in France, and Burdon-Sanderson, in

England, have been at great pains to demonstrate the fact that the activity of such fluids is not inherent in themselves, and belongs, not to matters dissolved in them, but to very minute particles suspended in them. They have shown, for example, that when vaccine lymph is allowed to diffuse into distilled water, the diffused liquid is incapable of conveying cow-pox by inoculation. It would almost seem, however, as if the same conclusions might have been safely deduced from the circumstance that most kinds of contagia are capable of being conveyed by the air. For a liquid, or a dissolved solid, must, under such circumstances, pass into the state of vapor, and must rapidly undergo attenuation, so as to become inert; while, during the brief interval in which it could be supposed to retain its infective power, it would be likely to affect a number of persons indiscriminately, instead of attacking only a few, and those capriciously, as is actually the case.

That contagia are "particulate" may therefore be taken as a fact beyond question. The next point is as to the nature of the particles. For a long time the dominant view was that they were inorganic, or at least devoid of life, that their properties were essentially chemical, and that they acted "catalytically," or after the manner of *ferments*. On the ground of this analogy, a new epithet—"zymotic"—was coined, and it is still sometimes applied to the whole class of infective diseases.

But of late evidence has been accumulating from various quarters, which almost, if not quite, amounts to proof that the contagia are living organisms of exceedingly minute size. There is, in the first place, the fact that in at least two infective diseases—relapsing fever and anthrax—the constant presence of such organisms has been positively demonstrated. Certain pathologists believe that they have detected similar bodies in ague, in enteric fever, in measles, in cow-pox, and in sheep-pox; but with regard to the value and the significance of their observations there appears to be more doubt. It is, of course, to be admitted that the discovery of organisms in a particular malady does not warrant the conclusion that they are the cause of it, although when I am discussing relapsing fever and anthrax, I shall bring forward other reasons in support of such a view, so far as each of these diseases is concerned.

The fact which seems to me to tell most strongly, is that so many other processes which bear a clear analogy to specific infection are constantly associated with the presence of minute organisms, and that some have been proved to be caused by them. This is the case, for example, with several varieties of fermentation. Mr. Lister, in a most interesting paper read before the Pathological Society in 1877, established, I think conclusively, that the lactic acid fermentation or souring of milk is essentially dependent upon what he termed the *Bacterium lactis;* and Pasteur had previously shown that the alcoholic and the butyric fermentations have a similar origin. So, again, that putrefaction is set up by organisms appears now to be certain; the success of Lister's method of treating wounds is nothing less than a demonstration that the discharges from them remain sweet so long as the organisms which induce decomposition can be kept at a distance. Lastly, it has been shown by several observers, among whom Koch deserves special mention, that various traumatic infective diseases are due to the entrance of organisms into the blood, and to their multiplication within the tissues.

The organisms concerned in these various processes differ extraordinarily in size, and therefore in the readiness with which the microscope reveals them. The *Torula cerevisiæ*, or yeast plant, which brings about the alcoholic fermentation, is made up of rounded or oval cells, with granules in their interior, which cells develop from one another by budding, and have a diameter of $\frac{3}{10000}$ or $\frac{4}{10000}$ of an inch. The *Bacterium lactis*, which causes the souring of milk, consists of oval or rounded bodies, arranged in

pairs or sometimes in chains, multiplying by fission, and measuring at the most $\frac{1}{20000}$ of an inch, that is, being no larger than the granules which are contained in the cells of the torula. Consequently, as Mr. Lister suggests, there is nothing improbable in the supposition that yet other organisms may exist which may be as much smaller than the *Bacterium lactis*, as it is smaller than the *torula*. The characters of the *spirochæta* of relapsing fever, and of the *bacillus* of charbon, will be given when I am describing those diseases, but it is aptly remarked by Heydenreich that were it not for the length of the spiral threads which constitute the former organism, it would in all probability have still escaped detection; rounded or oval bodies not exceeding in diameter the breadth of the spirochæta would be almost, if not quite, invisible with the highest powers of the microscope. In his observations on the traumatic infective diseases, indeed, Koch, by the use of staining liquids, and by a peculiar mode of illumination, has succeeded in demonstrating micrococci which far surpass in minuteness those which had before been recognized. It remains to be seen whether a like success will attend the application of his methods of investigation to the exanthemata and to the other specific fevers.

As the application of so many different names is not a little confusing, it may be convenient that I should append the following classification :—

1. Moulds (*Schimmelpilze* of Nägeli). These are long-branched filaments, which form numerous spores. To the pathologist they possess scarcely any interest, but they are sometimes found lining the interior of dry vomicæ in the lungs.

2. Budding fungi (*Sprosspilze* of Nägeli). These consist of rounded or oval cells, which give off buds, and may form beaded threads. I shall have to describe them as causing certain cutaneous affections.

3. Fission fungi or schizomycetes (*Spaltpilze* of Nägeli). These are exceedingly small bodies, which multiply by fission, and are often arranged in pairs. They also sometimes cohere into delicate threads or into cubical packets. Several different names are given to them :—

α. Sarcinæ constitute square or cubical packets, the result of fission in different planes.

β. Spirilla (of which the *spirochæta* is a variety) are spiral filaments, having a well-marked corkscrew motion.

γ. Bacilli are cylindrical bodies, which sometimes remain united after they have undergone fission, so as to form threads of considerable length.

δ. Rod-shaped bacteria are very much smaller than bacilli. They are often slightly constricted in the centre, or dumb-bell shaped.

ε. Spherical bacteria are also exceedingly minute. They may be called *micrococci*, but it is necessary to bear in mind that different writers have not always used this term with precisely the same meaning.

By Billroth rod-shaped and spherical bacteria are associated together under the name of *coccobacteria*.

Bacteria sometimes cohere into chains; they are sometimes aggregated into masses held together by a jelly-like material, in which case the name *zooglœa* is given to them.

It can be easily understood that there is often very great difficulty in distinguishing micrococci from lifeless granules of organic or inorganic matter. Active movements, when they are seen, are, of course, conclusive; but an oscillating motion is of no significance, being dependent upon vibrations transmitted from without to the liquid in which the granules float. Bodies which subdivide or which cohere into chains may safely be set down as organisms. Irregularity in size is almost sufficient to show that the granules which differ among themselves are not micrococci; but uniformity in this respect affords no positive proof that organisms are

present. The power of resisting liquor potassæ has sometimes been supposed to be a criterion; but I believe that it is valid only as against granules of caseine or of other protein compounds, as, for instance, in curdled milk.

It must be clearly understood that this classification is one of names rather than of things, for there is still the greatest uncertainty as to the real relations between the different forms of these minute organisms. Consequently it is often advantageous to be able to refer to them under a common name, without specifying their exact characters; and for that purpose the word "microzyme," suggested by Béchamp and adopted by Sanderson, appears to be preferable to any other.

It has been supposed by Beale that microzymes, or "disease germs" (as he terms them), are derived by direct descent from the "germinal matter" of the human body, or of plants or animals, of whatever kind. He supposes that particles of this germinal matter, having become detached, continue to retain the power of multiplying independently to an indefinite extent. Others have thought that micrococci and bacteria may represent stages in the development of moulds or other fungi. Thus, Hallier, some years ago, put forth a speculation according to which microzymes found in the stools of patients suffering from cholera were supposed to have originally descended from a *urocystis* or "smut-fungus," parasitic upon the rice plant in India. But all such views appear now to be falling into discredit.

So, again, with regard to the schizomycetes themselves. Cohn admits several species of them; Nägeli says that he does not feel sure of the existence of more than one species, exclusive of the sarcinæ. At one time Mr. Lister believed that he had obtained a spirillum from the *Bacterium lactis;* but he has since become convinced that this was a mistake. There are, in fact, almost innumerable sources of error, which may vitiate the results obtained in any long chain of experiments, such as those of Buchner and Dr. W. Nägeli, which Prof. von Nägeli himself cites with approval, and from which it appears that "*Bacteria lactis,* when placed in liquids containing extract of meat and sugar, undergo such an alteration that if afterward introduced into milk they render it ammoniacal, and do not for a hundred or more generations regain the power of setting up the lactic acid fermentation." Mr. Lister himself, however, found that this kind of bacteria, after cultivation in urine for several days, became somewhat less energetic in its action upon milk. Conversely, several observers have thought that septicæmic blood, and the products of acute inflammation, acquire an enormously increased virulence when transmitted through a series of animals in succession.

These observations are not only important in themselves, but it also seems possible that they may afford a clue to the comprehension of a group of diseases, which are markedly contagious, but which it is nevertheless rather difficult to regard as being always specific, in the sense of "having a definite cause, arising from without the body, distinct from the causes of all other diseases."

Mr. Hutchinson, in particular, has repeatedly expressed the opinion that the "contagious inflammations" (as he terms them) differ essentially from the "specific fevers," and that they are conveyed from one person to another by leucocytes, or living pus corpuscles, and not by microzymes. The instances he enumerated in the discussion on the Germ Theory, held by the Pathological Society in 1875, were Gonorrhœa, Erysipelas, purulent Ophthalmia, and Porrigo. The points on which he relied in support of his views were chiefly two: first, the frequency with which some of these diseases occur apart from any apparent origin in contagion; and secondly, the fact

that the severity of the inflammatory process in each disease varies widely in different cases, and may be modified at will in experimental inoculations, as when gonorrhœal pus is applied for the cure of pannus. This last argument seems, however, to have very little force, since we shall find that as much may be said of some maladies which are typically specific. But it is impossible to deny that erysipelas often appears to be directly caused by exposure to cold; and a similar origin can sometimes be traced for diphtheria, an affection of which the specific character is on other grounds doubted by some pathologists. It seems a simple way out of the difficulty to suppose that the diseases in question begin as simple inflammations, and that they acquire infective characters secondarily. But such a view by no means excludes microzymes from being, in all probability, the bearers of contagion. It seems to me that Lister's observations on the lactic acid fermentation teach an important lesson in regard to this question. One would certainly have thought that the souring of milk, which so constantly occurs when that fluid is left standing for a few hours in warm weather, must depend upon causes which are in operation everywhere, and that any microzymes concerned in the process would be likely to be devoid of specific characters, at least until they had germinated within the milk itself. But Lister found that although the *Bacterium lactis* appears to be universally present in dairies, it is scarce in the world at large; milk which has been boiled may be exposed to the air in ordinary rooms for half an hour at a time; but while other bacteria and filamentary fungi develop, it will rarely, under such circumstances, become curdled and sour, nor will it present the *Bacterium lactis*. Is it not probable that even though the occurrence of a common catarrhal inflammation favors the development of erysipelas or diphtheria, yet access of specific microzymes is the essential factor in the ætiology?

But some physicians of well-deserved authority think that certain of the more typical specific diseases sometimes arise *de novo*, and independently of contagion or of any virus derived, directly or indirectly, from previous cases. In reference to this question, it seems to me useless to insist on the fact that all such diseases must have had a commencement at some period of the world's history, just as a similar argument may fairly be disregarded when employed to support the theory of spontaneous generation of any of the lower plants or animals at the present time. On the other hand, I think that very little stress can be laid on the circumstance that the inhabitants of certain countries, especially islands, remain free from particular exanthemata for many years, but afterwards suffer severely, when a case is introduced from without; for even if a disease were capable of springing up *de novo* under certain conditions, a great length of time might obviously pass in a given locality without the conditions being fulfilled. What appears to my mind an argument of importance is that when the origin of an outbreak of fever or of any other contagious malady has been investigated with sufficient care, it has so often been traced in the most conclusive manner to a previous case, perhaps at a great distance, from which it may have been derived in some most indirect and unexpected way. The instances which I shall have to relate when discussing the ætiology of the several infective diseases render it, as I think, almost impossible that the development of any one of them *de novo* can ever be positively proved. And it is worthy of notice that particular instances, of which no satisfactory explanation can be given, occur in the case of smallpox—of which a spontaneous origin would generally be regarded as most unlikely—no less than in that of diphtheria, or typhus, or enteric fever, which are held by some authorities to be frequently so generated. Sir Thomas Watson cites Dr. Gregory as having stated that, of the cases admitted

into the Smallpox Hospital in his day, not one in twenty could be referred to any known source of infection. I am therefore strongly disposed to reject the doctrine that any of the specific diseases ever result from such causes alone as overcrowding or starvation ; or even from the inhalation of sewer-gas which contains no specific virus, but only the excrement of healthy persons. But it is another question whether the microzymes of an infective malady, after escaping from patients, may not sometimes go on multiplying for long periods of time, and spread to distant localities, before they again penetrate into the human body. In the miasmatic contagious diseases we believe that such a process of development always occurs. In the strictly contagious diseases there seems to be no reason why it should not occur sometimes. Again, we have yet to learn whether some of these maladies may not occasionally be derived from the lower animals, as favus certainly is from cats.

When the virus of an infective malady enters the human body, an interval occurs during which the health of the patient remains apparently undisturbed: this is called the period of *incubation.* In some diseases, as in smallpox, it is almost always of the same length. This fact is of great importance, because it enables us to say, when a person has been exposed to infection, that after the lapse of a certain number of days, if not already attacked, he is safe, and may mix with other people without risk to them. It is, in other words, the foundation upon which rests the practice of *Quarantine.* The duration of the incubation of each specific disease has accordingly been studied with care. The only cases in which it can be positively determined are, of course, those in which there has been but a single exposure to contagion ; but others, in which the exposure (though repeated) began only a few days before the patient's illness showed itself, are valid as proofs of a short incubation ; and yet others, in which the exposure ceased many days before he felt ill, are valid as proofs of a long incubation. It seems now to be established, beyond all doubt, that whereas the incubation of each specific disease is fixed within certain limits for the majority of cases, there are yet some cases in which the limits are widely overstepped. Thus scarlet fever, which ordinarily has an incubation of nearly a week, sometimes breaks out within twenty-four hours after the first exposure to its infection. It is undoubtedly hard to reconcile this fact with what is otherwise a most reasonable explanation of the usual occurrence of a longer interval, namely that it is occupied by the multiplication of the virus within the body. According to Nägeli schizomycetes commonly double their numbers in from twenty to twenty-five minutes at the temperature of the blood. Lister found that the *Bacterium lactis* took about an hour in completing the process of growth and subdivision. In diseases such as syphilis, in which a local lesion precedes the development of constitutional symptoms, the virus may be supposed at first to multiply itself locally (in syphilis, within the hard chancre) and not to enter the blood in any considerable quantity until a later period. There is evidence that in the rare instances of the syphilitic poison being communicated by vaccination, the resulting chancre has yielded an infective material within ten days afterward. On the other hand, an unfortunate case once occurred at the Charité Hospital of Berlin, in which variola was conveyed to a patient by the operation of skin-grafting, the graft having been taken from a person who happened to be in the incubation stage of the disease. This seems to show that from a very early period the whole of the skin contains the virus, which can have reached it only through the blood; and if so, the same thing must occur likewise in the exceptional instances in which smallpox is inoculated into the skin at a particular spot. In such cases, however, there is developed a "primary" or "mother-vesicle" which precedes the general eruption by

some days. It therefore seems to be very unlikely that in any disease the multiplication of the virus during the incubation remains altogether limited to a local lesion, even if it is more active there than elsewhere.

Such speculations are of considerable importance, because they touch a question which is still discussed, namely, whether in specific diseases generally the various local lesions that characterize them are to be regarded as sources of blood-infection or as consequences of it. Thus in diphtheria, one opinion is that microzymes first settle upon the mucous surface of the fauces, or upon whatever part happens to be the seat of the diphtheritic process, and that they afterwards penetrate its tissues and enter the blood. Some observers have even thought that in enteric fever blood-infection is secondary to the intestinal lesions; and Liebermeister endeavors to prove that in yellow fever there is a primary parenchymatous hepatitis. But in typhus and in relapsing fever we have diseases to which such a view is altogether inapplicable; and, in the exanthemata, fever or other constitutional symptoms generally precede the cutaneous eruptions by a considerable interval of time. Moreover, in diphtheria itself there is a well-marked tendency for the throat to be affected when the virus has originally been deposited upon some other mucous membrane or even upon the cutaneous surface, and when it may therefore be supposed to reach the fauces through the blood.

Physiological Action of the Microzymes.—With regard to the mode of production of the pyrexia and of the other symptoms of specific diseases by their contagia we know at present scarcely anything. The microzymes, in multiplying so enormously as they do, must, of course, abstract nutrient materials from the blood and from the tissues. By Nägeli the idea of a "struggle for existence" between these rival organisms is carried out in detail. It is shown that when a person is exposed to contagion his taking the disease or resisting it depends probably in part upon the number of microzymes which enter his body, in part upon the condition of his blood and of his tissues at the time. Every one who has experimentally cultivated the lower organisms knows that when more kinds of organisms than one are present, a slight change in the composition of the fluid in which they are placed may completely alter the result, enabling a microzyme which previously was weaker to outgrow that which had been the stronger. And so Nägeli imagines that slight alterations in the state of the blood may greatly favor or oppose a contagion in the competition which it undergoes. The analogy of the various fermentations suggests (as Lister has pointed out) that, besides appropriating the materials for their own growth, microzymes may also cause further disturbance by catalytically decomposing other substances of which they make no use. It is, therefore, not surprising that their presence sets up pyrexia, which I believe to be a quasi-physiological process, having for its object the destruction and removal of noxious matters, of whatever kind, from the circulating fluid. The evidence in support of such a view will be discussed hereafter.

Such a destruction of the contagious microzymes within the blood, as the result of the febrile process, would explain what has been, in the minds of many pathologists, one of the greatest obstacles to the acceptance of the so-called "germ-theory" of infective diseases, namely, the fact that so many of them run a definite course, and terminate in the recovery of the patient at the end of a fixed period of time. But a further hypothesis seems to be absolutely necessary. It is well known that most of these diseases occur only once in the life of the same individual; and perhaps no one of them is entirely devoid of such a protecting influence. I fail to see how this can be accounted for, except on the supposition that in the course of each disease the blood or the tissues undergo such a change that they no longer afford, and never afterward afford, the conditions requisite for the

development of the corresponding microzymes. Perhaps it is not necessary to imagine that a special pabulum is exhausted, and there certainly are very obvious objections to the notion that human beings in general are born provided with a number of different materials which serve no known purpose but that of affording nutriment to the various contagia, if they ever find their way into the body, at however advanced a period of life. It is, however, to be borne in mind that the facts of contagiousness to other individuals, and that of subsequent immunity for the person already attacked, are almost necessarily connected together. Were it not so, almost everybody who passes through any one of these maladies would catch it from himself again and again.

The duration of the protective action seems to be very variable. In some diseases, as in relapsing fever and in diphtheria, its duration is perhaps no greater than is just sufficient to prevent the patient from reinfecting himself. In others it lasts during the whole of life. Indeed, a very interesting question is whether it may not be transmitted from parent to child. As is well known, the exanthemata sometimes rage with extraordinary virulence when they are introduced into communities that have been free from them for a great length of time. No doubt other explanations of this fact may be given; but it is at least possible that the reason may be that the victims come of a stock which has not for many generations been exposed to the contagion. It would be very interesting to investigate to what extent differences in the severity of a specific disease, attacking various individuals under the same conditions, in countries where it is always more or less prevalent, may depend upon whether one or both of the parents of the several patients had had that disease in childhood, or at least before the patients themselves were born. It might even happen, in the case of a malady which commonly affects adults, that successive generations should in turn succumb to it, in consequence of the continued absence of such an inherited protection.

Stamping Out Infective Diseases.—These considerations seem to me to render very doubtful the advisability of attempting, even if it were practicable, to eradicate infectious diseases from this or any other country, and then to keep them off by a system of quarantine. Sir Thomas Watson recently gave to such a scheme the weight of his authority. But sooner or later each disease would be sure to find an entrance, and it would probably commit unheard of ravages among a population so long free from it. I doubt whether it is even right to teach the public to look upon the maladies in question as in a special sense "preventable." They are certainly not so much so as those affections which result from over indulgence, from exposure to cold, or from hard work unsuited to the physical powers of the individual. The true method of combating infection seems to be that of which we have an illustration in the practice of vaccination. And there is reason to hope that in the future this method may be far more widely extended than hitherto. Pasteur and Toussaint in France, and Dr. Greenfield in this country, have independently demonstrated the fact that by passing the virus of anthrax through guinea-pigs, its intensity can be diminished so that it is no longer fatal to bovine animals, and that after inoculation with this modified virus they are no longer susceptible of the disease. One could not render a greater service to humanity than by discovering how to transmit the contagion of scarlet fever through any of the lower animals in such a way that when reintroduced into the human subject it should convey protection without risk. I am not even sure whether it might not be well to expose children, on purpose, to infection from this disease, when it happens to be epidemic in a mild form. Such a procedure has sometimes been advocated, but medical opinion has hitherto been that it cannot be done with safety, because

a mild case often generates a severe one. But it is at least possible that this may only be true when the disease was originally of a severe type, and when it has chanced to produce a slight illness in an individual who (having inherited protection) is but little susceptible of it, so that it reverts to its original character when it passes on to a subject not thus guarded against it. For it is well known that, as a matter of fact, different epidemics of scarlet fever differ widely in intensity. Sometimes it is the most fatal of all contagious diseases; but sometimes it passes from house to house through an entire village, without destroying a single patient or giving rise to any dangerous sequelæ. May it not be that this is where the activity of the virus has been lowered by passing through a number of persons in succession, each of whom was only slightly susceptible of it? If so, we may possibly find a very simple means of obtaining a virus suitable for inoculation, without having recourse to the lower animals.

Method of Transference of Contagia.—We have still to consider how the microzymes of contagion or of miasm gain access to the human body.

According to Nägeli, the way in which they enter when suspended in the air is generally by penetrating into the capillaries of the lungs through the walls of the pulmonary alveoli. He is inclined to doubt whether they ever make their way through the mucous membrane of the mouth or the fauces, except when they settle upon it and first give rise to an intense local inflammation, as he (with so many other German writers) believes to be the case in diphtheria. But he thinks that they sometimes enter the blood through wounds or abrasions. It has been supposed that examples of such a mode of infection are sometimes afforded by the so-called "surgical scarlet fever," which so often breaks out in children after operations; but, as Dr. Goodhart has shown, in the "Guy's Hospital Reports" for 1879, that its occurrence is not prevented by the strictest "antiseptic" treatment, there appears to be more probability of its arising in the ordinary way. This, however, is a point which I shall have to discuss hereafter.

Contagia have traditionally been divided into those which are "fixed" and those which are "volatile." Miasmata have been regarded as of necessity volatile. But, as we have seen, it is certain that none of them are gases or vapors, and that they all consist of solid particles of exceedingly small size. Nevertheless, even recent writers have taught that the particles in question are capable of escaping with the water which evaporates from liquids or from moist solid surfaces, and of diffusing themselves in the air.

Nägeli, however, seems to have conclusively demonstrated the very important fact that this is impossible. He performed a series of experiments with U-shaped tubes connected together. In one bend he placed a liquid suitable for the growth of microzymes; another bend he filled with sand saturated with a putrefying liquid. Even when air was drawn through the apparatus he found that no microzymes passed from one tube to the other. And on theoretical grounds he shows that no other result could have been anticipated.

The full acceptance of Nägeli's conclusions means little less than a revolution in our conceptions of the process by which contagia escape from the human body or miasmata from the soil. We have been accustomed to think that the breath of a person suffering under an infective disease is apt to be highly charged with the virus, and that the offensive odors exhaled from his moist skin, or from his excreta, may be highly dangerous. According to Nägeli all such notions are erroneous; he even declares that the presence of fetor is generally a sign that the substances which give rise to it are still moist, and are therefore incapable of setting free any micro-

zymes which they may contain. But it is obvious that some parts of an infected mass may remain covered with fluid, while others have already undergone desiccation.

Of course, it is not disputed that liquids in which the microzymes of a specific disease are floating may convey it to healthy persons. Indeed, there are some maladies, such as hydrophobia and syphilis, which seem scarcely to be communicable except by a process of inoculation, the poison being introduced into a bite, or a wound, or a crack in the skin. And diphtheria is often spread by the direct transference of fluid secretions from one individual to another, as in the act of kissing, or when a patient coughs and expels mucus into the open mouth of a medical man who is looking into his throat. It is also to be remembered that the stings of insects may sometimes be the means of conveying a virus; cattle, for instance, are liable to be infected with anthrax by gad-flies.

Moreover, there is the clearest evidence (although this Nägeli rejects) that certain specific diseases are frequently transmitted by means of liquids which are swallowed. So far as drinking water is concerned, I shall have to cite the facts in full detail when I am describing enteric fever and cholera. Still more interest attaches to the remarkable facts which have recently been made out by English observers as to the conveyance of the poisons of enterica and of scarlatina by means of milk. It is possible that, even in water, the specific microzymes multiply greatly in the interval which elapses after it becomes contaminated by them, and before it reaches the human stomach. But there can be little doubt that milk affords far more favorable conditions for their growth; for Lister proved, in his experiments with this fluid, that (unlike even Pasteur's solution) it was capable of serving as a pabulum for almost any kind of microscopic organism. Once only did he meet with a bacterium that could not live in it. Now, I shall hereafter have to point out that in some of the cases in which specific diseases have been widely communicated in this way the quantity of virus which has been allowed to enter the milk has probably been very small. It, therefore, seems to me that the occurrence of milk epidemics tends powerfully to support the germ theory. I may observe that probably the only effectual method of purifying either milk or water, when contaminated with microzymes, is by boiling. Filtration seems to be altogether useless; and it is not likely that the addition of minute quantities of Condy's fluid would suffice to destroy them.

With these important reservations, however, I think that Nägeli is right in maintaining that infection generally occurs in a very different manner, namely, by means of microzymes which have undergone desiccation, and which rise into the air and remain suspended in it in the form of a fine dust; the particles, perhaps, even more minute than those which become visible in the track of a sunbeam. How little influence gravitation has in causing the subsidence of such particles is well illustrated by the facts that volcanic dust from Vesuvius is carried in the atmosphere to distant parts of Europe, and that the trade-winds convey another kind of dust right across the Atlantic Ocean from America.

The conditions under which microzymes thus become suspended in the air must be discussed separately for miasmata and for contagia.

(1) In the case of a *miasm*, at least any *miasm* of telluric origin, the first condition of its development is the presence of water in the interior or upon the surface of the ground, and the presence in this water of substances fit to serve as pabulum for the microzymes. Further, it is necessary that the level of the water should vary from time to time, so that particles of the soil itself, or the stems and the leaves of plants growing upon it, should undergo desiccation, and should receive deposits

of microzymes, also in a dry state. So far as the miasm of ague is concerned, I do not know that there is any necessity for supposing that it is ever derived from any depth below the surface of the ground, or for invoking the assistance of any force beyond that of the wind, for the purpose of carrying up the dried microzymes into the air. But, if any value at all is to be attached to the observations which Pettenkofer and others have made at Munich with regard to enteric fever and to cholera, microzymes concerned in the propagation of these "miasmatic-contagious" diseases must be assumed to come, in some cases, from the soil at a depth of several feet. The conditions which may be supposed to render this possible are very carefully argued out by Nägeli. In the first place, he thinks that in the ground-water, which saturates a porous soil below a certain level, the growth of such organisms is likely to be especially active towards the surface of this water. Hence, the slightest fall in the level of the water must cause an abundant settlement of microzymes upon stones, pieces of sand, and fragments of clay and humus, which come into contact with air as soon as the water recedes from them. In this subterranean atmosphere he conceives that currents are produced by various causes: changes of temperature, changes of pressure, winds, and lastly, the suction action of warm air into the interior of houses having deep foundations. Such currents, he thinks, may easily carry away the dry microzymes, especially if there is no precipitate of colloid matter from the water, to make them adhere closely to the substances on which they are deposited. A further condition, at least so far as concerns their escape into the air above, is that the superficial layers of the soil through which they must pass should not be damp, and should not be covered with a uniform carpet of vegetation.

(2) The microzymes of a *contagion* doubtless leave the human body mainly in the fluid secretions and excretions, the fæces, the urine, and the sweat. They may also be attached to the solid particles of cuticle which are constantly being shed from the surface; in the case of scarlet fever and in that of smallpox this mode of diffusion of the poison is believed to be of great importance. In either case, it is hardly necessary to say that their escape is due to no process of "elimination," and is of no advantage whatever to the patient. Indeed, the microzymes which find their way out of the body are probably altogether insignificant in number, when compared with those which remain in the blood or in the tissues, and undergo destruction there. The microzymes which adhere to fragments of cuticle are already dry; those which are contained in fæces, in urine, or sweat, must undergo desiccation before they can reach the air. Nothing is more favorable to this than the soaking of linen or other fabrics with any of the infected secretions. The microzymes remain upon the surface of the cloth when it dries, and are afterwards shaken off by the slightest movement. It is a most natural circumstance, therefore, that washerwomen and their families should be very liable to attack by contagious diseases; and instances in which infection has been traced to the hanging out of clothes to dry are readily explicable, if one considers how imperfectly the process of washing is often performed.

One cannot state, with any approach to certainty, how long a patient remains capable of infecting others after his recovery from a contagious disease. It is not unlikely that his secretions cease to contain microzymes, except in very small quantities, as soon as the pyrexia is at an end. This, at least, appears to be the natural inference, from the observations which have been made of the rapid disappearance of *spirillum* from the blood in relapsing fever. But, for a considerable time afterwards, fresh portions of infected cuticle may be supposed to be continually shed.

On the other hand, contagia sometimes adhere with great tenacity to the

walls of an apartment which has been tenanted by a patient suffering under an infective disease, and to bedding or clothes which have been used by him. Nägeli mentions that at Munich several masons fell ill with small-pox after scraping the ceiling of a room in which small-pox cases had been treated six or seven years before, and which had then been white-washed. Sir Thomas Watson relates the following instance: A house in which several persons had been attacked by scarlet fever was left empty for a year. When the family returned, a drawer in one of the bedrooms resisted for some time attempts to pull it open. A strip of flannel had got between the drawer and its frame, and had made the drawer stick. This piece of flannel the housemaid put playfully round her neck. An old nurse who was present, recognizing it as having been used as an application to the throat of one of the subjects of scarlet fever, snatched it away and burned it. The girl, however, soon sickened with the disease. Woolen substances seem to afford the most favorable conditions that can be imagined for the preservation of microzymes in a state of activity, short of their being enclosed in sealed glass tubes. By the hygrometric properties of such substances, contagia adherent to them may probably be prevented from undergoing too complete a desiccation, while they are at the same time protected from currents of air.

Destruction of Contagia.—Steps must always be taken to prevent a person affected with a contagious disease from being a source of danger to others. But what can really be done in this direction does not seem always to have been clearly apprehended by those who have laid down rules for the purpose. It would appear—

A. That during an illness, the utmost care should be exerted to hinder the contagion from being carried away from the sick-room by attendants, or in the patient's excreta, or upon his bed- or body-linen, or in any other way. In all probability its escape into the open air through windows or chimneys is of no consequence.

B. That at this time, it is impracticable effectually to disinfect the patient himself or his surroundings, and therefore that the attempt should not be made.

C. That the patient, after recovery, and the nurses also, should be most carefully disinfected; that minute pains should be taken to prevent clothes, books, and other articles from conveying contagion; and that the sick-room with all its contents should be thoroughly purified before healthy persons are allowed access to it.

The measures which should practically be adopted to ensure these objects seem to be the following:

1. The patient himself must be isolated in a separate room or suite of rooms, which, if possible, should be at the top of the house. An intervening passage, of which the windows can be opened, is, of course, an advantage. Ventilation should be carefully attended to, and it is well that there should be a fire, so as to maintain a draught up the chimney. The doors should be kept closed, and outside them there should be hung an old sheet saturated with a liquid solution of carbolic acid or chloride of lime, and never allowed to become dry. The addition of glycerine to this liquid is probably advisable. It seems to be useless to expose vessels containing carbolic acid in the sick-room itself, or to scatter chloride of lime upon the floor.

Those who nurse or wait upon the patient should not be allowed to enter other parts of the house, and, if possible, such persons should be chosen for this duty as have already had the disease. The medical attendant should take care, by ablutions, by change of clothing, and by exposing himself freely to the open air after each visit, to avoid carrying

contagion abroad. One is often asked whether the father of a sick child should be allowed to continue his work in an office or chambers, so as to come into contact with fellow-clerks and with the public generally. My own opinion is, that if he avoids contact with the patient and takes a good walk when he leaves the house in the morning, no appreciable risk is run, so long as he himself remains well.

All curtains, carpets, and padded chairs, all articles of wearing apparel in cupboards or in drawers, all unnecessary articles of furniture, of whatever kind, should be removed from the sick-room before the patient is placed in it, because their presence will make unnecessary difficulties afterwards. The supply of books or toys should be limited to such as are not too valuable to be burnt when they are done with. Any food or drink which the patient or the nurse may leave should be thrown away, and must never be taken by other persons. Cups, plates, and spoons should be washed in very hot water before they are allowed to go back to the kitchen; but, as far as possible, it is well to keep the same articles from day to day in use in the sick-room, and to wash them there.

2. All the excreta of the patient should be rendered innocuous by the addition of some disinfectant.

The experiments of Dr. Baxter, recorded in the sixth volume of the new series of Mr. Simon's "Reports," have shown that few at any rate of the substances commonly used really possess the power of destroying contagia. He employed *carbolic acid*, *sulphur dioxide*, *potassic permanganate*, and *chlorine*. Each of these substances was added in definite proportions to vaccine lymph, and a series of vaccinations were performed with the mixed liquids upon children, one arm of the child being inoculated with the lymph supposed to be disinfected, while the other arm was at the same time vaccinated with lymph that had been diluted with water to a corresponding degree. The results which he obtained were: that with chlorine and with potassic permanganate there is no security for the effectual disinfection short of the presence of free chlorine or of undecomposed permanganate in the liquid, after all chemical action has had time to subside. In the case of sulphur dioxide the criterion of success is that the liquid should be permanently and strongly acid; in the case of carbolic acid that it should contain at least 2 per cent. by weight of the pure acid. It is further necessary that the disinfectant should be thoroughly incorporated with the liquid; there must be no solid matters capable of shielding the contagious particles.

An additional point on which Dr. Baxter lays stress, and on which Nägeli had previously insisted, is that the addition of disinfectants in too small quantities may do harm instead of good. Probably all the substances that can destroy contagia are also antiseptic agents; that is, they can prevent putrefaction. And it appears that they are capable of doing this even when present in too small a quantity to disinfect. At first sight this seems contradictory. But it is not really so; for Dr. Baxter has shown, that when an infusion to which (for example) 2 or 3 per cent. of carbolic acid has been added fails to putrefy, the bacteria in it nevertheless remain alive, and that they begin to multiply if a little of the infusion is mixed with another liquid that can afford them pabulum. Now, it is this power of affecting other liquids, and not its capacity for undergoing putrefactive changes itself, which is really comparable with pathological infectiveness. And it is a well-ascertained fact that (except, perhaps, in the case of glanders) the occurrence of putrefaction in a liquid deprives it at once of whatever contagious property it may possess. Thus, if a disinfectant is added in too small quantity, it may easily happen that its action is precisely the opposite of what is intended: by preventing putrefaction, it keeps the contagious microzymes alive.

3. For the purpose of wiping off discharges from the nose or the mouth of the patient, pieces of rag should be used, and they should be burnt immediately afterwards. The sheets and body linen should be placed in a tub of water as soon as they are changed, and they should, of course, be washed apart from those of other persons. It is usual to soak them in a weak solution of carbolic acid or in diluted Condy's fluid; but this is probably of little use. The really important point is that they should be submitted to the action of water at a high temperature. The safest plan is to boil them in a copper. But there is reason to believe that a degree of heat far below 212° may be sufficient. Thus, Davaine found that the virus of anthrax diluted with water was destroyed in five minutes by a temperature of 131° F., and Dr. Baxter that even dry vaccine was rendered inert in thirty minutes by a temperature of 185° or upward. There appears to be no doubt that in the dry state contagia resist heat much better than when they are suspended in water.

The linen worn by the nurse should be treated in the same way as that of the patient. The dress should always be made of a material that will bear washing.

4. At the end of the illness, when it is believed that the patient is no longer giving off contagion from his body, he must be carefully washed, carbolic soap being perhaps preferable to other kinds. Special pains should be taken to cleanse the hair thoroughly. He should then be dressed in clothes none of which have been in the sick room, and should be moved into another apartment. Similar precautions should be taken whenever a nurse or an attendant goes away, at whatever period of the case.

5. The room which has been occupied by the patient during the disease must be carefully disinfected before healthy individuals are allowed to occupy it.

In the first place, movable articles that cannot be washed should, if possible, be exposed to dry heat. As I have already remarked, the temperature required for the destruction of contagia in the dry state appears to be considerably higher than that which suffices when they are suspended in a fluid. It varies with the length of exposure, the time needed being longer in proportion as the temperature is lower. Some hospitals for infective diseases have hot-air chambers, erected for the purpose, in which there is no difficulty in raising the temperature, even in the centre of a flock-bed, to 220°, 240° or 300° F. There seems to be no doubt that this must be completely effectual in killing any kind of virus.

The next step is to destroy any microzymes that may be floating in the atmosphere, or adhering to the sides of the apartment, or to the furniture. With this object it may be exposed to the fumes of sulphur or to chlorine gas. All the openings in the room, chimney, windows and doors, must be made as air-tight as possible. A good plan is to set fire to a few ounces of bisulphide of carbon in an iron pan supported over a pail of water. Or from a quarter to half a pound of brimstone broken into pieces may be placed in the pan and live coals used to ignite them. The room is then closed and left so for several hours. The ceiling should also be scraped and whitewashed, the walls should be repapered, the floor should be thoroughly scrubbed, and all the paint in the room, as well as the furniture, should be very carefully washed.

FEVER.

PYREXIA — THERMOMETRY — HYPERPYREXIA — THEORY OF FEVER — POSSIBLY BENEFICIAL EFFECT OF PYREXIA — IDIOPATHIC AND SYMPTOMATIC FEVER — RAISED TEMPERATURE WITHOUT FEVER — THE STAGES OF FEVER — CONCOMITANT PHENOMENA OF PYREXIA: NUTRITION: PULSE: RESPIRATION: MUSCLES — SUBNORMAL TEMPERATURE.

Among the varied effects of disease there is, perhaps, no one which is more commonly met with—and certainly there is no one which is more important—than PYREXIA or FEVER. It is not surprising, therefore, that the ancient writers should have recognized this condition, which by Galen was defined as "*Calor præter naturam.*"

But it is not a little strange that in modern times scarcely any attention was paid to it until very recently. The specific fevers, of course, were studied; but of the general febrile state little or no notice was taken. An increased action of the heart was considered as its chief characteristic.

It would be difficult to overstate the value of an advance which was made early in the second half of the present century, in the introduction into medical practice of the clinical thermometer, as a direct and simple method of determining the presence or absence of pyrexia. This was not, indeed, a novelty. About a hundred years previously the instrument had been used by Boerhaave and by his pupils Van Swieten and De Haen; and the last of these, who was the first clinical teacher at Vienna, had discovered the striking fact that in ague, during the cold stage, the temperature of the blood is increased. But when in 1850–51, Traube and Bärensprung independently published papers on the subject, no one else was in the habit of making thermometric observations at the bedside. From that date Wunderlich, of Leipzig, devoted infinite pains to the study of temperature in all diseases. In this country the practice was soon adopted by Parkes, Ringer, Aitken, and a host of others; and we may safely assert that it will never again be abandoned.

The use of the thermometer in clinical practice requires considerable care, if the results are to be relied on as being even tolerably accurate. The instrument is most commonly placed in the axilla, but sometimes in the closed mouth, the rectum, the fold of the groin, or elsewhere. Now, it is essential to observe that whichever of these places may be selected for the purpose, the temperature which is to be obtained is that not of any part of the cutaneous surface, but of the deeper structures of the body, an inch or more below the level of the skin. To determine the surface temperature of any region is quite another and a most difficult matter. Instruments which have been called surface thermometers have been constructed, with the receptacle for the mercury of such a shape that one side of it can lie flat on the skin while the other side is protected with a non-conducting material, so as to diminish as much as possible any loss of heat from it. But to cover up any part of the surface of the body is, *ipso facto*, to raise its temperature above that at which it previously stood.

Thus, when a thermometer bulb is placed deeply in the axilla, the fold of the groin, or elsewhere, the mercury goes on rising until it indicates a temperature corresponding very closely with that of the solid substance of

a limb, at the same distance from the skin as that at which the instrument is buried. Consequently it is extremely uncertain what length of time such an observation may take for its completion. A great deal depends upon whether the parts were closely in contact before the thermometer was introduced. Thus, if the patient is lying on one side in bed, one should always choose the more dependent of the two armpits, since it will give a fixed temperature far more quickly than the other one. When a thermometer has once risen to its full height in the axilla, a second one placed in the same spot immediately afterward will rise to the same point in from three to five minutes. But it is a very different matter if the arm has been separated from the chest, so that the axilla has contained air, and perhaps a thick fold of the underclothing. It must then itself rise through several degrees before it can bring the thermometer to a stationary point. And the length of time required for this to take place will vary indefinitely, according as the circulation of blood in the peripheral parts of the body is active or otherwise. When the skin is hot and turgid, as in scarlet fever, a comparatively short period will suffice; in the cold stage of ague, and still more during the collapse of cholera, it is doubtful whether an axillary temperature can ever be relied on. It is to be observed that this uncertainty is altogether independent of any defect of sensitiveness in the instrument which happens to be employed.

I have gone into the matter thus minutely because there is reason to believe that the scientific value of thermometric observations made in this country is largely impaired by the comparatively short space of time devoted to them. It is commonly thought that from three to five minutes suffice for this purpose; and it must, of course, be admitted that a temperature above the normal, however quickly obtained, is a positive fact, which from a practical point of view can never be worthless. Dr. Bäumler has shown ("*Brit. Med. Journ.*," 1864), by direct experiments, that even when all ordinary precautions are taken, it may easily happen that too low a reading, by .3 to .8 of a degree Fahrenheit, is arrived at, when the thermometer is withdrawn from the axilla at the end of five minutes. He found that from eleven to twenty-four minutes are required to give an absolutely trustworthy result. Liebermeister goes still further; for even of a period of from fifteen to thirty minutes, he does not say more than that it suffices for the majority of cases. The rule which he lays down is that the instrument should be observed to remain stationary for five minutes before it is removed.

It is essential, in placing the bulb in the axilla, that no clothes should be allowed to remain in contact with it, and that the skin should grasp it firmly. If there be any perspiration, the hollow must first be wiped dry. The arm should then be folded across the chest, and the hand may be made to take hold of the opposite arm, while the opposite hand supports the elbow of the side on which the thermometer is. Whenever the parts do not seem to meet closely round the instrument, some other region should be selected. The mouth is fairly suitable, if one can depend upon the patient's keeping it constantly closed and breathing entirely through the nose. The bulb may be placed either beneath the side of the tongue or at the back of the cheek. Dr. Bäumler found that from nine to eleven minutes sufficed to raise it to a fixed point there. But of all possible localities for thermometric observations, the rectum is by far the best. It would always be preferred, did not considerations of decency stand in the way; and in very young children, as well as in old people, this may often be neglected. An important advantage is the saving of time, for from three to six minutes are enough to give a temperature of scientific value. Consequently, it is advisable to make use of the rectum whenever much depends upon the exact temperature at the moment, or when an ice-bath may perhaps be required. And it is to be

noted that a rectal temperature can be taken with a registering thermometer when the body is immersed in cold water.

When, after the withdrawal of the instrument, the index has been read, it is common to dot down the result on a sheet of paper ruled for the purpose. Observations are repeated at regular intervals twice a day, every two hours, or even oftener, and each successive reading is recorded in a similar manner. Lines are then often traced from dot to dot, so as to form what is termed a "chart." It is, however, very important to remember that the result is entirely artificial. However short may be the intervals at which the thermometer is applied, there is no reason for supposing that the patient's temperature moves straight upward or straight downward from one point to another. And when the intervals are long, as when the instrument is employed only twice in the twenty-four hours, there is not even a probability that the dots marked on the chart each day represent respectively the true maximum and the true minimum. We shall, indeed, presently see that in disease, as in health, there are, as a rule, certain daily fluctuations, the temperature being generally highest at a particular hour in the evening and lowest in the morning. But even when the observations are made just at those times, there is no security that the rule is observed in that special case on any one day. And it is always highly probable that between each pair of observations there may have been two or three, or even several, ascents and descents of the temperature which altogether escape notice. I therefore prefer to make no chart at all, but to place the figures themselves in two vertical lines, one for the morning the other for the evening.

The course of pyrexia is naturally divisible into certain periods or stages, which, however, vary greatly in length in different diseases. First comes the "pyrogenetic" or "initial" stage; during its continuance the temperature rises more or less steadily, the rise being interrupted, if it extends one, two, or more days, by the daily fluctuations already alluded to. When it is of short duration, or when the rise is at first rapid, there is commonly a shivering fit or *rigor*. The second stage is the *fastigium;* in it the temperature reaches its *acme* or highest point; but, not infrequently, since this stage may last for several days or even two or three weeks, it is characterized (independently of the daily fluctuations) by a series of irregular slight ascents and descents, so that the highest point or one very near it may be touched again and again at longer or shorter intervals. The third stage is that of "defervescence," during which the temperature falls again to normal. Sometimes, however, it is separated from the second stage by an intervening period, called by Wunderlich the "amphibolic" stage, in which irregular exacerbations and remissions are observed. Or, again, there may at the end of the second stage be a marked rise of temperature, termed by Wunderlich the "*perturbatio critica.*" The third period, that of defervescence, is sometimes protracted, sometimes short and sudden. In the former case the pyrexia has been said to end by *lysis*, in the latter by *crisis*. The rule is to speak of a critical termination only when the fall to a normal temperature is completed within thirty-six hours; but sometimes the third stage begins with a slight and gradual descent, which after two or three days ends in a crisis. When defervescence is rapidly accomplished, it is generally accompanied by profuse sweating, and sometimes by an abundant flow of some other secretion.

That Wunderlich and others should thus have adopted the term "crisis" is, I think, to be regretted. For it is scarcely possible for any one who has had occasion to refer to the medical writings of the ancients to shake off entirely the remembrance of earlier mystical notions which formed a very important part of their teaching. When I come to describe the specific forms of fever, we shall find that each of them has a more or less definite duration,

and that one can often confidently look forward to a future day, a week or two distant, as that in which defervescence is likely to take place. But this is very far from what the Greek writers, or even some moderns, have meant by critical days, their idea being that all fevers, without reference to differences between them, should come to an end on certain days rather than on others, so that when one critical day was passed, the disease might be expected to run on until the next critical day occurred. The whole of the doctrine in question is absolutely without foundation.

After defervescence, the temperature often remains for some days slightly subnormal. It is also less stable than in health, being easily disturbed by slight causes, so that, for example, a rise of two or three degrees is apt to follow the first solid meal.

It is often convenient to be able to express in general terms the degree of pyrexia, without giving the actual temperature. And for this purpose Wunderlich's classification may be adopted with advantage; it is as follows:

1. *Subfebrile*, temp. 99.5°–100.4° (37.5°–38° Cent.).
2. *Slightly febrile*, temp. 100.4°–101.3° (38°–38.5° Cent.).
3. *Moderately febrile*, temp. 101.3°–102.2° in morning; 101.3°–103.1° in evening (38.5°–39° Cent. in morning; 38.5°–39.5° Cent. in evening).
4. *Decidedly febrile*, temp. about 103.1° in morning, about 104° in evening (39.5° Cent. in morning, 40.5° Cent. in evening).
5. *Highly febrile*, temp. above 103.1° in morning, above 104.9° in evening (39.5° Cent. in morning, 40.5° Cent. in evening).
6. *Hyperpyretic*, temp. approaching 107.6° or even higher (42° Cent.).

The routine use of these expressions may, however, under certain circumstances, suggest a false impression as to the gravity of particular cases. In children, fever is set up very easily; an evening temperature of 105° may be due to comparatively trifling causes; and on the following morning the thermometer may not rise far above the normal point. So, again, sensitive women sometimes show a "highly febrile" condition, when the result proves that no apparently adequate cause for it has been present. On the other hand, in old people the temperature is apt to be below what one would have expected, from the gravity of the case.

With regard to the term, *hyperpyrexia*, I shall have to make some remarks further on. At what point it should begin to be used is somewhat uncertain; but it is generally understood to mean such a temperature as is sufficient of itself to endanger life, if continued for any length of time. Cases in which the thermometer rises to 109° or 110° (43.5° Cent.) are very exceptional, and most observers think that they are invariably accompanied by severe and alarming symptoms. Just before the death of a patient from tetanus Wunderlich obtained a temperature of 112.55° (44.75° Cent.); this is said, both by him and by Liebermeister, to be the highest reading that has hitherto been certainly taken.

On the other hand, there have, in this country, been recorded a few instances in which, if they can be relied on, it would seem that far higher temperatures have occurred, not only without being followed by death, but sometimes without being attended with any serious symptoms whatever.

The first case of this kind was observed by Mr. J. W. Teale ("*Clin. Trans.*," 1875), in the person of a young lady, who, by a severe accident, had several ribs broken, and afterwards suffered from great tenderness over the dorsal vertebræ. Two months later her temperature was, one day, taken at 110°; and afterwards the index of the thermometer was, on four occasions, buried in the bulb at the top of the instrument, at a point above 122°. Sometimes these extraordinary temperatures were taken in the axillæ, sometimes between the thighs, or even in the rectum. She was at

first in an exceedingly weak state, but she gradually improved and regained fair health.

Other examples of "paradoxical temperatures," as they have been called, have been met wit by Dr. Donkin ("*Brit. Med. Journ.*," 1879). His first case was that of a nurse, who was recovering from enteric fever, when the thermometer was found, one night, to register 110°. Afterwards very high temperatures were repeatedly taken, on a single occasion one of 111.6°, yet no symptoms could be discovered accompanying this reading beyond a feeling described by the patient as one of "flushing" or "rushes of heat." But perhaps the most singular circumstance of all was the evanescent character of this pyrexia, if it deserves that name; once the thermometer rose to 107.2° in the right axilla, whereas five minutes later it stood at 98.6°. In the mouth, a temperature of 106° was once observed. It does not appear that the instrument was ever held *in situ* while such extraordinary results were being obtained, but the patient's hands were watched, the idea of imposture being well kept in mind. Seven other cases are cited by Dr. Donkin, all but one of them being in females.

In 1879 a remarkable instance of this kind occurred in Guy's Hospital, under Dr. Moxon. The patient, a girl of 22, had been in the ward for phthisis during ten months, when, on the evening of July 25th, her temperature was taken at 107.4° and about an hour afterwards at 110.8°. She appeared to be suffering somewhat from dyspnœa. On the following morning the thermometer stood at 99.8°. During the next few months the most extravagant variations of temperatures were recorded. On one occasion Dr. Mahomed obtained, simultaneously, a reading of 102° in one axilla, one of about 114° in the other axilla, one of 107° in the mouth. On changing over the instruments, the highest temperature was attained in the axilla, where it had before been lowest, that of the mouth being now 104°. Another day a small registering thermometer gave 102.6° in one axilla, while another one in the other axilla gave 109.4°; but directly afterwards, when two large instruments without indices were used, and when the patient's arms were held all the time, the temperature stood at 103° on each side. Dr. Mahomed noted that the skin always felt moist and of the ordinary temperature, even when a very high reading of the thermometer had just been obtained. He never got a high temperature with a non-registering thermometer, when he himself held the instrument in the axilla, keeping his hand pressed against the patient's arm. It is certainly difficult to avoid the conclusion that in this case some deception was practiced, although its nature was never discovered. The girl died of disease of the lungs on March 22d, 1880. Can a patient, without being observed, squeeze or rub the bulb of a thermometer, so as to drive the mercury up?

In studying the nature of pyrexia we may, in the first place, safely assume that the *sources* of febrile heat do not differ in kind from those which normally keep the human body at a temperature above that of the external air.

In 1863 the late Prof. Traube, of Berlin, propounded the theory that the *quantity* of heat generated during fever is also the same as in health, and that, in fact, pyrexia consists in a diminution in the amount of heat given off from the body. His theory has since been shown to be incorrect, but it is right to add that it formed the starting point of very numerous observations and experiments, by which our knowledge has been immensely increased.

There would be no difficulty in attributing either a rise or fall in the temperature of the body to an alteration in the loss of heat, while its production remains unchanged. But in pyrexia, when the temperature has

risen to a certain point, it is often stationary there for some hours; and for several days it may oscillate upward and downward, always remaining above normal; exactly as in health it oscillates above and below 98.4°. Now, it is clear that such a maintenance of a fixed high temperature is of itself fatal to Traube's doctrine, as it would equally be fatal to any hypothesis that should attribute fever merely to an increased *generation* of heat. For, in order that the temperature should remain stationary, the amount of heat generated in a given period of time and the amount of heat lost in the same period must be equal. Thus it is certain that in fever, as in health, the regulation of temperature still goes on, although the point at which the thermometer stands is different. Liebermeister, in 1864, took advantage of an attack of tonsillitis in his own person, to demonstrate this fact very clearly. His temperature having risen to between 102° and 103°, he exposed his body to cold air, and washed himself with cold water; and he found that this caused a slight rise of the axillary temperature, from contraction of the superficial vessels and diminution of loss of heat from the skin, exactly as would have occurred under normal circumstances. The same observer, in 1868, by noting the changes in the temperature of the water of a cold bath, in which he placed a man suffering from acute pneumonia, was able to calculate that the immersion caused a greatly increased production of heat, far more heat being given off than would have corresponded with the mere lowering of the temperature of the patient.* So also, as Cohnheim remarks, one may throw a fever patient into the most profuse perspiration by the subcutaneous injection of pilocarpine, when the loss of heat from the cutaneous surface must necessarily be enormously increased, without causing any fall in his temperature.

Another striking illustration of the fact that the physiological process of heat regulation is still in action during the course of pyrexia, is afforded by the persistence of the regular daily fluctuations of temperature to which I have already more than once alluded. Wunderlich, indeed, formerly thought that the range of these fluctuations was wider than in health, even when the fever was such as would commonly be called continuous. But Jürgensen and others seem now to have shown that they are identical with the normal fluctuations in every respect except that they take place at a higher thermometric level. In pyrexia, as in health, the rule is for the temperature to rise more or less constantly during the day, and to fall during the night. The minimum occurs at about 6 or 7 A. M., the maximum at about 6 P. M. The range of fluctuation appears to be generally as much as 1° C., or even a little more, so that it approaches 2° Fahr.

According to Liebermeister, these daily curves of temperature are probably due to the influence of muscular exertion and of food upon the body, notwithstanding, certain obvious objections to such an interpretation of them. One of the objections is that healthy men work and eat to a much later hour in the day than 6 o'clock, P. M., after which the temperature begins to fall; this Liebermeister meets by what is termed the "principle of compensation," according to which every rise in temperature tends to be followed, after an interval, by a fall below the normal point; he supposes, in fact, that the power of food and of exertion to raise the bodily heat is exhausted during the early part of the day, after which the inevitable recoil takes place, in spite of them. The other objection is that the daily fluctuations do not disappear when a person remains in bed all day, and takes no food. Liebermeister accounts for this by appealing to the influence of habit. Evidently the persistence of the fluctuations during pyrexia, when the patient is perhaps absolutely helpless and has fluid nourishment at regular intervals through

* The accuracy of this calculation has since been disputed by Winternitz and others.

the twenty-four hours, requires and admits of the very same explanation. It seems to me, therefore, that Liebermeister fully establishes the position which he takes up, namely that pyrexia consists, not in a mere rise of the temperature of the body, still less in increase of heat generation or in diminution of loss of heat, but in a change in the normal function of heat regulation, by which the production of heat and its loss are so balanced as to create and maintain, while the pyrexia lasts, a higher temperature instead of the normal temperature. One might imagine the index of the regulating machinery to be shifted upward, so that it is "set," not at 98.4°, but at 101°, 102°, 103°, or even a still higher point. Cohnheim, indeed, rejects this view, summarily and almost with contempt, evidently imagining that it contains only a verbal solution of the problem, but in reality (as I think) failing to appreciate what the problem is. Of course, we are at once met by the difficulty that of the nature and seat of the function of heat regulation we have no certain knowledge, although its existence is indisputable. Liebermeister's notion is that somewhere in the upper part of the cord there are two centres, more or less closely connected, but opposed in their action, one of which is termed by him the "excito-caloric" system, the other the "moderating" system. It must, however, be allowed that the stability of the process of heat regulation in fever is by no means so great as in health.

The view that pyrexia is a definite modification of a physiological process accords well with a doctrine which is now gaining ground, and which seems likely to bear valuable fruit, namely, that this morbid condition fulfills a purpose in the economy, comparable with that which we shall see to be fulfilled by inflammation. The conception of fever as having a salutary influence is, indeed, as old as Hippocrates, and it can be traced all through the middle ages, and down to our time. But our immediate predecessors recoiled with terror from all explanations that could be called "teleological." And it is only of late, since the contagious principles of some at least of the specific diseases have been shown to be living organisms, that it has been possible to understand how pyrexia may bring about its own cure, by destroying the very agents which set it up. In discussing Relapsing Fever, Ague, and Enteric Fever, I shall give evidence that this is the case.

The chief conditions under which pyrexia occurs fall naturally into two groups: sometimes it is "symptomatic" or secondary to a local inflammation; sometimes it is "idiopathic" and "essential," and depends on the entrance of a contagion into the blood. Now, recent researches seem to lead to the conclusion that "symptomatic" fever may likewise serve the purpose of freeing the blood from living organisms. It was experimentally shown by Breuer and Chrobak that the division of the nerves of the limb of an animal has no effect in preventing the development of fever as the result of inflammation in it. The inference seems to be inevitable that inflammatory fever is the result of the entrance of a morbid agent into the blood from the inflamed tissues, and this entirely accords with the observations of Billroth and Otto Weber, who, so far back as 1864, showed that pyrexia could be produced by injecting into the blood either fresh pus or decomposing substances of various kinds. With regard to the nature of the "pyretogenous" material in symptomatic fever there is more uncertainty. At one time the prevalent view was that it is possibly excited by the products of inflammation when they have re-entered the blood, either directly or through the lymph stream. But at present the more usual opinion is that local inflammations are themselves set up by the entrance of a poison from without, and that when this is the case the same poison making its way into the blood sets up the pyrexia.

One must, however, observe that the fact that a particular disturbance of a physiological function can, in certain cases, be shown to be for the benefit of the economy, is no reason whatever why the same kind of disturbance should not arise in other cases when no purpose is served by it. If there be centres for heat regulation in the cord, nothing is more likely than that they should be modified in their action by cerebral hemorrhage, or the status epilepticus. And, on the other hand, there is sometimes an absence of pyrexia as an accompaniment of intense inflammations (especially of the meninges and of the peritoneum), which can hardly be accounted for except on the supposition that some counter influence is exerted upon these centres, either directly or through the sympathetic or other nerves.

The irregular fever of idiopathic anæmia, of leucæmia, and of Hodgkin's disease has not yet received a satisfactory explanation; nor has that which accompanies the various forms of acute tuberculous affections.*

According to Liebermeister's definition of fever, the mere fact that the temperature of the body is raised is not of itself a proof that fever is present. In Tetanus the temperature is sometimes exceedingly high. But Dr. Parkes, in his Croonian Lectures for 1871, suggested that the heat of the body in that disease might perhaps owe a different origin from that of the specific and symptomatic fevers. A similar opinion has recently been expressed by Cohnheim, who would also exclude from pyrexia the phenomena of "heat-stroke." In each case it is probable that what occurs is not a shifting upward of the point to which heat regulation is adjusted, but a generation of heat in excess of that with which the process of heat regulation is able to deal. The same view may perhaps be taken of hyperpyrexia in general, the proof of its correctness being that the temperature never remains stationary at a very high point, and does not continue to oscillate backward and forward, but either goes on rising until death occurs, or falls again to a moderate level.

Physiological Course of Fever.—As we have seen, there is no ground for the belief that in pyrexia either the amount of heat generated or that which is lost is in any way fixed. Probably each of them may vary within very wide limits in different diseases, or in different periods of the same disease. All that is necessary is that they should constantly bear such a relation to one another, that the bodily temperature is at first raised to, and afterwards maintained at, a point more or less above the normal.

Now, in the *initial stage* of fever, there can be no question that the loss of heat from the cutaneous surface is greatly diminished. And as there is no reason whatever for supposing that the evaporation from the lungs undergoes a proportionate increase, we are probably justified in assuming that the total loss of heat is much less than under ordinary circumstances. Since the introduction of the thermometer into clinical practice, writers seem to have fallen into a most extraordinary mistake with regard to the surface-temperature during rigor. We have seen that, as De Haen long ago pointed out, the temperature of the deeper parts of the body is actually higher than normal, and, in fact, rises rapidly. Now, this has commonly been taken as proof of an altogether false conclusion, namely, that the coldness of the limbs and of the skin generally, which gives the patient so much suffering, is merely a "subjective sensation produced by the state of the peripheral nerves." Of course, the truth is that the low temperature of the surface is just as much a physical fact as the high temperature of the blood in internal organs. One has only to feel the shrunken hand of a man in the

* I may take this opportunity of remarking that it is very advisable to give up describing "hectic" as an independent variety of fever; such peculiarities and symptoms as belong to the pyrexia of phthisis will be mentioned when that disease is under discussion.

cold stage of ague, to be satisfied that he is under no illusion when he complains of being cold; and a surface thermometer affords precisely the same indication by the slowness with which it rises above the temperature of the surrounding air, and the comparatively low point which it reaches, however much time may be given to it. Thus Schülein, among certain observations of surface temperatures to which I shall presently allude, gives a case of tertian ague; and in his chart it is most striking to notice how, during each attack, at the precise moment when the temperature in the axilla was rising from 98° to 104° or 105° F., that between the toes fell still more sharply, from 95° to 86°, or even below 77°. The cause of the chilliness experienced by such a patient is that the peripheral arteries are contracted and do not allow of the passage of a sufficient quantity of blood to warm the superficial tissues, and to compensate for the loss which is always going on by radiation and conduction, to whatever extent the exhalation of fluid from the skin may be diminished. A necessary consequence is a great fall in the temperature of the skin, and one must not forget that this in turn involves a lowering of the amount of heat loss, which affords one of the means by which the rapid rise of temperature during rigor is brought about. It has, however, been calculated by Zimmermann and by Liebermeister that there is, besides, a very great increase of heat production during this stage.

During the *fastigium* the temperature of the body may remain stationary, or at any rate undergoes comparatively gentle oscillations, so that for its maintenance no such antagonism between heat production and heat loss is required as for the rapid rise in the initial period. However, in many instances there is still a marked distinction between the temperature of the skin and that of the deeper structures. These cases afford special proof of the importance of the clinical thermometer, inasmuch as the axillary or the rectal temperature is found to be above normal while the patient's skin feels cool to one's hand and he has no sensation of feverishness. But, of course, the comparatively low temperature of the surface is a reality; and since the heat loss by conduction and radiation is certainly less than when the skin is hot, it is fair to conclude that for the same degree of internal temperature the production of heat must also be less. On the other hand, there are certain diseases in which, when one places one's hand upon the patient's skin, one feels it to be pungent and burning in a very remarkable degree. Addison used to teach that in acute pneumonia the skin possessed a heat which was not observed in any other disease except perhaps scarlet fever. And I well recollect how disappointed I was when the thermometer was introduced into practice to find that his dictum seemed to have no physical foundation. An attempt was made to explain away the difficulty by saying that the surface was peculiarly dry in acute pneumonia; but this obviously was beside the question, since at any temperature above that of one's own hand a moist skin must undoubtedly feel hotter than a dry one, if it were possible to prevent the temperature from being lowered by the evaporation which inevitably occurs. The true solution of the difficulty is that in acute pneumonia the temperature of the surface is maintained at a point which is very nearly as high as that of the deeper parts, instead of being far below it, as in most other diseases, even when there is no moisture upon the skin. This has recently been established by Schülein, as the result of a series of careful observations published in "*Virchow's Archiv*" for 1876, upon the relation between surface temperature and internal temperature in various diseases. His method was to insert a thermometer with a very small bulb between the first two toes, fastening them together by means of an elastic ring. And he found that whereas in other diseases (including enteric fever, acute rheumatism, and phthisis) the instrument in this position always indicated a much lower temperature than one placed in the axilla, there were three diseases in

which the difference was very slight indeed, namely, acute pneumonia, measles and scarlet fever. In these almost every movement of the axillary temperature upward or downward was accompanied by precisely the same movement of the temperature between the toes; whereas, no such relation could be traced between the two sets of observations in any other febrile complaint. It must, however, be remarked that in many diseases surface temperatures are from time to time taken which are absolutely as high as those in acute pneumonia; and it is probable, as I have taught for a long time in my lectures, that a still more striking confirmation of Addison's doctrine would be obtained if we were only to note the rapidity with which a surface thermometer rises when applied to the skin in different complaints. For in that way we should be estimating the amount of heat which the surface is giving off by conduction; and it is this that one's hand appreciates when laid upon a patient's skin. I have no doubt, however, that at least one other disease would have to be added to those enumerated by Addison, namely, acute rheumatism when complicated by hyperpyrexia. It is evident that in order to maintain such a uniform high temperature of the superficial as well as of the deeper parts, in spite of the great loss of heat which must necessarily occur, the generation of heat must be far greater in all these complaints than in others in which only the interior of the body is raised to a similar degree of heat. So, also, when the skin perspires freely during the *fastigium*, the temperature within remaining high, there can be no doubt that the production of heat must be proportionately increased.

Of the stage of *defervescence*, I have only to remark, that the profuse sweating which so often accompanies, when crisis takes place, plays an important part in bringing about the rapid fall of internal temperature. But it is not to be regarded as the cause of the defervescence; the fact being that the state of pyrexia has come to an end, and that the heat regulation is, therefore, no longer set for an abnormally high point.

Concomitants of Fever; Tissue Change and Wasting.—Liebermeister's theory of fever, which I have adopted, renders it almost unnecessary to enter into any detail as to the sources of febrile heat, these being in fact identical with those which are enumerated in works on physiology as maintaining the normal temperature of the body. But I must not omit to point out that science has now advanced far beyond the point at which Virchow stood when, in 1854, he declared that the elevation of temperature in pyrexia "must arise from an increased tissue change." Liebermeister and other observers have indeed shown that the amount of urea excreted in the urine during fever surpasses by at least 70 per cent. that which is voided by a healthy person living on the same diet. But it is now well known that the production of heat may be largely dependent upon an increased oxidation of various substances, such as sugar, which have never formed part of the substance of the body. And both Leyden and Liebermeister have proved that the excretion of carbonic acid gas also is excessive in fever, the increase amounting, probably, to at least 50 per cent. It is essential to bear in mind, however, that neither the quantity of urea, nor that of carbonic acid, is nearly so great as that which may be discharged, without any rise of temperature, by a person who is taking active exercise, or even by one who has eaten a large meal of animal food with much fat. Thus we are brought back again to the same point as before, namely, that pyrexia cannot be due to any special rate of heat production, but must depend upon an alteration in the point for which heat regulation is set.

There is, however, no more striking feature of pyrexia than the wasting of the body which accompanies it; and this often seems to go on quite as rapidly as one could have expected, even if Virchow's dictum had been true

in the strictest sense. Formerly there was a tendency to pay too little attention to fever as being, in itself, a cause of loss of weight, and to ascribe this result rather to a drain of pus or of any other discharges which might occur in a particular case. It may therefore be worth while to cite a case of Liebermeister's, in which a patient, who had an abscess discharging about three ounces of pus daily, lost from fifteen to twenty pounds in weight during two months, while his temperature was at 101° or 102°, but regained it afterwards when he had no fever, notwithstanding that the suppuration still went on as before. No doubt an inability to digest food plays an important part in bringing about the wasting; it is well known that the secretion of milk ceases during pyrexia, and one may fairly assume that there is a similar interruption in the formation of saliva (to which, in part, the dryness of the mouth is due), and also of the gastric and intestinal juices. That it is not merely a question of loss of appetite, appears probable from an observation of Niemeyer's, who, comparing together a healthy man and one suffering from fever during a period of two days, in which they lived on exactly the same food, found that the latter lost much more weight than the former. Liebermeister remarks that the wasting is sometimes concealed by the presence of an excessive amount of water in the tissues; he has observed that from this cause the weight of the body may be raised by as much as ten pounds, although there may be only a very slight œdema of the ankles to indicate the presence of such an abnormal condition. He believes that persons who are in good health and well nourished invariably lose flesh when they become the subjects of pyrexia; but he admits that those who are already wasted may undergo no further emaciation, and may even gain weight, in spite of the supervention of a febrile illness. According to a calculation made by Leyden, the daily loss of substance in fever amounts on an average to 7 parts per 1000 of the whole body weight. But, as Cohnheim has pointed out, if this be correct, it should take only about eight weeks of a tolerably severe fever to kill a fairly-nourished man by the mere destruction of his tissues; at least, if one may appeal to the observations of Chossat, which showed that to the higher animals a loss of 40 per cent. of their weight is directly fatal.

The blood doubtless wastes simultaneously with the solid structures, and Parkes and others have supposed that the excess of pigment in the urine depends upon an undue disintegration of red discs. But hæmometric observations which have been made by Drs. Baxter and Willcocks, lead to the conclusion that, with a single exception, febrile diseases cause no diminution in the proportion, either of discs or of hæmoglobin, in a given amount of blood. The exception is ague, in which Kelsch is said to have demonstrated a very great decrease of corpuscles. In all other febrile states the whole volume of the blood is lowered, while the relation of its fluid or solid constituents remains normal. If so, a point on which Dr. Parkes used to insist can hardly be maintained, namely, that there is a special retention of water in the body during fever.

Increased Rapidity of Pulse.—We have seen that it is only of late years that elevation of temperature has been recognized as the characteristic and essential feature of pyrexia. Previously, acceleration of the pulse, rapidity of breathing, diminution of the secretions, and a variety of other phenomena, were supposed to be not less important indications of it. Now, it is a matter of some consequence to determine how far these several symptoms are directly dependent upon the increased heat of the body, or whether they must be regarded as independent effects of the various causes of fever. And in the opinion of the best modern writers, the rapid pulse which generally accompanies pyrexia is a result of the heightened temperature; Cohnheim adduces in support of this view the well-known fact that the frog's heart beats more

quickly in proportion to the warmth of the surrounding air; Liebermeister insists on the effect which hot and cold baths respectively have, when they raise or lower the temperature of the body, of simultaneously accelerating or slackening the pulse. Liebermeister even lays down as the rule, based upon a considerable number of observations (which, however, vary widely among themselves), that for each degree Centigrade above the normal temperature there should be a rise of the pulse by eight beats above the standard rate of eighty. It is admitted, of course, that various other circumstances may affect the pulse rate of febrile patients, as of healthy persons, or of those suffering from other diseases. Cohnheim insists on the effect which stimulation of the vagi may have in slowing it, and cites the fact that in basilar meningitis the pulse is often infrequent, in spite of high fever. But neither he nor Liebermeister seem to have looked out for those negative instances by which their theory could best be brought to the test, namely, the exceptional cases in which a severe inflammatory process, or some other condition that usually gives rise to pyrexia, happens to run its course without elevation of temperature. Such instances are sometimes met with in peritonitis, in diphtheria, and in meningitis; and I think that the pulse is often, though not always, quickened. Does not this fact, if it be one, cast some doubt on the view which the German authors adopt?

The increased frequency of the breathing in pyrexia is also attributed to the action of heated blood upon the respiratory centre; and this view is supported by a very interesting experiment, performed by Goldstein in Fick's laboratory, of warming the carotid blood alone; he found that the respiration was thereby accelerated. Cohnheim adduces in illustration of this theory, the fact that in acute pneumonia the breathing ceases to be rapid after the crisis of the fever, notwithstanding that the affected part of the lung still remains hepatized.

On the other hand, this writer is indisposed to admit that the cerebral symptoms which attend so many cases of fever are dependent solely upon the increased temperature of the blood and of the substance of the brain. He points out that in relapsing fever the thermometer rises to a great height with but little disturbance of the sensorium, and that in febrile diseases generally no relation can be traced between the intensity of the fever and the severity of the delirium or stupor. He does not even allow that the question is settled by the marked effect of a cold bath in restoring the mental faculties of a patient suffering from fever; for, he observes, it may be a consequence rather of the improvement of circulation which results from the bath.

It is important to observe that the rapidity with which the blood flows in pyrexia, and the arterial blood tension, are by no means proportioned to the acceleration of the heart's action. At the commencement of fever, when the pulse feels full and hard, it is probable that the circulation is often really more active than in health. Much stress was formerly laid upon the "*sthenic*" character of such cases. In the "*Med. Times and Gaz.*" for 1873, Dr. Mahomed has given sphygmographic tracings of the pulse in various forms of fever, showing that this term is not without meaning; for in certain cases the vessels are full, the arterial tension is high, and the pulse bears considerable pressure. It is, therefore, a mistake to say that a febrile pulse is necessarily dicrotic. But, on the other hand, when pyrexia has lasted for any length of time, it almost always assumes an "*asthenic*" type, and in many instances this is the case from the very first. It would then seem that the circulation is more sluggish than natural, one proof of this being that the blood tends to stagnate in the more dependent parts of the body, so that what are termed hypostatic congestions arise in the lower lobes of the lungs, and beneath the

integument of the back, of the gluteal region and elsewhere. The pulse still remains quick, but it is now soft, feeble, and often small. The sphygmograph shows a highly characteristic condition of arterial relaxation, with low pressure; dicrotism becomes a prominent feature; and in many cases the pulse is even hyperdicrotic. In fact, at this stage of fever the heart, although its beats follow one another so quickly, empties itself incompletely, and discharges less rather than more blood into the arteries. Often, indeed, thrombi form in those parts of the heart's chambers which are furthest from the orifices; but this is a matter into which I shall have fully to enter hereafter.

Until recently, the impairment of the cardiac beats was held to be sufficiently explained by the fact that its muscle, like all the other tissues, becomes badly nourished as pyrexia goes on. Of late, however, some observers have thought that the cause of the enfeeblement of the heart's action in pyrexia is not merely its wasted condition, but the occurrence of definite morbid changes in its texture. When I come to speak of enteric fever I shall have to describe an affection, known as "parenchymatous degeneration" or "cloudy swelling," which is found in the liver, in the kidneys, in the heart, in the voluntary muscles, and elsewhere. This, however, is by no means peculiar to any one febrile disease, and Liebermeister and others have maintained that it is the direct result of elevation of the body heat, reaching a certain degree of intensity, and protracted over a somewhat long period of time. This view is supported by the experiments of Dr. Wickham Legg, who found ("*Path. Trans.*," Vol. xxiv) that the liver, the heart, and kidneys were granular in animals killed by the action of heat, even when its duration was only for a few hours. On the other hand, Cohnheim maintains that, although guinea pigs kept for several days in a high temperature exhibit a fatty change in their tissues, there is nothing really resembling "cloudy swelling." And he insists on the absence of such an appearance in acute pleuro-pneumonia, and its occasional presence when there has been no fever (as in carbonic oxide poisoning) as proofs that Liebermeister's opinion is erroneous. He even declares that the statements as to the frequency of degenerative changes in febrile diseases are overdrawn, and that in a large majority of the bodies of those who have had intense pyrexia, fatty gland cells and muscle fibres are not at all more common than they would have been if death had taken place in a different way. The question is one of great importance, and I shall have to revert to it hereafter in discussing the supposed causes of cardiac dilatation and of permanent failure of the muscular substance of the heart.

I shall not attempt in this place to discuss the *treatment* of pyrexia by baths and "antipyretic" remedies. Enteric fever and acute rheumatism afford by far the most frequent occasions for their employment, and it will be more convenient, when I am describing these diseases, to go into the full details which are necessary.

Subnormal Temperature.—It is an interesting question whether the temperature of the body ever undergoes a change in the direction opposite to pyrexia, the heat regulation centre being set at a point below the normal instead of above it. I shall hereafter have to cite many instances in which a thermometer placed in the axilla remains unduly low, but probably in these cases the internal organs generally maintain a temperature nearly as high as ever, at least until death is obviously impending. Cohnheim says that this is the case with those who suffer from inanition as the result of stricture of the œsophagus, from diffused paralysis, or from extreme anæmia. And even were it not so, we should still have to show that an

alteration in the heat regulation had occurred, and that the failure to maintain the normal temperature was not a result of an insufficient production of heat. Such a lowering of temperature by "*force majeure*," as Liebermeister terms it, is well seen in persons who are picked up insensible in the streets during cold weather. Cohnheim alludes to cases of this kind, in which the thermometer in the rectum has not risen above 86°, 79°, or even 75° F. The pupils are then dilated and sluggish, while the pulse and the respiration are greatly reduced in frequency; but, unless things have gone too far for recovery to take place, such patients, under suitable treatment, regain their normal temperature within a few hours, or even pass into a condition of reactive pyrexia.

INFLAMMATION.

HISTORICAL REVIEW: VIRCHOW: WILLIAM ADDISON, WALLER AND COHNHEIM: GOODSIR—DIRECT OBSERVATION OF INFLAMMATION IN ANIMALS—REDNESS, SWELLING, PAIN, HEAT, PYREXIA—EXUDATION: MEMBRANOUS, CATARRHAL, PURULENT, DIPHTHERITIC, GANGRENOUS INFLAMMATION—REPAIR—CAUSES OF INFLAMMATION: IRRITANTS AND PREDISPOSITION—CHRONIC INFLAMMATION.

No one, at whatever period of the world's history, could have applied himself to the study of disease, without seeing that the hot, painful, reddened state of the eye which follows the entrance of a particle of dust within the lids, is typical of a morbid change that is of frequent occurrence in every part of the body accessible to observation. Hence, it is not at all surprising that before the end of the second century we find, in the writings of Galen, inflammation (φλεγμονή) already characterized by its four cardinal symptoms, which, for so many generations since, have been known as *calor*, *rubor*, *tumor*, *dolor*. To use the words of the late Prof. Hughes Bennett, inflammation has been, in all ages, "the pivot upon which medical philosophy has revolved." But, for that very reason, there is but little use in our now attempting to recapitulate the different views and theories, with regard to it, which have been proposed. It is scarcely too much to say that only the most recent advances of pathology and histology have rendered possible a real understanding of inflammation.

Very soon after the promulgation of Schwann's doctrine, in 1839, that nucleated cells constitute a principal element of animal structures, pathologists discovered the presence of these bodies in inflamed parts; but, at first, it was supposed that they developed themselves in the interstices of the tissues, by molecular aggregation, in accordance with the revived opinion that this was a common mode of origin for cells in general. In his "Cellular Pathology," however, published in 1858, Virchow laid down, as a dogma, *omnis cellula e cellulâ*. And upon this foundation he erected a complete theoretical system, assuming that the cells of the connective tissues throughout the body undergo multiplication or "proliferation," so as to generate the "exudation cells" or "pus cells" of inflammation.

Even before Virchow's views were made known, certain observations had already been recorded in this country, which, although they at first attracted little or no notice, nevertheless involved conceptions as to the real nature of cells that were far higher, as they have since proved to be more accurate, than his. But the first shock to the edifice which Virchow had so carefully erected was given by one of his assistants, Recklinghausen. He showed, in 1863, that pus cells, instead of being rounded bladders, each with its cell wall and its nucleus, are, during life, solid masses of gelatinous substance, which are constantly changing their form—now throwing out delicate processes, and now drawing them in again—and that they actually possess the power of moving from place to place. Of course, this discovery at once deprived Virchow's proliferation theory of the impregnable position which it had seemed to hold as a necessary consequence of the law *omnis cellula e cellulâ*. Four years later, in 1867, Cohnheim, who also was a pupil of Virchow, pointed out that in the mesentery of the frog, when it becomes inflamed under exposure to the air, the white corpuscles of the blood can, with the aid of

the microscope, be distinctly seen to make their way through the walls of the smaller vessels, so as to become pus cells. This very fact, however, had been demonstrated as far back as 1846, by the late Dr. Augustus Waller, then in practice at Kensington. His investigations were made on the tongue of the frog. Still earlier, in 1843, Dr. William Addison, of Great Malvern, had insisted on the transformation of the colorless corpuscles of the blood into pus cells, and described the former not only as "lining the vessels" in the web of the frog when inflamed, but also as "lying among the fibres forming their walls and exterior to their boundary." But, in truth, as Dr. Waller himself remarks, such a conception was even then not unheard of. Gendrin, in 1826, suggested that "the globules of blood are decolorized and excreted in the form of pus." The recent resuscitation of views which for so long a time appeared to be incompatible with common sense, should make us cautious in rejecting any statements that are based upon direct observation, however much they may clash with our preconceived opinions.

In those countries in which Cohnheim's experiment is not forbidden by law, it is performed in the following manner: A male frog, which has been rendered quiet by curare, has an incision made into the abdominal cavity, large enough to allow a loop of intestine to be drawn out with its mesentery. It is then placed upon its back, on a broad glass plate, which has been fitted with a thin glass disc for the convenient reception of the mesentery, and with a semicircle of cork to which the intestine is to be pinned. Under the microscope, the circulation can now be seen to go on naturally. But the contact of air appears to act as an irritant, and inflammation soon sets in. In from fifteen to twenty minutes the arteries are observed slowly to dilate, until at the end of an hour or two their diameter is nearly twice as great as before. A little later a similar change begins in the veins. At first the blood stream perhaps flows more quickly than before, but very soon its rapidity begins to diminish, so that the oval red blood-discs can be distinctly seen in the arteries, at least during the diastole. A striking change is now noticeable in the veins. Leucocytes begin to fall out of the middle of the current and loiter against the sides of the vessel, rolling over and over, and sticking from time to time, when their soft substance yields to the pressure of the fluid upon them, so that they become pear-shaped. Before long, the channel of the veins becomes lined by a thick layer of these bodies, which altogether cease to move, while the central current of red discs goes on as before. In the capillaries, too, the leucocytes are arrested here and there; but between them there are seen many red discs also adhering to the walls. But the most wonderful thing of all is that outside the contour of these vessels there gradually appear a number of minute rounded bodies, each of which is connected with a leucocyte within. Thus the leucocytes may be said to have assumed a dumb-bell form. By an imperceptible process, the extra-vascular parts of them keep growing bigger, the intra-vascular parts smaller, until at length the latter altogether disappear, whereupon the leucocytes resume a more or less globular shape: they now lie close to the vein, but outside it. In the capillaries red blood-discs, as well as leucocytes, pass out. The result is, that large numbers of leucocytes are soon collected, not only in the immediate neighborhood of the vessels, but in all the interstices of the surrounding tissues. At the same time a quantity of fluid exudes, which reaches the free surface of the mesentery and coagulates there, forming a membranous layer, itself thickly set with leucocytes.

The separation of the leucocytes from the red discs flowing through the mid-channel of the veins, and their loitering against the sides of these vessels and of the capillaries, are direct physical results of the slowing of the blood-current. The process of *emigration* must be referred to the power

of spontaneous locomotion possessed by the leucocytes themselves. It seems, however, to depend to a certain extent upon their being under pressure from the blood, for Cohnheim has observed that it instantly ceases when the flow of blood through the main artery of the part is arrested. But, on the other hand, it is not due solely to blood pressure, for this is actually less than under normal conditions where no emigration seems to go on, at least in the case of tissues which are not in the embryonic state. My colleague, Dr. Laidlaw Purves, and Prof. Julius Arnold, of Heidelberg, have maintained that the points at which leucocytes emerge from the vessels are always situated in the meeting lines between the endothelial cells. But Cohnheim remarks that, at any rate, actual orifices or stomata can hardly be present, since they must necessarily give exit also to an unaltered liquor sanguinis, whereas the fluid which exudes during inflammation is of different chemical composition, and contains less solid matter in solution. It has been suggested that emigration may be considered to take place without any breach in the vascular walls, if they and the leucocytes be regarded as colloid substances, and as becoming temporarily continuous with one another; and I am one of those who think that this view is fairly illustrated by the experiments of Dr. Norris, who has shown that if a film of soapsuds be stretched across an iron ring, a moistened glass rod, or even a spherical soap bubble, can be passed into it on one side and brought out on the other, leaving it unbroken.

Before the time of Virchow, a favorite topic of speculation had been whether parts into which no vessels penetrate could be said to undergo inflammation. And one of the merits of his system was that it placed the question on its right footing by showing that the distinction between vascular and non-vascular tissues was, after all, only one of degree, and that a minute islet of liver substance ought to be regarded as being outside the blood current, no less than the cornea or the cartilage of a joint. Nor did Cohnheim's investigations modify this conclusion, but rather carried it a step further; for he actually proved that leucocytes could penetrate into the interior of the cornea from the blood. It had been shown, by Von Recklinghausen, that when finely divided vermilion was injected into a lymph cavity in the frog, the leucocytes would take this substance into their interior, or "feed" upon it; and Cohnheim found that after injecting pigment into one of the aortæ many of the leucocytes which appeared in the cornea under inflammation were colored, so that they had evidently been derived from the circulatory fluid. Still, however, it could be maintained that others of them were formed by proliferation. This view was, and is still, vigorously upheld by Stricker, who described the fixed corpuscles of the corneal tissue as breaking up into amœboid masses, containing bodies which he believed to be the germs of future pus cells. But other observers afterwards pointed out that by more refined histological methods the stellate corpuscles could be seen in a cornea which was already full of leucocytes; and although it seems to be a fact that their nuclei do undergo division, this is probably an indication (as Dr. Thin has suggested) of commencing disintegration rather than of germination. The observations which Cohnheim, writing in 1877, deemed most worthy of notice, as running counter to his own, were some which had been made by Böttcher. He cauterized the minutest possible spot in the centre of the frog's cornea, and showed that pus cells were to be found in the neighborhood of the injured part, whereas there was no marginal opacity such as would have indicated that they had come from the conjunctival vessels. Cohnheim's rejoinder was that they might have been derived from the fluid secretion which covers the free surface of the eyeball. And this, which at first seemed to me a very far-fetched solution of the difficulty, appears to have been since established by the experiment of placing a

dead cornea, in which a puncture has been made, into a healthy conjunctival sac for a few hours; "it remains transparent," says Dr. Sanderson in his Lumleian Lectures for 1882, "everywhere excepting in the neighborhood of the injury," so that a breach of surface is evidently all that is necessary to enable leucocytes from the conjunctival fluid to make their way into corneal tissue. It has also been shown that a limited part of the cornea may be destroyed without any opacity of the adjacent tissue resulting, if the caustic employed be chloride of zinc, which does not disintegrate the protective epithelial covering.

To me it seems impossible to obtain an adequate conception of inflammation, without taking into account its relation to the physiological process of repair. By Dr. Sanderson himself it is defined as the "physiological effect of the damage done to a tissue when it is injured." He, however, goes on to say that its "essential nature is fully expressed in the word *damage*," and I do not think that he anywhere suggests that a useful purpose is served by the emigration of leucocytes. Yet to call a process physiological is surely to imply that it is likely to be beneficial to the organism rather than the reverse. Pathologists have indeed been accustomed to describe the healing of wounds and the restoration of the substance of injured parts, as not only independent of, but even opposed to, the occurrence of inflammation in the same structures; and so long as the pus cells found in an inflamed area were supposed to be formed by the breaking up of the fixed elements of the tissues in proliferation, it might well seem that such a process was altogether different from the suppuration which was described as constituting one factor of "repair by granulation." But now that the leucocytes are believed to be always derived from the blood, it is obvious that no such distinction can be drawn.

The solution of the difficulty seems to me to lie in the fact that when a part is at all severely injured, the removal of damaged tissue elements is an essential preliminary to the work of reconstruction and repair. I have for some years past ventured to teach that the emigration of leucocytes in inflammation serves this especial purpose of clearing away such portions of an injured structure as are no longer fit to remain. We have seen that leucocytes are capable of taking up granules of vermilion; and it is well known that they also "feed" upon milk globules, or particles of myelin, when those substances are placed in their way. May we not assume that they can also remove damaged cells or fibres? I did not know, until recently, that this very doctrine was promulgated forty years ago, by the late Prof. Goodsir. In his "Anatomical and Pathological Observations" (1845) speaking of "a rapidly extending ulcerated surface," he described "a peculiarly endowed cellular layer, which takes up progressively the place of the subjacent textures;" and a little further on, he attributes the separation of a dead portion of bone to the "cells of the newly-formed cellular mass," contained in the Haversian canals . . . . taking it up as nourishment, and substituting themselves in its stead."* The same explanation is obviously applicable to the detachment of a slough in the softer tissues, and perhaps even to the formation of an abscess cavity, and to the thinning of the roof of an abscess which is about to "point." We may also have recourse to it when we find that, in certain parts of a solid organ, a dense infiltration of leucocytes is attended with the disappearance of the normal structure, although there is no obvious gap or breach of continuity: this, for example, may often be observed around the Malpighian capsules of the kidney, when it is in a state of inflammation.

Such an hypothesis seems to me far more satisfactory than the view of

* See pp. 404, 406 of the second volume of Goodsir's Memoirs, republished by his successor, Prof. Turner, of Edinburgh, in 1868.

Cohnheim, who appears to place the inflammatory process on a merely physical basis: ascribing it to a molecular change in the walls of the blood vessels, wrought directly by the irritant which sets up the inflammation. In support of this view he cites the experiments of Winiwarter, who found that the vessels of an inflamed part allowed a solution of gelatin to transude through them under a lower pressure than under normal conditions. But I think that the change in question is only a part of the general softening of all the tissues, which has long been known to constitute one of the phenomena of inflammation. An acutely inflamed bone is, as Paget points out, so soft that a knife will easily penetrate it; the ligaments of an inflamed joint yield, allowing the bones to be displaced and distorted; and we are accustomed, in the dead-house, to regard a diminished resistance of the texture of a lung as one of the best rough tests of it having been the seat of acute pneumonia.

Before we quit the study of inflammation in cold-blooded animals, I have to mention some further phenomena which belong to its more severe forms. When croton oil is smeared over the tongue of a frog, the superficial capillaries pass into a condition which has long been known as *stasis*. It consists in complete arrest of the circulation through them, with coagulation of the blood which they contain; and it corresponds with the condition which in larger vessels is called *thrombosis:* in fact, the two are often associated together. But a still better way of observing severe inflammation is to apply caustic to a circumscribed spot on the tongue of the frog. This produces what may be described as a series of concentric zones. In the middle all the tissues are killed; or, in other words, an *eschar* is formed. Next comes an area of absolute stasis, in which, according to Cohnheim, the vessels alone are destroyed. Further outward, the blood stream is almost stagnant, and the capillaries are choked with red discs, which escape from them in large numbers. Beyond this again the emigration of leucocytes is the principal change. Last comes a zone in which the vessels are merely dilated, while the circulation through them is somewhat retarded. However, it is not clear to me that stasis has been proved always to depend upon the occurrence of alterations in the vascular walls, exceeding in intensity those in the other tissues. At any rate, no such conclusion can fairly be drawn from the well-known experiments made by Ryneck, of Gratz, in 1870. He showed that stasis could be set up by irritation in the web of a frog, after defibrinated mammalian blood, or even milk, had been substituted for the natural circulating fluid of the animal; but that, on the other hand, when a solution of chromic acid or sulphate of copper had once been passed through the blood vessels, even for a few moments, no such effect could be produced. But these observations, although they disproved the now obsolete doctrine that inflammation is dependent upon a change in the blood, appear to have little or no bearing upon the questions which are under discussion at the present time.

In warm-blooded animals the attempt to watch the inflammatory process directly with the microscope was at first found to be attended with considerable difficulties. But these have now been overcome; and in "*Virchow's Archiv*" for 1878, Prof. Thoma, of Heidelberg, describes a method by which he succeeded with dogs, cats, rabbits, and guinea pigs, the part selected for observation being the mesentery or the omentum. The only difference between his results and those obtained by Cohnheim seems to be that he could not satisfy himself that the calibre of the arteries became wider. Emigration of corpuscles and exudation of coagulable fluid occurred in precisely the same way as in frogs.

I may now proceed to trace out in detail such explanations of the four

cardinal symptoms of inflammation as accord best with our present views; and in doing so I shall follow Cohnheim closely.

1. *Rubor.*—The redness of inflammation depends partly upon the dilatation of the veins and other blood vessels of the affected area (for Thoma himself saw the smaller veins became wider), partly upon the accumulation of blood discs in its capillaries. If the separate vessels are visible, the redness is spoken of as "injection;" if they cannot be seen, it is said to be "diffused." The tint is somewhat purple, which seems to be due not only to the preponderance of dilated veins, but also to an augmented deoxidation of the blood in the capillaries, in consequence of the retardation of the current through them. When stasis occurs, this also causes redness; and at all but the earliest stages hemorrhages also play an important part in the production of rubor. They often give to it a punctiform character, and they may be further distinguished (except from stasis) by not fading beneath the pressure of the finger. On the other hand, the presence of any considerable quantity of exudation tends to conceal the red color, even where it does not actually compress and empty the vessels: thus, at advanced periods of the morbid process, the affected tissues often become gray, or even yellow. When non-vascular structures become inflamed, the redness, of course, shows itself in the highly vascular parts around, from which they derive their nutriment; for instance, in the conjunctiva at the margin of the cornea, and in the synovial membrane about articular cartilages. After death, so much of an inflammatory redness disappears as was due to fluid blood in the interior of the vessels.

2. *Tumor.*—This may, to some extent, be due to vascular turgescence, but the chief cause of it is exudation. Lassar, Cohnheim's assistant, showed, some years ago, that the stream which flows away through the lymphatics of an inflamed part is greatly augmented. For example, he irritated the paw of a dog, sometimes by immersing it for a short time in hot water, and sometimes by rubbing in croton oil; and he found that the lymphatics in the limb above became greatly dilated, and that when he introduced a cannula into one of them the fluid which escaped through it was increased to several times its normal quantity. Evidently, therefore, the swelling corresponds, not with the whole amount of exudation, but with the excess of it, as compared with what is taken up again. Cohnheim even holds that inflammation may run its course, and be attended with exudation and with the emigration of leucocytes, and yet lead to no swelling, provided that reabsorption goes on at a proportionately rapid rate. He suggests, for instance, that this may be the case with the eruptions of scarlet fever and other exanthemata, in some of which the subsequent occurrence of desquamation of the cuticle shows that there was more than a mere hyperæmia. In describing erysipelas, I shall mention what is perhaps a more striking example of the same thing.

In distributing itself through the inflamed structures, exudation follows the lines of least resistance. In connective tissue, and in the stroma of a parenchymatous organ, it fills the natural interstices. Wherever there is a gap or interspace of any size it accumulates. It covers the free surfaces of serous membranes. Mucous membranes throw it off, to mix with their natural secretions; this, for example, is one reason why the urine becomes albuminous in nephritis. In the skin, it penetrates through the deeper soft layers of the cuticle, and it often raises the horny layer to form small collections of fluid, which are termed vesicles or pustules.

3. *Dolor.*—The pain of inflammation is usually, and no doubt correctly, attributed to the compression of the sensory nerves by distended vessels and afterwards by exudation. Thus it often has a throbbing character, being momentarily intensified by each pulsation of the heart. And it is

generally accompanied by "tenderness," or (in other words) is increased by pressure from without. Doubtless a general relation can be traced between the amount of pain induced by inflammation in different parts and the abundance of their sensory nerves; but, on the other hand, it is well known that some tissues, such as ligaments and bones, may become exceedingly painful when inflamed, although they are not naturally very sensitive. The determining factor in such cases appears to be the degree of resistance which is offered to distention and swelling.

4. *Calor.*—That those parts which are most accessible to observation become hotter than natural when inflamed, is matter of common knowledge; any one may satisfy himself of it by placing one hand over an acutely swollen joint or a recently formed abscess, and the other upon the corresponding spot on the opposite of the body. But even quite recently contradictory statements have been made as to the relation between the temperature of an inflamed part and that of the blood and of the deep internal organs.

In 1860 Mr. Simon made a series of observations with a small thermo-electric apparatus, so shaped that it could be thrust like a pin into the soft tissues, or even into the interior of large vessels; and he found not only that the arterial blood flowing towards a severely injured limb in a dog was always less warm than the venous blood flowing away from it, but that the inflamed tissues themselves were warmer than either kind of blood. But he did not attempt to measure the amounts of difference of temperature which he believed that he detected; and if (as is possible) they were very small, there may be the less difficulty in supposing that there was some source of error in his experiments. However, it appears to be certain that he was wrong in concluding that inflammation is attended with a special local production of heat. For Jacobson, of Königsberg, who afterwards investigated this question with more accurate apparatus, found ("*Virchow's Arch.*," 1870) that the temperature of the skin and of the muscles down to the bone never, even under conditions of the most intense inflammation, reaches that which exists in the rectum, the vagina, or the peritoneal cavity, at the same time. The difference was generally as much as 2° or 3.5° Fahr. And as for the relation between the temperature of one inflamed part and that of the opposite side of the body, the difference was always less in proportion as the distance from the surface was greater. Thus, whereas a rabbit's ear when inflamed was warmer than the other ear by 7° or 8° Fahr., the temperature of the deep muscles of a limb under the same circumstances seldom exceeded that of the same structures on the opposite side by more than 1°. It would follow that the internal organs under inflammation continue to be of a temperature approximately the same as that of the blood. Jacobson showed by direct experiment that in pleurisy the affected cavity was either of the same temperature as, or even slightly colder than, the other one. Still more recently, in December, 1879, M. Peter brought before the Paris Academy of Medicine a series of observations made upon patients affected with chronic peritonitis, and drew from them the conclusion that there was a very considerable local production of heat independently of the general bodily temperature. But what he really showed was nothing more than that the temperature of the abdominal wall may in such cases exceed that of the axilla by as much as 3.5° Fahr. Unfortunately he omitted to place a thermometer in the rectum, and, therefore, I think it may be fairly contended that in his cases the temperature of the inflamed part may after all have been no higher than that of the blood.

If Jacobson's observations are correct, the real increase of temperature in the skin and other superficial structures when inflamed may yet receive a very simple explanation. For the blood current is to be regarded as a great warming apparatus, which carries to the tissues all over the body heat that is

generated chiefly in the liver and other glands, the lungs, and the muscles. If there be no additional local production of heat, the temperature of each part must be the resultant of four varying conditions: (1) the temperature of the arterial blood supplied to it; (2) the proportion between the space occupied by its vascular channels and that occupied by the extravascular material which it contains; (3) the degree of rapidity with which its vessels are traversed by the blood; (4) the greater or less extent to which heat is dissipated from it by conduction, by evaporation or otherwise.

Now, I think it is obvious that none of these conditions is liable to so great fluctuations as the second and third. We have already seen that the vessels of an inflamed part are dilated. That the flow of blood through an inflamed part is much augmented has long been suspected. Sir William Lawrence is said to have roughly demonstrated this long ago by bleeding from both arms, simultaneously, a patient whose hand and forearm on one side were inflamed. But Cohnheim has recently established the same fact by careful experiments. Having set up inflammation in the paw of a dog, he measured the amount of blood which afterwards escaped through a cannula from the principal vein of the limb; and he found that it was sometimes more than twice as much as flowed from the corresponding vein on the opposite side in the same length of time. The only exceptions were, when extensive gangrene set in, and when there was very profuse suppuration. Any such increased velocity in the blood-current of an inflamed part must necessarily raise its temperature.*

The second of the conditions above mentioned explains why sometimes an inflamed part is not apparently hot. I have already cited Cohnheim's observation, that when profuse suppuration was going on in a dog's paw there was sometimes no excess of blood flowing through its vessels. And it must also be borne in mind that the presence of inflammatory exudation in any considerable quantity tends to lower the temperature, since it is just so much added to those extravascular substances which have to be warmed by the blood-current. No wonder, therefore, that in cases in which the inflammatory process runs its course slowly, one occasionally meets with what are termed "cold abscesses." But I am not sure whether it has been shown that they can develop, from beginning to end, without any elevation of temperature.

The characters of *inflammatory exudation* present many differences, according to circumstances which are as yet but imperfectly known.

In some cases, especially where a serous membrane is the seat of inflammation, the exudation assumes the form of a solid fibrillated material—"plastic lymph," as it used to be called. This is, no doubt, the same substance as the fibrin of coagulated blood, and is developed in the same manner. Where this, with the leucocytes contained in its meshes, seems, as in a dry pleurisy, to constitute the entire exudation, it must be formed very rapidly, for it appears as a thin, uniform layer upon the sloping surface, instead of accumulating in the more dependent parts of the serous cavity. But, perhaps the whole of the exudation never does solidify; a fluid residue may have been removed by the active absorbing process which we have seen to be at work.

As Cohnheim remarks, we should *à priori* expect that inflammatory

* No doubt, if superficial vessels are involved in the inflammatory process, more heat is at the same time dissipated, and this tends to prevent the surface temperature from approaching that of the internal organs. But a point of considerable importance is that such an increased dissipation of heat actually makes the surface feel hotter to one's hand than it would otherwise feel. I have alluded to this in describing the relation between heat of skin as measured by the thermometer and as is estimated by the perceptions of another person: *supra*, p. 58.

exudation would always contain fibrinogen, since that substance is one of the constituents of the blood plasma. And, as white blood corpuscles are certainly seldom absent, it becomes an important question to determine why fibrin, instead of being constantly present, is found principally in inflammation of certain structures—the serous and synovial membranes and the alveoli of the lungs. Now, in the first place, it is to be observed that so far from the composition of inflammatory exudation throughout the body being uniform, almost every part yields a material which possesses characters of its own. One peculiarity of mucous membranes is that when they are but slightly inflamed, as in catarrh from exposure to cold, they throw off an abundance of more or less altered epithelial cells. In the lungs or in the kidneys such "catarrhal products" are often seen in immense numbers; not only do they accumulate in and fill up the pulmonary alveoli or the renal tubes, but a great many of them are also carried away in the sputum or in the urine. The relation between catarrhal inflammation and inflammation in general constitutes a question of the greatest importance, especially in reference to tubercle, as we shall see hereafter; it has, however, been almost ignored by those who have recently written upon the subject. I doubt if we can, at present, form any other idea of it than that the excessive formation of epithelial cells is a result of the greatly augmented supply of pabulum offered to them in the inflammatory exudation. A tissue which in normal circumstances is constantly undergoing renewal may fairly be supposed to be ready to grow still more actively when such a stimulus is supplied to it.

On the other hand, when a serous membrane is affected with an inflammation of but moderate severity, it pours out a transparent fluid which accumulates in its cavity. There may be a layer of fibrin upon the free surfaces of the membrane; or shreds and flocculent masses of it may be floating in the fluid. But sometimes not a trace of fibrin is to be seen. And in other cases fibrinogen is present, but for some reason remains uncoagulated. Virchow many years ago pointed out that the fluid effusion, after its removal from the pleura by paracentesis, would often throw down a coagulum of fibrin, and that if this were separated by stirring, a fresh one might be formed in the course of the following day, and so on, for several days in succession.

In the more intense inflammations of serous membranes the exudation is *purulent;* and this, as a rule, possesses no coagulating power. Intermediate conditions, however, are frequent, in which the surfaces are coated with fibrin, while the cavity contains liquid pus; and in the pericardium I believe that pus unmixed with plastic lymph is seldom, if ever, seen. But this is an exception, for in the case of all other serous membranes it often happens that every part of the surface retains its natural smoothness and lustre, notwithstanding that pus is present in large amount.

The quantity of pus which collects in a large serous cavity, such as the pleura, is often enormous; and since it may be formed very rapidly the question has sometimes been raised whether the blood could possibly have yielded a sufficient number of leucocytes. But, as Cohnheim remarks, this difficulty in reality applies far less to the emigration theory than to that of pus formation by proliferation. It has been shown that while inflammation is going on the blood throughout the body contains an excess of leucocytes; and, indeed, the well known hyperinotic condition of it may be said to be nothing more than a rough illustration of the same fact. One can easily suppose that the lymph glands and the spleen throw into the circulating fluid a largely increased supply of leucocytes to make up for the drain that is going on. At the same time it appears very probable that pus cells may themselves multiply by fission. Dr. Thin has figured a dumbbell-shaped

leucocyte which seemed to be dividing into two, from the inflamed cornea of a rabbit; and similar appearances have been observed in cold-blooded animals by Stricker, Klein, and Ranvier.* I have often thought that since leucocytes evidently must have nutriment to enable them to multiply, the absence of fibrinogen in pus may perhaps be accounted for by its having been used up by them.

We have seen that the rarity of plastic lymph upon the free surfaces of mucous membranes is far from being a solitary breach of a general law for inflammatory exudations throughout the body. And I am by no means sure that the exceptional frequency with which a fibrinous layer is found lining the higher air passages may not be due to the same unknown causes which render the alveoli of the lungs also capable of a "croupous" as well as of a "catarrhal" inflammation. But in connection with this question certain points have recently been raised which deserve careful consideration. Cohnheim has drawn attention to the fact that in the interstices of the tissues generally exudation comparatively seldom coagulates; and he has shown that this probably depends not upon the absence of fibrin factors, but rather upon a controlling influence which living structures in general may be supposed to exert over its formation in their immediate vicinity. It would follow that whenever we find solid exudation in the connective tissue of any part of the body or within the stroma of an organ, we may be sure that the vitality of the part was endangered, if not altogether lost, before coagulation took place. Now, I am about to point out that there is a very important class of inflammations in which the exudation of fibrin is thus associated with a local death of the inflamed structures. And Wertheim has lately asserted that a destruction of the epithelium of the trachea is a necessary antecedent to the development of a croupous membrane in the trachea, both in animals under experiment and in the human subject during disease. But, so far as I know, the suggestion has not yet been made that the endothelium of a serous cavity must die before a layer of plastic lymph can be formed upon it. And yet inflammatory exudation is surely far less removed from the influence of living structures when it is contained in the pleural or peritoneal cavities than when it is free upon a mucous surface.

The inflammations to which I have just referred as constantly combining fibrinous exudation with a loss of vitality in the affected tissues are those which morbid anatomists now call "diphtheritic." The term is an unfortunate one, since it suggests an impression that they are connected with the disease Diphtheria, whereas this is by no means generally the case. Still, I think we cannot avoid using it, for I know of no alternative name, and the condition itself is one which demands recognition. It is that in which a free surface, usually a mucous membrane, becomes coated with an adherent layer which in part at least consists of the upper strata of the mucous membrane itself, infiltrated with a fibrinous material. The distinction between a *croupous* and a *diphtheritic* false membrane is not merely, as was formerly taught, that the one is easily separable while the other is firmly attached. It is chiefly in the air passages, where there is a thick basement membrane, that even croupous membranes lie loose; upon the fauces and elsewhere they often adhere closely, if their fibrillation is dense and strong. A surface affected with diphtheritic inflammation looks

* It is important to notice that the peculiar appearance of the nucleus of a pus cell—the replacement of the single nucleus of a blood leucocyte by two, three, or even five irregular granules—is an indication not of multiplication by fission, but of impending disintegration and death. Cohnheim, indeed, says that pus cells must be alive, for they are capable of amœboid movements; but I suppose that this is only when the pus is of very recent formation.

dry, and is of a slate-gray or pale yellowish color unless it has been accidentally stained by any colored fluid, such as bile. It feels rough and granular, and a still more marked character of it is its tough, elastic hardness. In the dead body, if an incision is made into a diphtheritic membrane, the dry gray appearance is found to penetrate to some depth, and to cease somewhat abruptly. Under the microscope is to be seen a granular, more or less distinctly fibrillated substance, containing in its meshes altered epithelial cells, and leucocytes which have lost their nuclei. In the more superficial part of such a false membrane the fibrin is often developed into thick and glistening fibres; in the deeper layers, where it is infiltrating the interstices of a pre-existing tissue, it has no room to show even fibrillation, and it looks granular. As I have already shown, so much of the substance of the part as is involved in the diphtheritic process has already lost its vitality, and the only way in which the disease can end (unless it destroys the life of the patient) is by the separation and shedding of all the dead tissues, with the false membrane of which they form a part. This detachment is effected by *ulceration*. From the first there was an inflammatory action of more or less intensity in the living structures beneath. But now leucocytes collect in large numbers along the boundary line, and I suppose that the connecting material is directly eaten away and absorbed by them. Perhaps the best specimens of the shedding of an extensive diphtheritic membrane are afforded by the urinary bladder. This organ is exceedingly liable to diphtheritic inflammation, which often involves the whole thickness of its mucous coat; and it is no uncommon thing for the *mucosa* to be cast off almost entire. A year or two ago I made an autopsy in the case of a woman from whose bladder during life a piece of considerable size had been removed. All the rest of the vesical mucous membrane lay loose, as a thick, ash-gray substance, with markings on one surface corresponding with those of the hypertrophied muscular trabeculæ.

It is obvious that recovery from a diphtheritic inflammation, when possible at all, involves the regeneration of the tissue which has undergone destruction, or at least its replacement by cicatricial tissue. After the separation of a croupous membrane there is nothing to be renewed unless it is the epithelium.

Not only is diphtheritic inflammation characterized by loss of vitality of the affected tissues, but also by their putrefaction; as is shown by their dirty gray color, and by the fetid odor which they give off. Indeed, this form of inflammation is but one member of a group, which may properly receive the name of putrid "inflammations," and in many of which large masses of tissue undergo death—*mortification* or *gangrene*.

It is customary to describe several forms of gangrene, and to refer them to different causes. But I think that the more one studies these supposed varieties the more difficult is it to regard the distinctions between them as fundamental. Of course, it is true that a part may be directly killed in various ways without the intervention of an inflammatory change in it; as, for instance, by escharotics or by certain degrees of either heat or cold, according to the length of time during which they are in operation. But in every case, I believe that a less intense application of the destroying agent would set up a violent inflammation, which itself would end in gangrene. So, again, when the death of a part is due to a failure of its blood supply, no doubt it does not always first undergo inflammation throughout its whole substance. But here, also, it seems impossible to draw a hard and fast line. Surgeons are now familiar with the fact that in many instances senile gangrene of the foot, which used to be attributed merely to obstruction of the arteries, arises from slight injury to a toe nail, and begins as an inflammatory process.

And even when a limb undergoes mortification as the direct result of embolism of its main artery there is always at the margin of the gangrenous part an active inflammation, which leads to a further death of the tissues. Indeed, Cohnheim has shown that the effects of a temporary ligature applied round the ear of a rabbit depend entirely upon the duration of the constriction. After a certain length of time the blood fails to penetrate into the vessels, when the ligature is removed, and the part dies; but in other experiments, which were terminated at earlier periods, the result was only an intense inflammatory swelling of the ear with hemorrhages into it. Lastly, in those forms of gangrene which seem to be determined by the occurrence of pressure, whether directly, by its lowering the vitality of the tissues, or indirectly, by its cutting off the blood stream through them, there are always further conditions: a previous enfeeblement of the circulation, and some slight local injury which under normal conditions would have produced no appreciable ill effects. Thus, bed sores occur chiefly in those cases in which there is also a marked tendency to hypostatic congestion of every part that happens to be dependent; and I believe that the gangrene is always preceded by an inflammation that can be directly attributed to such causes as irritation of the skin by excreta, or bruising of it during even gentle washing and drying, or friction of it against folds in the sheets. Thus, if we exclude what may be termed the immediate or instantaneous occurrence of gangrene, I believe that all forms of it may be said to arise out of an inflammatory process.

We have seen that putrefaction is an essential part of gangrene. But in different cases there are great variations in the rate at which chemical changes in a dead tissue advance, and in the degree of fetor produced by them. In the "senile" form, the affected parts dry up and shrivel into a hard material like that of a mummy; and "dry" gangrene was formerly described as the characteristic effect of obstruction of the arterial blood supply. But it is now known that the desiccation depends upon the escape of fluid from the surface in consequence of detachment of the epidermis. And it is only when mortification spreads very slowly indeed through the more massive segments of a limb that they can "mummify." The gangrene which ensues upon embolism of a large artery is, I believe, always more or less moist. But it often happens that deep structures remain soft, while superficial ones dry up into a horny mass, which is termed an *eschar*. The color of gangrenous parts is generally greenish or purplish black, but sometimes gray; it is due to chemical changes in pigment which has escaped from the blood discs and has diffused itself through the tissues. The horrible odors so often emitted are attributed to the formation of ammonia, sulphuretted hydrogen, volatile fatty acids, etc. Gases frequently collect to such an extent as to render mortified tissues emphysematous, so that they crackle or "crepitate" when they are touched. In other cases they are saturated with a dark, thick, red fluid, which perhaps also raises the cuticle into bullæ. That structures which have undergone mortification are completely devoid of sensibility and of any other vital function, is a matter of course. They also feel cold, since their supply of warmth is cut off with their supply of blood; but it is to be noted that very superficial patches of cutaneous gangrene sometimes receive so much heat by conduction from the subjacent textures, that their temperature scarcely appears lower than that of the skin in their neighborhood.

Gangrene is often from the first "circumscribed," or limited to a certain part, and even when it spreads from one spot to another, its progress—if, indeed, the patient's life is saved—is at last always arrested. A "line of demarcation," as it is termed, is then formed. Here active inflammatory

changes develop themselves, an abundant exudation of leucocytes takes place, and the connection between the living and the dead tissues is gradually eaten away, until the latter become completely detached and are cast of.

Hitherto we have been regarding the different characters of inflammatory exudation as consequences either of special tendencies which we find to be manifested by the several tissues, or of the greater or less intensity of the irritation by which the inflammatory process was set up. There is, however, another side to this question. Some years ago Sir James Paget examined the fluid contained in blisters raised by the application of cantharides to the skin, in thirty patients of St. Bartholomew's Hospital. In some cases it formed a firm, elastic, fibrinous coagulum, in others it was purulent. The former condition was observed in persons who were in sound health, the latter in those who were suffering from advanced phthisis, or a similar disease. And, with a little practice, he found himself able to form a fair opinion, from the contents of a blister, as to the degree to which the person was cachectic, and as to the extent to which inflammation in him would tend to be "adhesive" or "suppurative." Again, in those who are suffering from Bright's disease of the kidneys it is well known that the exudation produced by even slight inflammations is apt to be very profuse and watery. Lassar found, by direct experiment, that when hydræmia was induced by the free injection of a saline solution into the veins of a dog, in whose paw inflammation had previously been set up, the lymph which escaped from the lymphatics of the limb became much increased, while the amount of solid matter in it was as greatly lowered. On the other hand, patients affected with dropsy from renal disease are especially liable to gangrenous forms of inflammation, and the same thing may be said of those who labor under diabetes.

Up to this point we have been dealing with the advance of the inflam matory process. But, except when it is cut short by the death of the patient, it always, sooner or later, enters upon a further stage, one of *subsidence* and *repair*. If it is of slight intensity, and if its exciting cause does not continue in operation, it may end directly in what is termed *resolution*. The circulation through the vessels of the affected tissues then gradually resumes its natural condition; any leucocytes which may have escaped into the part pass away through the lymphatic channels; any solid exudation that may have been formed in it undergoes conversion into a fatty emulsion, which is likewise carried off in the lymph stream. On the other hand, if pus cells have accumulated in large quantities in a serous cavity, or in the sac of an abscess, they die there and break up into granules. The fluid belonging to them is then absorbed, while the solid matters remain as an opaque yellowish mass. This process is known as *caseation;* it is one to which I shall frequently have occasion to allude. Such cheesy residues are supposed by many pathologists to be incapable of undergoing further absorption; but, for my own part, I think that this is very doubtful. They often become the seat of deposits of earthy salts, which give to them the consistence of mortar, or may even render them as hard as bone. The tissues which surround them always present formative changes such as I am about to describe. These, however, must sometimes be regarded as the result of a fresh irritation set up by the dead and caseating matters, which may then be said to act as "foreign bodies."

In all other cases, the *formation of new tissues* constitutes a direct and essential part of the subsidence of inflammation. Thus, when a slough or

an eschar becomes detached, or when an abscess discharges its pus, a hollow space is left, which has to be filled up by a process of growth. And even where the morbid change does not go beyond the exudation of plastic lymph, this is often, to some extent, developed into a permanent structure. The steps by which such varieties are brought about have been studied with peculiar success by Ziegler (1876). He embedded in the subcutaneous tissues of dogs a series of minute flat chambers, each consisting of a pair of glass plates, with a capillary space between them, cemented together at the corners, but open at the sides. These he removed after an interval of several days or even of some weeks, and he was fortunate enough to find not only that leucocytes had penetrated into the space between the plates, but sometimes that tissue elements and even blood vessels had developed there. The earliest change consisted in the formation of large granular cells with vesicular nuclei. These he terms "epithelioid" cells, on account of their appearance. They are generally round, but sometimes oval or even irregular in shape. Their size is very variable, but sometimes they are larger than the epithelial cells of the tongue. They evidently arise out of leucocytes, which appear to grow at the expense of other leucocytes, whose protoplasm they appropriate or absorb. Indeed, some go on developing until they become what are termed *giant cells*—irregular masses of protoplasm—each containing a large number of nuclei. That these also are formed by the growth of a single cell, which destroys several others, there is no reason to doubt. In support of this view, Ziegler lays stress on the fact that they are constantly found surrounded by an empty space. At first, he supposed that all the nuclei of a giant cell were developed by fission from the nucleus of the leucocyte in which it had its origin; but he has since admitted that the nuclei of the absorbed cells may perhaps persist in it. Lastly, another feature of some of Ziegler's preparations is a *reticulum*, forming sharply outlined septa round the individual cells. This appears to indicate a tendency towards tissue production, but there is no reason to suppose that it is a permanent structure.

The really important agents in the formation of connective tissue seem to be the so-called "epithelioid" cells. They become pear-shaped or spindle-shaped, and give off bundles of delicate fibrils. These fibrils assume parallel directions, and ultimately their extremities unite with those of fibrils derived from other cells. But there is one condition which appears to be essential to such a result; namely, the development of blood vessels in the exudation. They seem to arise partly as buds and loops from the sides of already existing capillaries, partly out of rows of cells, placed end to end, so as to open into one another and form hollow tubes.*

When much suppuration occurred around Ziegler's glass chambers, no formative changes were discernible in them. This corresponds with the well-known fact that only fibrinous effusions into a serous cavity are capable of organization. But it is particularly to be observed that in speaking of "plastic lymph" as undergoing development into connective tissue and vessels one means, not the fibrin itself, but the cells that are included in it. The "fibrillated" structure of the lymph has nothing whatever to do with the processes that subsequently go on in it.†

* Ziegler was originally disposed to think that the giant cells were specially destined to undergo conversion into vessels. But he has since given up this opinion, and teaches that they sometimes develop into connective tissue and sometimes into vessels, while others of them disintegrate and disappear.

† In human pathology, it is chiefly in granulations that one has an opportunity of observing formative changes in leucocytes: but Cohnheim speaks of large round or elliptical

The connective tissue which is developed out of inflammatory exudation is sometimes of an enormous thickness, as in the pleura. It very slowly reaches its permanent condition. At first it contains no elastic fibres; according to Paget, they may appear as long as twelve months after its first formation. Lymphatic channels in it have been particularly described by Van der Kolk; and nerve fibres are said to have been discovered. Bone is often formed where the part of the body is one of those that naturally contain bone. In the cardiac valves, amorphous calcareous matter may be deposited in large quantity. Both adhesions and cicatrices have a strong tendency to contract, and this may give rise to very serious consequences if vital organs are concerned. But, on the other hand, adhesions sometimes, after a long time, relax and soften, even if they do not altogether disappear. Paget cites the following observation of Bichat's: a man had made from twelve to fifteen attempts at suicide, at different times, by stabbing himself in the abdomen. In the situation of the more recent wounds, it was found that the intestines adhered to the parietes; but the adhesions corresponding with the older ones were reduced to narrow bands, or had even become divided and were hanging free. In cicatrices also a marked "loosening" may at length take place. These facts, as well as the ultimate subsidence of scleroderma, deserve to be borne in mind in reference to analogous changes in internal organs, which we are too apt to regard as necessarily permanent.

Hitherto I have said nothing about the development of epidermis or of *epithelium* during the subsidence of inflammation, although it is a necessary step in the process, not only after ulceration of the skin or of a mucous membrane, but in many other circumstances. For on this question, the most modern doctrine is one to which I hesitate to give my assent. That the cuticle which ultimately covers a granulating wound always arises in contact with pre-existing epidermis is well known; Reverdin's practice of skin-grafting is an interesting illustration of it. But this fact by no means necessarily involves the truth of the doctrine that epithelial cells are always actually developed out of cells of the same kind; it would be quite explicable upon such a theory as that which Rindfleisch propounds under the name of "epithelial infection," the existing epithelial cells being supposed to influence the adjacent granulation tissue, so as to lead its cells to grow into epithelium. However, on the basis of the differentiation of the cellular elements of the embryo into three layers, it is now taught by the most advanced pathologists that it is impossible for leucocytes, which belong to the middle layer, to form epithelial cells, these being constituents of the upper and lower ones. We shall see further on that in relation to malignant tumors, this question is one of the highest importance.

With regard to the *causes* of inflammation in general, little need be said in the present chapter; they will be discussed hereafter, in detail. But it must be pointed out that the analogy of the pathological processes concerned compels us to classify as inflammatory a large number of affections in which we cannot directly recognize the operation of any irritant upon the vessels or the tissues, so that we have to fall back upon theoretical conceptions of a more or less unsatisfactory kind. Concerning the mode of origin of the large and important class of internal inflammations due to exposure to cold we know absolutely nothing. That cold often excites pleurisy, pneumonia, myelitis, nephritis, and a host of so-called

cells or even cells with many nuclei, as being generally discernible in small numbers in the pus which is discharged from a wound or an abscess of not less than a week's standing.

catarrhal affections, is certain. But I am inclined to think that there is no general principle to which their ætiology can be referred, and that the relation between these several diseases and their cause is different for each one of them. A point of importance is that there are some organs and tissues which, so far as we know, are not capable of inflammation as the result of cold. This is the case, for example, with the brain; and, I believe, it will be found to be so with the peritoneum likewise, if one is sufficiently careful to exclude all those forms of peritonitis which start from the uterus, or from some other neighboring structure.

There can be no doubt, however, that many inflammations depend only in part upon the external cause that excites them; the larger share of their ætiology belongs to what may be fairly termed a "predisposing" morbid condition of the tissue itself or of its blood vessels. And it would seem that this has an important bearing upon the way in which some inflammations spread from point to point, and even from tissue to tissue, without decrease in their severity. When an irritant is applied to any part of the body its effects may extend for some little distance beyond, but their intensity generally rapidly diminishes—unless, indeed, the resulting morbid process belongs to a separate group of affections, which will presently be described as "septic" or "infective." But many of those inflammations which are less obviously traceable to an external cause diffuse themselves very widely. For instance, we may find pleurisy complicated with pericarditis or peritonitis, pneumonia leading to meningitis or to nephritis, and so on. In one remarkable case, in which I made an autopsy, the patient, a boy, was admitted into hospital, under Dr. Taylor, for what at first appeared to be meningitis, but a day or two later the symptoms seemed rather to be those of peritonitis. After death, the oldest lesion was found to be a chronic pericarditis, probably of three or four weeks' standing; but in addition there was acute pleurisy, diffused acute peritonitis, and equally universal acute meningitis. I may add, that in no part of the body was there any trace of tubercles, nor even any caseation. In such cases we have no reason to suppose that any specific infective agency had been at work. To what can we attribute the spread of the disease, if not to the circumstance that its main cause was an unsound state of the affected tissues altogether antecedent to irritation of them?

We have seen that the characters of the morbid process lead us to identify as inflammatory a number of affections which are by no means obviously traceable to irritation of any kind. They also enable us to draw a similar conclusion in another large group of cases, notwithstanding the absence of most, or even all, of the so-called "cardinal" symptoms. The inflammations in question are what are termed *chronic*. No doubt Cohnheim is right in saying that the reason why they are so often unattended with heat, or redness, or pain, is that their extent is so limited; and we may perhaps follow him in assuming that their prolonged duration is chiefly due to the fact that the newly-formed vessels and tissues act as causes of fresh irritation. It has often been said that there is great difficulty in determining what affections should and what should not be classed as chronic inflammations. But I think that this has mainly arisen from the fact that until recently pathologists were strongly biased towards regarding the changes in question as either "degenerations" or "hypertrophies." Let me cite as examples *arteritis deformans*, with its terminations in atheroma and in calcification, the fibroid thickenings of cardiac valves, the secondary sclerosis of the spinal cord and of other nervous tissues. The inflammatory nature of all these affections is now generally allowed. As regards hypertrophies, however, it must be admitted that

there is in reality no boundary line between them and inflammation, at least so far as the connective and epithelial tissues are concerned.

I shall not in this work attempt to lay down rules for the *treatment* of inflammation in general. The affections to which alone such rules would apply almost all belong to the domain of surgery rather than to that of medicine, so that I should be repeating, to no good purpose, what will be found in full detail in surgical text-books.

TUBERCLE.

ANATOMY AND HISTOLOGY OF TUBERCLE: VIEWS OF LAENNEC, BAYLE, AND LOUIS: OF VIRCHOW, SCHÜPPEL, AND ZIEGLER—TUBERCLE A PRODUCT OF MODIFIED INFLAMMATION—INFILTRATING TUBERCLE—CASEOUS AND FIBROID TRANSFORMATIONS OF TUBERCLE—KOCH'S TUBERCLE-BACILLUS—RESULT OF EXPERIMENTS ON ANIMALS: INOCULATION OF TUBERCLE: PERLSUCHT: INFECTION BY MOUTH AND BY LUNGS—ÆTIOLOGY OF TUBERCLE: SPREAD OF TUBERCLE IN THE BODY—COURSE OF ACUTE TUBERCULOSIS—VARIETIES OF CHRONIC TUBERCULOSIS: PHTHISIS PULMONUM, LARYNGIS ET ILEI; TUBERCULAR INFLAMMATION OF SEROUS MEMBRANES: GENITO-RENAL TUBERCULOSIS; ADDISON'S DISEASE; TUBERCULOSIS OF THE LYMPH GLANDS AND SPLEEN; TUBERCULAR DISEASE OF JOINTS; MULTIPLE TUBERCLE GENERALLY—CONCLUDING HISTORICAL RETROSPECT.

We find in the deadhouse that certain peculiar diseases, which affect, in turn, almost every organ, agree in presenting minute rounded bodies which are called *tubercles*, or *tubercular granulations*. But the relation of these bodies to the diseases in question has, throughout the present century, been a matter of never-ending discussion.

The importance of the controversy may be judged from the fact that it concerns the most frequent of all fatal maladies, pulmonary phthisis. And in proof of the extent to which opinions have differed, the words of Laennec, in 1819, may be contrasted with those of Niemeyer, in 1867: the former taught that "the existence of tubercles in the lungs is the cause, and constitutes the true anatomical character of Consumption;" the latter, that in the majority of cases tubercles, if found in the lungs after death, "have been of recent origin and have complicated the disease when it was already in an advanced stage." My own view is, and has always been, entirely in accord with that of the older writer.

One great stumbling-block in the way of a right understanding of the nature of tubercles has been an imperfect observation of the changes in appearance which they present in different stages of their formation. In this respect Laennec was far more accurate than many who have succeeded him. He described them, as, first, "having the appearance of small, semi-transparent grains, grayish or colorless, varying from the size of a millet seed to that of a hemp seed. . . . Afterwards they gradually increase in size, and as they do so they become yellowish and opaque, beginning in the centre. . . . Several unite together to form larger masses, pale yellow, opaque, and of the consistence of very firm cheese. . . . At length they soften and finally "liquefy;" this change also begins in the centre, and progressively approaches the circumference." The gray, semi-transparent granules were termed by Laennec "miliary tubercle;" to the yellow, cheesy masses he applied the unfortunate name of "crude or immature tubercle." Louis, whom Laennec's premature death left the leading pathologist of Paris, limited the application of the word tubercle to the yellow, opaque stage of it; his method of statement being that the gray, semi-transparent granules "undergo conversion into tuberculous matter." By subsequent writers the early gray stage was ignored altogether. Perhaps they were influenced by the misleading term "crude" tubercle, applied to the yellow stage of it. At any rate, they taught that the yellow material was deposited, as such, from the blood.

Virchow, therefore, effected a most important reform of pathological doctrine when he showed that a yellow, cheesy substance, identical with "crude" tubercle, may arise out of decaying matters of various kinds, from simple inflammatory exudations up to sarcomatous and carcinomatous growths; in other words, that *caseation* is merely a mode of retrograde metamorphosis. Nevertheless, I think that Virchow greatly underrated the extent to which cheesy masses in the lungs, and in other organs, are really of tuberculous origin. And one or two of the writers who have followed him almost go so far as to deny that tubercles ever caseate at all.

A proof that gray and yellow tubercles may be stages of the same morbid product is, as Louis long ago pointed out, that the successive changes described by Laennec may often be observed to occupy definite positions in the lung. Toward the base there are gray, semi-transparent tubercles; higher up they are of opaline aspect, and yellowish in their interior; higher still they are yellowish-white throughout their entire substance. He might have added that they become larger and approach one another more closely from below upward. Now, since it is certain that the upper lobe of the lung is almost always the earliest seat of tubercles, and that they gradually spread downward through the organ, the conclusion seems indisputable that they are first gray and afterward become yellow. It is, however, also a fact that tubercles often undergo caseation before they are large enough to be visible with the naked eye. Both in the pia mater and in the liver, I have repeatedly found tubercles of microscopic size, which were already opaque, not only in the centre, but in the greater part of their substance. It is therefore quite possible that no gray tubercles may be discoverable in a diseased organ, even when tuberculosis has been rapidly destroying it. Again, another fact, which is still more important, is that tubercles, instead of caseating, often undergo a different change, which leaves them permanently gray. The earliest observation of tubercles in this condition dates further back than the writings of Laennec himself. A few years before the publication of his great work, in 1827, another French physician, Bayle, had divided phthisis into different species, only the first of which was termed by him "tubercular." To the second species he gave the name of "Granular Phthisis," and described the lungs as "stuffed with transparent, shining granulations, of cartilaginous nature and consistence, never opaque, and without any tendency to soften." Now, it is a curious circumstance that those pathologists who (as we have seen) came to regard a yellow, cheesy condition as typical of tubercle, habitually spoke of recently-formed gray tubercles as the "gray granulations of Bayle;" for a perusal of the four cases of granular phthisis, related in detail by that writer, shows clearly that, in three of them, at any rate, the disease was of a *chronic* kind. Such a degree of hardness as he attributes to his granulations is, indeed, inconsistent with their having been at an early stage of their development; and he also expressly mentions that they are sometimes pigmented, or speckled with brilliant black dots and lines, which will be shown elsewhere to be characteristic of *chronic* tubercular affections of the lungs and bronchial glands. In fact, the gray granulations of Bayle are really tubercles which, instead of caseating, have undergone a fibroid change, which makes them persistently hard and semi-transparent.

Miliary tubercles, then, may be of three kinds:—

1. *Soft, gray granulations*, always of recent formation, and essentially transitory in their characters.

2. *Yellow granulations*, of either recent or old formation, tending, if they undergo further change, to soften or liquefy.

3. *Hard, gray granulations*, always chronic, and liable to no change, except pigmentation.

Histological Characters of Tubercle, According to Different Authorities.—It may well be supposed that of late years histologists have had much to say with regard to the nature of the tubercular process. But, for my own part, I venture to think that their views on this subject are fundamentally erroneous. Their object has been to discover some microscopical characters which should serve to distinguish tubercular lesions from all others. Thus, Lebert, many years ago, thought that he had found a specific "tubercle corpuscle" in the yellow cheesy material which was at that time taken for the typical form of the morbid product. Since Virchow taught that this material is already in a state of decay and degeneration, the search has been actively prosecuted in tubercles still gray and recent. Virchow himself held that their histology is that of a form of lymphoma. According to him, individual tubercles are comparable with the Malpighian bodies of the spleen. And, as he believed that they are apt to arise where no such lymphoid structures are naturally present, he regarded them as "heteroplastic" new growths. Subsequent writers further developed this view by insisting on the existence of a reticulated stroma, such as belongs to other lymphomata. It was also suggested that the true seat of tubercles was perhaps the interior of the lymph channels throughout the body, and that their cells might probably be formed by a proliferation of the lymphatic endothelium. On the other hand, Dr. Burdon-Sanderson endeavored, in 1868, to demonstrate in those parts in which tubercles are most apt to occur, the normal presence of lymphoid tissue, an overgrowth of which might lead to their formation, and thus really render them "homœoplastic."

But about that time the attention of histologists became drawn to another element of tubercles, which has played a principal part in all the various theories that have since been advanced with regard to them. That very large cells are sometimes to be found in a tubercle has long been known; Virchow himself had spoken of cells containing twelve nuclei, or even more. Langhans and Schüppel, however, now insisted on the frequency of the presence of such bodies, and applied to them the name *Riesenzellen* or giant cells, which had previously been invented by Virchow for similar elements occurring in certain sarcomatous tumors. According to Dr. Hamilton, whose papers in the "*Practitioner*" for 1880 are illustrated by beautiful drawings, giant cells occur with the utmost regularity in all tubercles. They are from ten to thirty or forty times larger than the lymphoid cells, which are also present. They are sometimes placed in the centre of a tubercle, sometimes laterally. When a giant cell is young, it seems to consist of a large mass of granular protoplasm, sometimes with many nuclei in it, sometimes without them. As it grows older, the peripheral part of it (the "periplast") becomes organized, and constitutes an almost fibrous mantle-like sheath, in which great numbers of round or oval nuclei may be perceived, and which ultimately gives off processes in continuity with the reticulated stroma of the rest of the tubercle. Further on, Dr. Hamilton speaks of a tubercle as made up of one or more "giant-cell systems." He imagines that even the "lymphoid" and "epithelioid" cells which are found in the tubercle have originally been connective-tissue nuclei, which have become detached and thrown off into the meshes of the reticulum. But I must confess that, without attempting to challenge the accuracy of Dr. Hamilton's observations, I cannot help thinking that there is much that is artificial in his descriptions. Histologists, indeed, are by no means agreed as to the origin of the giant cells of tubercle. Dr. Hamilton's view is that each of them arises by the progressive growth of a single large connective-tissue element. But Schüppel himself attributed them to the coalescence of leucocytes in the interior of capillary vessels.

And both Dr. Klein and Julius Arnold have recently stated that they may be produced by the fusion together of a number of epithelial cells, of which the nuclei persist. If such opinions are correct, it is, of course, impossible that giant cells can possess the significance in regard to the development of tubercles which Dr. Hamilton and others would assign to them. I suspect that in this instance, as in many others, a striking name has conveyed a false impression as to the dignity and importance of the thing to which the name is given. And, after all, it must be remembered that giant cells are very far from being as conspicuous or as easily demonstrable elements of a tubercle as they are of a "myeloid" sarcomatous tumor.

Again, the conception of giant cells as the essential and distinguishing elements of tubercles has of late years been rudely shaken from another side. I have already quoted, at p. 77, the experiments of Ziegler, who has shown, what had been to some extent recognized before him, that giant cells are formed in ordinary inflammatory exudation, during the course of its "organization" or development into tissue. I did not then mention that Ziegler's researches were undertaken with the special object of throwing light upon the structure of tubercle, and upon the relations between tuberculosis and inflammation. Such, however, was the case. Moreover, nodules, having the histological characters of tubercles, but of microscopic size, had already been found by other observers in places where their presence certainly would not have been expected. I do not now refer to Köster's discovery of tubercles (sometimes visible even by the naked eye) in the granulating tissue projecting into the interior of diseased joints; nor am I alluding to Friedländer's statement that similar bodies are constantly present in lupus and in the walls of scrofulous ulcers of the skin: for all of these affections are believed to bear a more or less close relation to tuberculosis. But it is impossible to say the same of certain other lesions in each of which Friedländer or Köster or Cohnheim has found microscopic tubercles. Among them are a shallow ulcer of the *os uteri* in an old woman who died of apoplexy, the stroma of a cancerous tumor, the floor of a phagedænic chancre, and bands of adhesion in the pleura or in the peritoneum. Friedländer, indeed, maintains that all such cases are examples of a "local tuberculosis," comparable with a scrofulous testis, or even with a pulmonary phthisis, which does not happen to lead to a general outbreak of tubercles. But it seems to me that this is to take a very strained and partial view of the facts.

Probable Connection of Tubercle with Inflammatory Exudation.—The general conclusion which I wish to draw from all these observations with regard to the histology of tubercle is, that there is nothing in them to prevent our accepting the doctrine that *Tuberculosis is a modification of the inflammatory process*, if such a doctrine should commend itself to us on other grounds. Some of the reasons which have led me, for one, to accept it will be stated further on. My present object is to point out that it seems to afford the only possible solution of a difficulty which histologists regard as insuperable, but which I am sure cannot be really so. I have already referred to Niemeyer's rejection of the opinion that tubercles are an essential feature of pulmonary phthisis. Now, the fundamental fact upon which his teaching was based was the microscopical observation that in many specimens of phthisical lungs the morbid process which precedes the occurrence of caseation is not the development of a lymphoid tissue, but a filling of the alveoli with epithelial cells, or in other words, a "catarrhal pneumonia." He accordingly declared that in reality a chronic catarrhal pneumonia, ending in cheesy infiltration, constitutes the primary anatomical lesion in many cases of phthisis. His opinions have since been adopted by many other path-

ologists. Dr. Hamilton, for example, asserts that dry yellow nodules, such as were described in the lungs by Laennec, are generally nothing but patches of catarrhal pneumonia; bodies having an aspect similar to that of a tubercle, but as large as millet seeds, usually (he says) prove to be groups of air vesicles affected with catarrhal pneumonia. Now, when I come to the chronic form of pulmonary disease I shall bring forward the evidence which has always led me to maintain the unity of phthisis and its essentially tubercular character; and, indeed, this view seems to have been finally established by Koch's observations with regard to the bacillus of tubercle, to which I shall refer in the present chapter. But it is the greatest possible mistake to suppose that the cases in which catarrhal pneumonia must be separated from tuberculosis can be limited to those of phthisis, if the separation is to be made at all. On the contrary, the principal point on which Dr. Wilson Fox insisted, in opening the discussion on tubercle at the Pathological Society, in 1873, was that, in the disease known as acute tuberculosis, occurring in children, granulations "composed of epithelial proliferation" are generally found in larger numbers than those made up of lymphatic cells. And in that very year a German observer, Hering, actually went so far as to declare that the ordinary fatal miliary tuberculosis of the lungs ought to have its name changed into that of an acute disseminated catarrhal pneumonia. But this would, after all, leave the difficulty untouched; because, as Dr. Fox pointed out, granulations made up of an interstitial small cell growth are generally also present in such cases, and some granulations consist partly of one kind of elements, partly of the other. Moreover, it has recently been shown by Julius Arnold that precisely similar epithelial changes occur in the liver and in the kidneys, as in the lungs, when affected by acute tuberculosis; and Gaule has demonstrated a caseating catarrh of the seminal tubules in the so-called scrofulous disease of the testis. Now, if the tuberculous process be regarded as a modification of inflammation, it is, I think, possible to solve the difficulty without giving up the use of the term tubercle, and without ignoring the important distinctions that undoubtedly exist between ordinary inflammatory affections and those that are tuberculous. In speaking of catarrhal inflammation (*supra*, p. 72) I have already pointed out that its relation to the inflammatory process in general is still imperfectly understood. But, at least, this is certain, that the same kind of irritation which in most other tissues leads to an exudation of leucocytes, causes, when applied to epithelial tissues, an overgrowth of epithelial cells. Ziegler has thrown out the very plausible suggestion that a tubercle owes its rounded form and its definite size (varying only within rather narrow limits) to the fact that it is produced by the circumscribed action of an irritant of but slight intensity upon the spot which afterwards becomes its centre. And he contrasts the effect of such an irritant with the far more powerful operation of a septic particle, such as gives rise to a miliary abscess. Now, it is, I think, easy to conceive that whereas the circumscribed irritation of a minute area of connective tissue shall result in the formation of a granulation made up mainly of leucocytes, the same kind of irritation applied to an epithelial area shall cause a granulation consisting of epithelial cells. The two bodies may differ completely in their histology, and yet we may be quite right in giving both of them the same name. This is what I meant when I said that the views of histologists with regard to the subject of tubercle seemed to me fundamentally erroneous.

Specific Character of Tubercular Inflammation; Infiltrating Tubercle.— The conception of tubercle which I have unfolded in the last paragraph is one which I have now for several years past taught in my lectures on

pathology. Koch's discovery of a tubercle bacillus appears to me to supply exactly the "irritant of but slight intensity" that is needed for the theory. The slow growth of this organism, to which Koch himself draws attention, accounts for the fact that the morbid process which results from its action is so much less acute than infective inflammations in general, with which tuberculosis must henceforth be regarded as comparable. I cannot doubt that the other "Infections-Geschwülste" of Klebs and Cohnheim, syphilis, glanders, leprosy, are also to be looked upon as modifications of inflammation, so far as concerns the pathological process which arises from the specific virus of each of these diseases.

It is not the least of the advantages of this view of tubercle that it enables us to understand how an affection (whether of the lung or of any other organ) may fail to present the characteristic granulations, and yet be really tuberculous. Laennec long ago described what he termed a tuberculous "infiltration of the lungs," considerable portions of the pulmonary tissue becoming solidified by a grayish and semi-transparent, or by a paler and yellowish-white material; either without any previous development of distinct tubercles, or around tubercles already formed. Subsequent observers have very generally declared such lesions to be "pneumonic." But, for my own part, I am prepared to maintain that in their naked-eye characters these "infiltrations" differ altogether from anything that is seen in ordinary pneumonia. And it has always appeared to me a striking point that, even when the growing edge of a patch of pinkish gray infiltration appears quite homogeneous, one often finds that the less recent parts of it, where caseation is commencing, show yellow granulations corresponding exactly with tubercles in size and in general appearance. Now, it is easy to see how a uniform consolidation should result from the irritation set up by bacilli, if they happen to be scattered very close to one another through the pulmonary tissue. Again, in speaking of tubercular meningitis I shall have to point out that in association with an acute tuberculosis of other organs an affection of the membranes of the brain is not infrequent, of which the characters seem to be those of a simple inflammation, no tubercles being discoverable. In such cases, the presence of recently-formed tubercles elsewhere appears to me to render it almost certain that the meningitis must be essentially of the same nature.

Caseation of Tubercle.—Another peculiarity of tubercular lesions, which is, I think, scarcely less characteristic than the presence of the granulations themselves, is their tendency to caseate. It appears to me that pathologists since Virchow have generally been far too ready to assume that caseation is of frequent occurrence in affections that are merely inflammatory. It is, of course, indisputable that the pus contained in a serous sac or in a large abscess may, and often does, dry up into a cheesy mass. The same thing is observed in the crypts of the tonsil, in the cæcal appendix, and in other mucous pouches, with which, perhaps, the alveoli of the lungs may fairly be compared. It is also seen in the walls of arteries when they become atheromatous. But it appears to me to be a very important circumstance that in every one of these instances the inflammatory exudation which caseates is more or less beyond the range of the blood vessels. Is it a fact that simple inflammation ever leads to caseation in the substance of a vascular solid organ—in the liver, or in the spleen, or even in a lymph gland—unless a definite abscess has been formed? Such an occurrence is commonly spoken of as if it were the most natural thing possible. But I think it is difficult to find instances of it that will bear hostile criticism. It seems to me that in a healthy person inflammation generally runs its course so as to leave no *débris* behind it, except, indeed, when an abscess forms and fails to

discharge itself. As regards the spleen and the lymph glands, I shall have to discuss this question again when I come to speak of the diseases of those organs. In the solitary follicles of the intestine it used to be supposed that caseation was often met with, quite independently of the tuberculous process, but my own observations have taught me that it is scarcely ever seen unless other parts of the body contain tubercular lesions. One of the most typical features of tuberculosis seems to me to be the presence of spreading ulcers in cavities, with yellow walls of definite thickness, the substance of which regularly undergoes caseation almost as soon as it is formed. Wherever such an appearance is formed, whether as a pulmonary vomica, or in the liver, the kidney, the prostate, or the testicle, I believe that it may be safely set down as tuberculosis.

Those pathologists who have recognized that caseation is especially apt to occur in tuberculous lesions have commonly attributed it to the absence of blood vessels in tubercles. Thus Virchow's comparison between tubercles and the Malpighian bodies of the spleen was objected to, and with justice, on the ground that the latter contain capillaries. Every museum of morbid anatomy contains injected specimens of phthisical lungs, from which it appears that not only isolated tubercles, but also patches of caseous infiltration are absolutely non-vascular. No new vessels seem to be formed in them, and the pre-existing vessels of the pulmonary tissue undergo obliteration. The further process of softening and liquefaction, by which vomicæ are formed, appears to be essentially of a chemical nature.

Fibroid Transformation.—But in many cases, as I have already mentioned, tubercles, instead of caseating, undergo fibroid transformation by their cellular elements developing into fibrous tissue. What determines them to take the one course rather than the other is as yet uncertain; but it would seem that caseation is less frequent in proportion as the age of the patient is older. In organs other than the lungs, comparatively little is known about "fibroid" tubercles. But Schüppel has demonstrated their occurrence in lymph glands, and has shown that the reticulated stroma grows at the expense of the cells, and is converted into a transparent, almost homogeneous, indistinctly fibrillated material. And Rindfleisch has recorded a remarkable specimen of fibrous tubercles in the great omentum. The occurrence of a similar change in a tubercular infiltration is, I believe, the origin of the affection which is known as fibroid phthisis. Even in such cases, there seems still to be a deficient vascularization; for Rindfleisch has insisted on the impossibility of forcing injection into the interior of indurated masses in the lungs, and contrasts their state with the abundant supply of vessels to the newly-formed connective tissue in cirrhosis of the liver and in granular diseases of the kidney. No writer has laid more stress on the fibrous transformation of tubercles than Dr. Hamilton, who, indeed, declares that the natural ultimate destiny of those bodies is to become little fibrous tumors, and that many of the so-called cirrhoses of organs, both in children and in adults, are in reality the remains of outbreaks of tubercle.

The Tubercle-bacillus.—In the "*Berliner Klinische Wochenschrift*" for April 10th, 1882, Dr. Koch made known the very important fact that he had discovered bacilli in the tubercular diseases of man and of animals, and that he had succeeded in cultivating these bacilli upon the coagulated serum of blood, and had been able to set up tuberculosis in healthy animals by inoculating the cultivated products. The organisms themselves he described as in length equal to a quarter or half the diameter of a red blood disc, and in breadth one-fifth to one-sixth of their length. When prepared by a particular method of double staining they retain a blue color, whereas nuclei and other tissue elements appear brown. This not only facilitates their recognition, but also

serves as a point of distinction between them and all other kinds of bacilli, except those of leprosy, which, indeed, resemble them very closely, and only differ in being still more slender and in having pointed ends. The bacilli of tubercle while lying in the living tissues, produce spores which are oval in form, from two to four in number, placed at equal distances in their length. When grown upon the coagulum of blood serum, the organisms become aggregated together into flat, scale-like masses, hardly as large as poppy seeds, which can be lifted off entire, and which are so firm that it requires some force to break them up. Their development takes place very slowly, about ten days elapsing after infection of the coagulum before any change is discoverable. Another condition necessary to their growth is a uniform temperature of 86°–106° F.

Koch does not think that previous observers who had described micrococci, or even rod-shaped bodies, in tubercle, had seen the same organisms which were discovered by him. They are not generally present in large numbers, except where the tuberculous process is recent and active; if it is slow, they are often to be found only within the giant cells, perhaps one or two in each cell. They appear to possess no power of spontaneous movement.

The cases in which Koch found his bacilli were the following: (1) Eleven cases of miliary tuberculosis. In miliary tubercles of the lungs they were never absent, but in those which had caseated they often existed only at the edges. They were present, too, in miliary tubercles of the spleen, liver, and kidneys, and in great numbers in the gray tubercles of basal meningitis. And they also occurred in the cheesy bronchial glands from the same cases. (2) Twelve cases of caseous bronchitis and pneumonia. (3) One case of solitary tubercular tumor of the brain, the bacilli lying within giant cells contained in the tissue immediately surrounding the cheesy mass. (4) Two cases of intestinal tuberculosis. The bacilli were present also in the corresponding mesenteric glands. (5) Two out of three cases of freshly extirpated scrofulous glands. (6) Two out of four cases of fungating inflammation of joints.

Previous Experiments in the Artificial Production of Tubercle.—Koch's discovery constitutes the final link in a chain of evidence which has been long accumulating, and which places tuberculosis among the specific diseases. In 1865 a French observer, M. Villemin, made known a fact which roused the greatest possible interest in the medical world; he found that in certain animals, particularly rabbits and guinea-pigs, tuberculosis could be set up by the inoculation of tuberculous matter from the human subject. His experiments were soon repeated in this country by Mr. Simon, Dr. Sanderson, Dr. Wilson Fox, and others. Tubercles from the pia mater or from serous membranes, caseous substances from the lungs of phthisical patients, even the sputa yielded by such patients during life, were introduced beneath the skin of animals. These were afterwards killed, or were allowed to die of the disease which followed the operation and generally proved fatal in from six to ten weeks. It was then found that pus or a dry, cheesy substance had been formed at the seat of inoculation, and that bands of induration extended away from it into the adjacent subcutaneous tissue. The lymph glands in the neighborhood were enlarged and caseous. Nodules of various sizes were present in the lungs, the liver, the spleen, and the peritoneum. These resembled tubercles, both to the naked eye and in their histology. They consisted of a gray, semi-transparent material, which underwent caseation from the centre outward. The infective character of the process was further shown by the fact that matter taken from the body of an animal after death was capable of setting up the same disease in other animals.

After a time, however, certain observations were made which seemed to alter completely the bearing of these experiments. Dr. Sanderson found that in guinea pigs an "artificial tuberculosis" arose after the inoculation of pus from the secondary abscesses of pyæmic patients, or even after the mere introduction of a clean seton of unbleached cotton. Dr. Wilson Fox independently arrived at a like result, by inoculating guinea pigs with pieces of putrid muscle, or with vaccine lymph. In Germany, Cohnheim and Fränkel introduced into the peritoneal cavity of animals of the same species portions of new growths, pieces of healthy organs of the human body, or even bits of charpie or gutta percha; and they succeeded in setting up a tubercular process not only in the serous cavity itself, but also in the lungs and in the liver. It even happened that guinea pigs in which incisions were made, without any injection, perished afterwards with inspissated abscesses at the seat of injury, and with miliary tubercles in their organs; so that it became necessary to give up experimenting upon such animals, and to have recourse to dogs, as not being liable to be made tuberculous by trifling wounds. But, after injecting into the jugular veins of these dogs pus from guinea pigs which had themselves been inoculated with non-tuberculous materials, it was found that the dogs likewise became the victims of miliary tuberculosis.

These observations fitted in perfectly with a theory of tuberculosis which had been propounded a few years before the publication of Villemin's experiments, but which, at the time, had attracted little notice. In 1857, Buhl, of Munich, had suggested that in the human subject acute miliary tuberculosis was due to the absorption into the blood of caseous matters from non-encapsuled yellow tubercles, or from pulmonary cavities not surrounded on all sides by dense fibroid tissue. Indeed, a similar opinion had been expressed earlier still by Dittrich, of Erlangen. It afterwards became the fashion to regard tubercle as always a secondary product, the origin of which was sought for in "caseous foci," of which the formation was supposed to precede, in all cases, the development of tuberculous lesions. In ordinary cases of phthisis it was thought that a catarrhal pneumonia, with secondary more or less extensive caseation, was the earliest change, and that this might go on for a great length of time before any tubercles made their appearance, so that perhaps the disease might become tubercular only at its very end. Such a view was, of course, in entire accordance with the teachings of Niemeyer already referred to (pp. 81, 84). The acute tuberculosis of children was traced back, in a large number of instances, to a simple intestinal or bronchial catarrh; this was supposed to lead first to swelling of the corresponding abdominal or thoracic lymph glands, and then to their caseation; and when once caseation had commenced the conditions of the development of tubercle were assumed to be present. Dr. Hamilton has recently maintained this doctrine with great vigor; he imagines that in the process of caseation some material, probably a ferment, is elaborated, which acts as an irritant upon the tissues to which it is carried. Now, I have always taught that such a theory of tuberculosis is altogether inconsistent with the broad facts of human pathology, whatever might be its application to the "artificial tuberculosis" of animals. Nothing is more certain than that, in man, the inspissated pus of a common abscess, or the caseous matter of an atheromatous artery, or of a degenerating new growth or gumma, does *not* produce tuberculosis. The few cases that have been recorded as illustrations of such an occurrence serve, by their rarity, to tell against the conclusion they are intended to support. Thus, Dr. Murchison some years ago exhibited at the Pathological Society a specimen in which a simple ulcer of the duodenum (an affection generally entirely free from all trace of caseation)

had been followed by tubercular disease in the lungs. Is there the smallest reason for supposing that this was anything more than an accidental coincidence? Dr. Hamilton insists upon a case in which a woman died, thirty-three days after her delivery, of general miliary tuberculosis. She had been attacked by a rigor on the seventh day, and her symptoms were at first mainly those of peritonitis. In many places, says Dr. Hamilton, more especially behind the uterus, the peritonitic fibrinous effusion had become caseous, and here and there softening had occurred. And he assumes that this was the starting point of the tuberculosis. But it seems to be very doubtful, according to the observations which we shall presently find to have been made by Cohnheim with regard to the rate of development of acute tuberculosis, whether it would have been possible for the process to have run its course in twenty-five or twenty-six days, even supposing that the tuberculous "ferment" became "elaborated" at the very moment when the peritonitis first began. And, after all, there is not the slightest difficulty in supposing that the woman's illness was, from the first, an acute tubercular peritonitis, forming, as so often happens, the clinically obvious part of a more widely diffused miliary tuberculosis. I must therefore repeat that cases of acute tuberculosis in man, attributable to infection from non-tubercular lesions, are so scarce that it is impossible to attach any significance to them.

Meantime, however, it has turned out that the experiments which were supposed to prove that tuberculosis could be set up in animals by the inoculation of non-tuberculous matters had been misinterpreted. Klebs showed that in all probability they were vitiated either by the accidental presence of the specific poison of tubercle at the time when the operation was performed, or by the wound becoming infected with it afterward. Cohnheim has since frankly acknowledged that, having repeated his investigations, both at Kiel and at Breslau, he has utterly failed to obtain the same results as before; and he is now among the warmest supporters of the specific nature of tubercle. On referring to Dr. Wilson Fox's observations, I find that all the animals which were inoculated by him, whether with tuberculous or non-tuberculous substances, were placed in a single house, so that it was quite possible for them to have infected one another accidentally. Moreover, Dr. Fox himself points out that tuberculosis was produced with far more certainty by the inoculation of tubercle than by the other methods which he employed.*

Cohnheim has since made a further advance, by showing that in rabbits and in guinea pigs tuberculosis has a tolerably definite period of incubation: from fourteen to twenty-one days. When a minute fragment of tuberculous matter is introduced into the anterior chamber of a white rabbit's eye, through an incision in the cornea, the slightest reaction which arises after the operation quickly subsides, provided that the tubercle is perfectly fresh. The fragment can now be seen through the transparent cornea, and it diminishes in size day by day until it may altogether vanish. Then, at the end of the period of two or three weeks, an eruption of small, transparent, gray, granulations appears on the iris. Afterward they caseate, and a destructive inflammation of the whole eyeball often results. Ultimately, the animal may die of a general miliary tuberculosis, in from five weeks to three months, or later; or the diffusion of tubercles may be

* [At a meeting of the Pathological Society, on December 4th, 1883, Dr. Fox stated that at his request the experiments referred to in the text had been recently repeated by Dr. Dawson Williams. The details of this series of experiments were stated, with the precautions against accidental infection; the results were absolutely negative. Dr. Fox mentioned some possible sources of fallacy in his first series of inoculations, one of which was that suggested above, by Dr. Fagge.—Ed.].

limited to the lungs or to the peritoneal cavity, or, lastly, there may be no development of them in any organ outside the eyeball.

To complete my account of the experimental study of tuberculosis in the lower animals, I have to describe two sets of observations, of which the object has been to illustrate certain methods by which the specific virus of tubercle may be supposed to make its way into the human body.

(1) One of them relates to the disease of cows and oxen, which is known in Germany as *Perlsucht*, and which is now admitted by most observers to be identical with tuberculosis, although the morbid appearances, as seen by the naked eye, differ considerably from those with which we are familiar in man. It is a chronic affection, which may run on for some years without impairing the animal's health. In fact, milch cows, particularly older ones, afford almost the only instances in which any symptoms appear during life: these are chiefly cough and wasting. It appears that animals thus affected are often sold to the butcher, so that beside the risk of drinking their milk during the early part of their illness, there is the further risk (at least to the poor) of actually eating the diseased tissues, perhaps concealed in sausages. The frequency of Perlsucht may be judged from the fact that at Augsburg 2 per cent. of all cattle slaughtered were proved to be tuberculous, and of cows as many as 5 per cent. Its experimental propagation in other than bovine animals has been studied by Gerlach, Klebs, Orth and others. Not only have pigs, lambs, rabbits and guinea-pigs been successfully infected by the milk from diseased cows, but a like result has been attained by feeding animals with portions of the affected tissues removed after the cows were slaughtered, and also by inoculating them beneath the skin with such materials. Klebs found that even boiling the milk did not, in two experiments, render it inert. A further step in the investigation has been taken by Bollinger, who thinks that a broad distinction can be drawn between the Herbivora and the Carnivora as regards their liability to be affected with tuberculosis through the alimentary canal; he supposes that animals of the latter kind secrete a gastric juice which has a more active power of destroying the virus. Man, as an omnivorous creature, ought (like the pig) to occupy an intermediate position.

One is strongly tempted to find in these experiments an explanation of the frequency of mesenteric disease in children, in whom milk forms so important an article of diet. But it seems to be a difficulty that no one has yet noticed the epidemic prevalence of the disease in the inmates of a nursing establishment, or among the children of the customers of any particular dairyman. The only approach to such an observation that I have met with is one recorded in 1877 by Dr. W. H. Spencer, of Clifton. More than twenty boys in an industrial school fell ill of what was supposed to be enteric fever. Most of them recovered, but four died; and in each fatal case the organs were found to be full of tubercles. It is, however, to be observed that in every instance caseation of bronchial glands was present, which could not have occurred within the duration of the illness of the boys, this being only three or four weeks.

Dr. Creighton has recorded several cases in which he believes that post-mortem appearances found in the human subject justify the conclusion that the disease was derived from the cow. His point of view, however, is that Perlsucht, or bovine tuberculosis, is not identical with the tuberculosis which chiefly occurs in man, and that it will henceforth be possible to separate, by certain characters, cases having such an origin from those which may be regarded as proper to the human species. And he cites Gerlach as having stated that in some of the animals that were infected by him from the cow the "peculiarity of Perlsucht was unmistakable." In addition to there being gray, translucent tubercles, like those which are seen in man

and in apes, the characters on which Dr. Creighton relies are the following: The peritoneum and the pleura present round or oval nodules, of the size of lentils, sometimes pendulous, sometimes connected with one another by fibrous cords, sometimes flat and cohering together so as to resemble the duckweed on the surface of a pond. In the lungs there may be soft, whitish masses up to the size of a walnut, some round, others wedge-shaped and like infarctions. These organs may also have in their interior closed, smooth-walled cavities, giving them somewhat the appearance of a crumpet. It is to be observed that most of the cases in which Dr. Creighton found such lesions occurred not in children, but in adults of all ages. Now, I must confess that the prospect of our being able in future to distinguish from other cases of tuberculosis in man such as are of a bovine origin, does not seem to me to be promising. But even if what Dr. Creighton terms the "structural mimicry of infection" really goes so far as to give special characters to bovine cases, it yet would not follow that the two diseases are fundamentally distinct. Koch found his bacillus in ten cases of Perlsucht, chiefly in the interior of giant cells surrounding the calcified nodules in the lungs, but also in the bronchial and even in the mesenteric glands.

(2) The other set of experiments was made by Tappeiner, in order to determine whether tuberculosis could be induced in animals by the inhalation of the sputa of phthisical patients diffused in the air as a spray. His method was to employ six grammes of the sputa at a time, and the inhalations were continued for six hours a day during fourteen days in succession, the animals being placed, for the purpose, in a narrow wooden box. Dogs were used in this inquiry, and the earliest period at which tubercles were found in the lungs, after the commencement of the inhalations, was on the twenty-third day. In no single instance were the results negative.

Bearing of the above Experiments on Human Pathology.—If we now turn to consider the bearing of these experimental observations upon tuberculosis as it is seen in the human subject, we shall, I think, be driven to the conclusion that they are applicable only within very narrow limits.

There are two questions to be discussed, which must be carefully kept apart, although they are often confounded together. One concerns the ætiology of tubercular diseases, the other refers to the way in which tubercular lesions spread through the body.

(*a*) *As to the Origin of Tubercle.*—The view that infection from without is the most essential part of the *ætiology of tubercular diseases* seems to me to be altogether inconsistent with clinical observation. It is in regard to phthisis that the matter has been most fully worked out, and I must not anticipate the account of the causes of that disease, which I shall have to give when I come to describe it. But I may briefly state that there is the clearest evidence that phthisis does not ordinarily spread from patients to other patients, or to relations, nurses or medical men. On the other hand, it has been proved that its prevalence is greatly augmented by such conditions as overcrowding and defective ventilation, and also by exposure to cold and wet, and by dampness of soil. Overcrowding and defective ventilation may be supposed to act by lowering the general health. But it is difficult to see how exposure to cold and wet can induce the disease except by their liability to set up catarrhal affections of the pulmonary and bronchial tissues; and I am disposed to think that this must also be the *modus operandi* of dampness of soil. I believe, too, that the affections of the lungs which are due to the inhalation of dust are really tubercular, and not (as is generally taught) of a different nature. Again, hereditary transmission plays a very important part in the ætiology of phthisis.

It is not easy to frame a theory of the ætiology of tubercular diseases which shall accord with these facts. There are probably great differences in the *susceptibility* of different persons to the virus, or, perhaps more correctly speaking, in the *resistance* which they offer to it. And notwithstanding the enormous mortality from phthisis and the affections allied to it, I cannot doubt that this resistance is far greater in the human subject than in the rabbit or in the guinea pig. For one must remember that those who die of tubercular diseases have commonly lived for several years, during which time they have most likely been exposed to tubercular infection again and again. It is also necessary to admit that this resistance varies in the same individual at different periods, and that it is not absent even among those in whose families phthisis is hereditary. Otherwise we should find that when one member of such a family was attacked by the disease it would quickly spread to other members. Now, the fact is, that even if several brothers and sisters die in succession, their illnesses are usually separated by considerable intervals of time. But the principal difficulty is to understand how an affection that is associated with the growth of a bacillus can bear any relation to causes which might be expected to set up a simple inflammatory process. One possible solution is that the microzyme may be so widely diffused that it is continually finding access to the body, but that it fails to implant itself there until the local resistance of the tissues happens to be lowered by the occurrence of inflammation in it. But this seems to be inconsistent with Koch's statement that the bacillus cannot grow unless the temperature is above 86° F. Cohnheim appears to think that the bacillus may remain latent in the body for an indefinite time, until some accidental circumstance calls it forth into active growth. He even imagines that the hereditary transmission of phthisis means the direct transference of the tubercular virus, either in the spermatozoa or in the ovum, as virus is transmitted in the case of the *pébrine* of silkworms. But it seems to me that there is a third possibility, namely, that under certain unknown conditions the occurrence of what is at first a simple inflammatory process may cause microzymes already existing in the body to acquire specific properties that they did not previously possess, and to convert the simple inflammation into a tuberculous one. For this hypothesis there are abundant analogies among the acute infective inflammations, of which an account has already been given.

(*b*) *As to the Spread of Acute Tuberculosis.*—Let us now pass on to consider to what extent the recent experimental observations upon tubercle throw light upon the *mode of spreading of tubercular lesions within the human body.* It is, I think, impossible to deny that they clear up very many of the difficulties which surrounded this part of the subject.

An acute general miliary tuberculosis, with its myriads of minute centres of tissue irritation, is hardly explicable in any other way than by the dissemination throughout the blood stream of minute particles of the tubercular virus, in the form of bacilli, or of their spores. Buhl himself failed to find a caseous mass which, on his view, could have been the starting point of general tuberculosis, in no fewer than thirty out of three hundred cases; and this notwithstanding that he accepted as satisfactory such lesions as a small embolic plug in an organ, or a degenerating purulent exudation in a serous cavity. The probability now seems to be that the real starting point is often a very recent patch of tubercles which happens to be so situated that an infection of the blood stream is a necessary result of its presence. Thus, for instance, Weigert, discovered, in many instances of acute tuberculosis, a caseous mass of tubercle, growing into the interior of a pulmonary vein, by extension from the pleura, or the bronchial glands, or the mediastinum. In quoting this observation, Cohn-

heim alludes to Ponfick's cases of tuberculous infiltration of the wall of the thoracic duct as another possible source of blood infection; this, however, is a very rare lesion.

But it must not be supposed that the growth and the distribution of tubercles, when they are ever so widely scattered throughout the body, are regulated solely by the way in which the bacilli are disseminated through the blood stream. There seems to be a curious difference between the artificial tuberculosis of animals and the disease as it is seen in men, with respect to the distribution of the tubercles. In the former affection they are found beneath the pleural surface more abundantly than in the substance of the lung, and Dr. Sanderson speaks of them as having sometimes the form of blunt cones. In both respects artificial tuberculosis resembles the two other great examples of tissue infection by the blood—pyæmia and malignant tumor. A third point of similarity between the three affections is that in all of them the liver forms a conspicuous seat of the secondary lesions. In artificial tuberculosis Dr. Sanderson seems to have found tubercles in the liver more often than in the lungs, but Dr. Wilson Fox observed little difference between the two organs in this respect. On the other hand, in the general miliary tuberculosis of man the liver is much less frequently and less obviously affected than the lung, although no doubt the microscope shows that hepatic tubercles are far more often present than used to be supposed. In the lung itself the tubercles are not more numerous beneath the pleural surface than elsewhere, and they do not possess the conical or wedge-like form. But, further, in many, if not in most cases of acute tuberculosis, the distribution of tubercles in the lung is such as to show clearly that the proclivities of the affected tissues play a part in determining it. In the upper lobe the tubercles are more abundant, larger, and more advanced in their development than in the lower lobe. They often gradually diminish, both in number and in size, from the apex downward. They may be quite caseous above, gray and semi-transparent below. In other words, even when the virus is disseminated by the blood stream to all parts of the lungs at once, the result is an affection which bears some resemblance to an ordinary local phthisis, so far as concerns the distribution of the tubercles. This fact seems to me to be very important, because it helps to explain what would be otherwise unintelligible—the frequent limitation of acute tuberculosis to a single organ or tissue. In tubercular meningitis, for example, it often happens that not a single tubercle exists anywhere except in the cerebro-spinal membranes; and yet one can hardly doubt that bacilli in immense numbers must have been distributed by the blood equally to all parts of the body.*

Distribution of Chronic Tuberculosis.—In chronic tubercular affections the mode of spreading seems to be by the gradual invasion of the lymph channels from point to point. Among the most striking appearances are the lines of progressive thickening and caseation, with or without the formation of distinct tubercles, which run in the course of the sub-peritoneal lymphatics from the floors of intestinal ulcers. It does not often happen that the process of local infections fails to respect an anatomical boundary line between two widely different tissues; but in children I have two or three times seen a continuous cheesy mass, with a festooned growing border, extend from bronchial glands directly through the lung substance. And once I observed a similar instance in an adult. A still more striking case seems to have been met with by Buhl. A child had caries, with caseation, of the lower dorsal vertebræ; the pleura

* It is, however, possible that, in this very acute disease, infection is really carried from some one starting point throughout the subarachnoid space, by the movements of the cerebro-spinal fluid.

became adherent, and the ulcerative process spread continuously into the base of the left lung. And Grancher ("*Arch. de Phys.*," 1878) mentions a case of tubercular peritonitis, in which tubercles penetrated the diaphragm and infected the pleura covering the inferior surface of the lung, without adhesion having taken place. But in most instances of chronic tuberculosis tissue proclivities seem to me to play a most important part in determining and in limiting the distribution of the tubercles. It is, indeed, difficult to see how the bacilli can fail to enter the blood current in greater or less numbers in many of these cases; and if so, the resistance of the tissues in general can afford the only reason why lesions are not always found scattered irregularly through all parts of the body. Instead of this, the fact is, as I pointed out in the *Pathological Transactions*, for 1874, that several distinct varieties of chronic tuberculosis can be recognized, each of which has peculiarities of its own, in regard to the organs and tissues attacked. And I would particularly remark that a parallel can generally be found for the distribution of each of these several varieties in the similar distribution of some other non-tuberculous disease; so that one is forcibly reminded of what Darwin calls the "correlated variation" of organs in their first development; as an instance of which, he cites the curious fact that male cats which are entirely white and have blue eyes are generally deaf.

The following are the varieties of chronic tuberculosis to which I have referred:—

1. Pulmonary phthisis is very commonly accompanied by tuberculosis and ulceration of the air passages (especially of the larynx), and of the intestine (especially the lower end of the ileum). Some observers think that the affection of the lungs directly causes the lesions in question, the larynx being infected by the sputa which pass over it, and the ileum by the sputa which are swallowed. There is some experimental evidence in support of such a view. But it seems worthy of notice that in enteric fever likewise, ulceration of Peyer's patches and the solitary follicles goes with ulceration over the bases of the arytenoid cartilages. And also, in acute pneumonia, ulcers are apt to form at the same spots within the larynx.

2. Dr. Wilks long ago pointed out that tubercle sometimes attacks the pleura, the peritoneum, and the pericardium, without affecting any of the solid viscera. I have already remarked (p. 79) that the same distribution is observed in some cases of simple inflammation.

3. Tuberculous disease which began in one kidney, not only spreads to an extraordinary extent, by continuity along the genito-urinary mucous membrane and the corresponding submucous tissue, but is also commonly attended with like affections of the opposite kidney, and (in the male) of one or both of the testes. I fail to see how this can be adequately accounted for by local infection. And if infection occurs by the blood-stream, tissue proclivities must surely play an important part in the result. Have we not an analogous fact in the occurrence of a "sympathetic" orchitis in a case of renal calculus? There is also a relation between tuberculous disease of the kidney and Pott's disease of the lumbar vertebræ at the same level; but in this instance direct extension probably occurs.

4. Addison's disease of the adrenals (the tuberculous nature of which affection I do not doubt) is sometimes associated with spinal caries at the same level, which, indeed, probably precedes the adrenal lesion in point of time. I have seen two examples of a still more remarkable connection, between solitary tubercle of the brain and tubercle affecting the adrenals in the form of one or more scattered cheesy masses. Virchow has pointed out that gliomatous growths sometimes occur simultaneously in the brain and in the adrenals, and I believe that facts have confirmed this observation.

5. Tuberculous disease of the lymph glands often spreads widely

throughout the body, and this is not merely by obvious continuity, for it may affect groups of glands widely distant from one another. It is also very generally attended with tuberculosis of the spleen, an organ which is not otherwise very apt to be the seat of tubercles, at least in chronic cases. A precisely similar association is seen in Hodgkin's disease.

6. Several of the joints are often affected by tuberculous disease in succession, without any corresponding affection of other structures. Here, again, one is reminded of the frequent limitation of pyæmia to joints—a peculiarly apt illustration of the effect of tissue proclivities in determining the localization of lesions, even when they are due to blood infection.

In conclusion, I must add that it seems to me an open question whether multiple tubercular affections in the same subject are not sometimes altogether independent of one another. I was once much struck by finding in an infant, six months old, two large masses of tubercular glands, entirely distinct from each other, one being in the chest, the other in the abdomen. And one not infrequently meets with cases in which the distribution of tubercular lesions is altogether capricious, without any one of them being obviously of older date than the rest. Thus, I have seen phthisis associated with a tubercular affection of one testis and of the vesiculæ seminales, in a man, and in a woman with a similar disease of the uterus and of an adrenal body. In a child who died with a tubercle in the cerebellum there was a mass of caseous glands in the abdomen. Similar instances might be multiplied indefinitely. But, of course, it is possible that all or any of them may have been due to chance infection of the blood stream, the virus happening to be carried to a spot where it met with no resistance sufficient to interfere with its action.

Historical Retrospect.—I have, in the present chapter, carefully abstained from entering into an historical inquiry into the successive views that have been advanced as to the nature of tuberculosis; but it may perhaps now be interesting to ask whether any one among the writers of the early part of this century expressed opinions approaching at all closely to those which seem to accord best with the present state of pathological knowledge. As regards the doctrine of the unity of phthisis, indeed, we need not look beyond Laennec. But his conceptions as to the relation between that disease and inflammation may be summed up in the statement that inflammation might occasionally be excited by the irritation caused by a numerous crop of tubercles. Nor can I agree with those who would assign a large share of credit to Reinhardt. It is true that he, in 1850, identified gray tubercle and gray infiltration with inflammatory processes, but, on the other hand, he maintained that yellow or cheesy tuberculous matter never arose out of the gray, but was merely inspissated pus. Among English physicians, my distinguished teacher, Thomas Adams, is often spoken of as having been the first, in 1845, to challenge the views of Laennec. But it seems to me that what he taught was that many cases of phthisis were pneumonic and non-tubercular, rather than that tubercles themselves were inflammatory products. I find no one expressing himself so clearly on the latter point as Prof. Alison, of Edinburgh, who, in his "Outlines of Pathology," published in 1844, laid it down as his opinion that "in certain constitutions, tubercles and all their consequences are direct effects of inflammatory action." Still more definite were the views of Dr. C. J. B. Williams, who classified gray and yellow tubercles as cacoplastic and aplastic varieties of coagulable lymph, "differing from the normal plasma, not in kind, but in degree, of vitality and capacity of organization." Would the pathological knowledge of his day have admitted of a more exact statement of what seems now to be the truth?

TUMORS.

NEW GROWTHS—INNOCENT AND MALIGNANT TUMORS—VIRCHOW'S CHARACTERS OF MALIGNANCY—MALIGNANCY NOT AN ATTRIBUTE OF STRUCTURE—HISTOLOGICAL CLASSIFICATION OF TUMORS—MALIGNANT OSTEOID TUMOR—SARCOMA: ROUND, SPINDLE, AND GIANT CELLS; HEMORRHAGIC AND ALVEOLAR SARCOMA: MELANOTIC SARCOMA—MULTIPLE SARCOMATA AS A GENERAL DISORDER—OSTEITIS DEFORMANS—LYMPHOMA—CARCINOMA—HEREDITY—THEORY OF NEW GROWTHS.

We have seen that, throughout the present century, theories with regard to Inflammation and to Tubercle have been incessantly changing, and have not seldom oscillated backward and forward. The doctrine of Tumor has been equally unstable, but with this difference, that the progress of investigation has opened up a succession of views, most of which had never before presented themselves to the minds of pathologists, and, indeed, could not have been understood at earlier stages of the science. The scope of the word Tumor has itself undergone limitation. Originally it was applied in practice to all swellings which were not obviously inflammatory, so that it included hydroceles, retention cysts of all kinds, hydatids, hypertrophy of the spleen, uniform enlargements of the liver, etc. Hence, when attention was first drawn to the fact that the more important tumors consisted of solid tissues which were altogether of recent development, it seemed necessary to distinguish them by a special name; and they were accordingly termed *pseudo-plasms* or *neoplasms*, or *new growths*. But, since tissue formation likewise occurs in inflammation and in tubercle, such an attempt to introduce an accurate nomenclature involves great difficulty of definition. And it is now, I think, becoming the general practice to use the word tumor in a sufficiently restricted sense.

One of the most conspicuous features of a tumor is that it grows from a centre or centres, paying little or no regard to the configuration of the structures among which it lies. When its seat is a mucous membrane, it often projects from the surface, and may even hang by a more or less distinct pedicle, in which case it is spoken of as a *polypus*. In a solid organ it may form a rounded mass, which is called a *nodule* or a *tuber*, according to its size. At a time when the life history of the cystic entozoa was all but unknown, nothing was more natural than that even solid growths should be imagined to have a parasitic origin. This notion, however, has proved without foundation. Their life is that of the individual in whom they develop.

It would be impossible to devote the slightest consideration to the study of tumors without observing that whereas some of them may remain for years without affecting the health or causing any inconvenience, others rapidly destroy the patient who is unhappily affected with them. The distinction between "*innocent*" and "*malignant*" growths is, therefore, a most natural and obvious one. A very little further observation would bring to light the fact that whereas many of the former kind are like the natural tissues of the body—fat, or cartilage, or bone—many of the latter kind are quite unlike those tissues. And the microscope accentuated this difference, by showing that some soft, white, malignant

tumors, which by Laennec and others had been compared with brain substance or marrow, and called *encephaloid* or *medullary*, have in reality an intimate structure which bears no resemblance to normal tissues. Thus a fundamental division was made between *homologous* growths, generally innocent, and *heterologous* growths, generally malignant. And it was further supposed that, although there are several varieties of malignant growths, differing in character and appearance, yet all these belong to a single disease, which was termed Cancer. The *word* "cancer," or its equivalent *carcinoma*, was of much earlier origin; it dates, indeed, back to Celsus. But hitherto its tendency to ulcerate and to eat away the natural structures had been regarded as the fundamental character of this affection rather than the presence in it of any new-formed structures. Thus, not only had the venereal "chancre" been confounded with it, but even the "cancrum oris" of children, which we now term noma. Henceforth all of these were finally separated from it.

The early pathological histologists, of whom Lebert may be regarded as the chief, saw no difficulty in supposing that the tissue of a heterologous growth might differ *in toto* from all the normal tissues of the human body. They made it their aim to discover some particular specific element or "cancer cell," the presence of which might constitute an infallible criterion, and solve the frequent difficulties which arose in the anatomical diagnosis of tumors.

But in 1847, and the years which followed, the genius of Virchow placed this question in an altogether new light. It had already been shown by Johannes Müller that the structure of every growth, however homologous in appearance, always corresponded in its ultimate elements with some natural tissue; if not with any of the permanent tissues, at least with some one of those which exist during early embryonic life. Virchow carried the same idea into full detail. As he pointed out, the physiological type of the tailed and irregular cells which had been supposed to characterize cancer is to be found in the epithelium of certain mucous membranes; while the round or spindle-shaped cells which really make up the substance of many malignant tumors correspond with those which are seen during the development of connective tissue, or (as Billroth has since taught) of muscle or of nerve tissue. And, in general, he insisted on the principle that the structure of a tumor in man must necessarily be human; so that, for instance, such a tumor might contain hairs, but never feathers; whereas, in a bird a tumor might have feathers in it, but not hairs. For Virchow, therefore, no growth was *heterologous* in the sense hitherto attached to that term. The distinction, as he drew it, was between tumors resembling in structure the tissues in which they were placed, and those differing from them. Thus, he said that the same growth which in one situation would be homologous, would in another situation be heterologous. And he further pointed out the necessity of recognizing that between certain tissues of the body there naturally exist close relations which are altogether wanting between them and other tissues, and that such relations have important bearings upon pathology. Thus, all the structures belonging to the connective-tissue series being physiologically related to one another, the presence of bone, or of cartilage, or of fat in fibrous tissue, would not be inconsistent with homology, whereas that of epithelium would be. Such views have since undergone many further developments, to which I shall presently have to allude.

A still greater advance was made by Virchow in regard to the mode of development of tumors. It had before been generally held that cancer was something deposited from the blood. Chemists had made analyses in the hope of finding in it some peculiar principle for which the name "carcinomatin" had been invented in advance. Its cause was believed to be

a "dyscrasia" or altered constitution of the blood. The fact that active tubercle and cancer are but seldom found in the bodies of the same individuals was supposed to support this notion, it being imagined that the blood, or, at least, the formative part of it, could hardly present two different dyscrasiæ at the same time. But wounds and injuries undergo repair in exactly the same way in those who are affected with cancer as in other persons: which could scarcely be if the doctrine in question were true. Indeed, were that doctrine pushed to an extreme, I suppose that all parts of the body ought to become cancerous simultaneously, or at least all such tissues as are undergoing constant nutritive changes, for which they depend upon exudation from the blood.

Virchow, in accordance with his dogma *omnis cellula e cellulâ*, maintained that the substance of a cancer was developed *in situ* by the growth of the cells and other structures of which it was composed. He was, indeed, mistaken in the view which he advanced, that they arose by a proliferation of the connective-tissue corpuscles. But, after all, this was an insignificant detail in comparison with his rejection of the notion that cancerous and other tumors were merely "deposits."

One necessary consequence of Virchow's conception of cancer was that the so-called cancerous cachexia, instead of preceding the formation of malignant growths within the body, must be a result of their actual presence. And, so far as I know, no one has since appealed to definite clinical experience in a contrary sense. No doubt one of the earliest signs of an internal tumor is sometimes the fact that the patient becomes cachectic, that his skin acquires a waxy-yellow color, and that his features look pinched and sharp. But in such cases the tumor really exists long before the indications of it have appeared. It wears down its victim by pain and distress of mind, even if it does not attain a large size or undergo ulceration and hemorrhage. Lastly, it is certain that some innocent growths, when they are so placed as to cause severe pain and to give rise to much bleeding, are accompanied with a well-marked cachexia. I may cite, for example, certain uterine polypi, and even hemorrhoids and non-malignant tumors of the rectum.

Again, Virchow defined the term *malignancy* far more clearly than previous writers. Hitherto the pain caused by a cancer, and its tendency to destroy life, had been enumerated among the signs that it was malignant. He set these points altogether aside, and insisted that the real indications of malignancy were four in number:—

1. The *local progression* of a growth, its tendency to increase by extending into the tissues beyond it. A point on which much stress has since been laid, is the fact that malignant tumors show little or no respect to the natural boundary lines marking off one kind of tissue from another. Thus, I recently saw a case in which a cancer of the bladder, having doubtless started in the mucous membrane, ate its way through the whole substance of the organ, through both laminæ of the peritoneum, through the muscles and aponeuroses, and all the other structures of the abdominal wall, until it formed an immense ulcerated cavity, reaching from the pubes to the umbilicus.

There is, however, one exception to this power of overcoming the resistance offered by the different tissues, in the fact that malignant growths comparatively seldom penetrate the walls of the larger arteries, whereas they frequently extend into the interior of veins, even into the venæ cavæ. Mr. de Morgan remarked that cicatricial structures sometimes form a barrier to the spread of cancer, so that when recurrence takes place after an operation the disease creeps along one side of the scar for a considerable distance, without passing over to the other side.

It is a point of some interest that cancers are capable of passing across a serous cavity without previous union between the two surfaces. Of this I observed a very remarkable instance some years ago. The fundus of the uterus being occupied by a malignant growth, the great omentum hung down into the pelvis, so as to lie in contact with it; these parts were not in the slightest degree adherent to one another, but the lower edge of the omentum contained a mass of the disease. It is not improbable that infection in such cases is the result of active amœboid movements on the part of the cells of the primary tumor; Waldeyer and Carmalt ("*Virch. Anat. u. Phys.*," lv) have at any rate detected slow changes of form in cells from a cancer of the breast, and in those of a round-cell sarcoma of the axilla, when placed on a warm stage; they did not, however, observe them move from one spot to another.

2. Its *tendency to return in loco* after having been extirpated by the surgeon. This is one of the facts which used to be urged in favor of the constitutional origin of cancer. According to the modern view, it depends upon the circumstance that a malignant tumor always has outgrowths far beyond the area in which the tissues can be seen by the naked eye to be affected. But I ought in candor to add that I have not heard that surgeons who adopt the new theory are generally able to obviate the return of the disease by increasing, to whatever extent, the size of the mass which they excise. A point, however, which is worthy of notice, is that in the case of double organs, like the breasts, the removal of the whole of one gland for a malignant growth is not followed by recurrence in the organ on the opposite side, as might certainly have been expected if the constitutional view were correct. Were such an instance ever to occur, one would probably be right in regarding it rather as an example of the independent development of a primary cancer in each breast; for this sometimes, though rarely, happens when no operation has been performed.

3. Its *spreading to lymph glands*. As a rule, the glands first attacked are those which immediately receive the lymphatic vessels from the diseased part. But sometimes it seems to pass over certain peripheral glands, and to begin by affecting others, which lie nearer to the thoracic duct. Even glands which are altogether remote may become seats of the growth, but in such cases it probably has been brought to them by their blood vessels. There can be little doubt that the radicles of lymphatics around the margins of a tumor take up from it something which is carried into the lymph stream and deposited elsewhere. And there is no difficulty in supposing that this something consists of nuclei, or even of cells, from the growth; for Virchow has shown that sometimes an entire lymphatic network—beneath the pulmonary pleura, for example, or the peritoneum—may become filled with such materials, so as to appear as if it had been artificially injected ("Kr. Geschw.," p. 52, fig. 4). Indeed, as we shall presently see, Köster has endeavored to prove that the "local progression" of cancer takes place along the lymph spaces. Whether lymphatic channels exist in the interior of tumors is said by Cohnheim to be still open to question, although Van der Kolk long ago, as he believed, discovered them.

4. Its *becoming generalized*, i. e., *spreading to different organs and tissues by infection of the blood, or, as it is termed, "metastasis."*

With regard to this point a fundamental distinction must be laid down. It is not the mere multiplicity of tumors which indicates that they are malignant. Sebaceous cysts and fatty tumors are sometimes present in great number and yet are perfectly innocent. Perhaps there is no form of malignant disease in which so many nodules are found as in some cases of molluscum fibrosum. But all such innocent multiple tumors have their seat in some particular tissue, and remain limited to it. On the other

hand, cancerous growths may appear in almost every tissue of the body at the same time.

Again, it is a most important feature of most cases in which death has been due to malignant tumors that the pathologist can easily recognize some one mass of the growth as having been the starting point of all the others, either by its characters, or in consequence of what he knows as to the origin and mode of distribution of the particular kind of neoplasm. Even when such a primary growth is seated in an internal organ, and gave rise to no symptoms during life, it is almost always plainly distinguishable from the rest. The difference consists not so much in its greater size (though this may be sufficiently striking) as in its being of firmer consistence, which suggests that it developed more slowly; or an advanced state of degeneration, or extensive ulceration, may show that it is of earlier date. With regard to certain theoretical considerations which also assist in determining the question, I shall have much to say further on. It is, as a matter of fact, exceedingly rare to find in the same case two malignant tumors, with equal claims to priority.

Another curious circumstance is that organs and tissues which are especially apt to be the seats of primary growths seldom have a tendency to be affected with secondary or "metastatic" growths of the same kind. For instance, a primary cancer is very often developed in one of the breasts, but in such cases we do not commonly find that the opposite breast contains any of the secondary cancers when they appear; nor are they met with in the cervix uteri, another locality from which cancer frequently starts.

Again, in many cases the distribution of secondary nodules or tubera is so limited as clearly to indicate in what way they were derived from the primary growth. For example, a malignant tumor in the stomach or in the intestine may give rise to secondary nodules in no other organ except the liver, showing that something was carried by the blood of the portal vein which served to propagate the disease; or, the primary growth being in one of the limbs, the secondary growths may occur only in the lungs, so that the infection obviously took place through the systemic veins and the pulmonary artery. I have already remarked that malignant tumors often penetrate into the interior of veins; there is, therefore, no difficulty in understanding how nuclei, or cells, or even pieces of the growth, may get washed away by the blood current, and carried to distant parts. Cancerous thrombi form soft, colorless masses, parts of which are blended inseparably with the coats of the vessel, while other parts, often much elongated and lobulated, project into its channel, either perfectly smooth on the surface, or coated with a layer of fibrin. Some time ago I met with an instance in which, the primary growth being in the liver, and the hepatic veins extensively involved, the lower border of one lung contained a whitish-yellow wedge-shaped mass, exactly like an infarctus due to embolism from an ordinary thrombosis: Dr. Pye-Smith, however, under whose charge the case had been during life, found that it was really a secondary nodule of the tumor. Dr. Moxon mentions in vol. xviii of the "*Guy's Hospital Reports*," that he had twice seen cancer growing in clots which lay within the cavity of the right ventricle at its apex. He does not state that the growth had been brought there by the blood of the systemic veins, but I believe that this was the case. In 1871 Dr. Payne showed to the Pathological Society a heart in which both the right auricular appendix and the apex of the left ventricle contained similar cancerous thrombi projecting from between the muscular trabeculæ. In that instance, it must be supposed that the infecting agent traversed the pulmonary capillaries to get from one side of the heart to the other.

I must not omit to mention that the distribution of secondary growths is

often different from what would have been expected on anatomical grounds. Thus, I examined a case of epithelioma of the œsophagus, in which the lungs were healthy, but a secondary nodule existed in one kidney. Frequently the metastatic formation of cancer is limited to some particular tissue throughout the body, without any obvious relation to the seat of the primary growth. Thus, in a woman with cancer of the breast almost every bone may become cancerous, while all viscera remain entirely free from the disease.

To complete the description of the ways in which malignant growths multiply in the body, a fifth point should perhaps be added to those enumerated by Virchow, namely, the possibility of portions of a tumor becoming *transplanted* when set free upon a mucous surface by ulceration or otherwise. In 1868 Dr. Moxon showed to the Pathological Society a specimen in which he thought that an epithelioma of the œsophagus extending into the trachea had in this way infected the lungs. Secondary nodules were seated in the interior of the lower lobes, and not beneath the pleura, as is usually the case. They occupied the centres of lobules, and small tubes could be traced up to them. He also mentions that Mr. Simon had exhibited specimens in which "cancer germs" had appeared to take root in the bladder after descending the ureter from the kidney. But I think that all supposed facts of this kind should be cautiously weighed before they are accepted, for there can be no doubt that the particles detached from the face of a malignant growth are, as a rule, dead and already in a state of decay. It has been imagined that a woman with cancer of the uterus may infect the penis of her husband, but Cohnheim maintains that this never occurs. I believe that no experimental proof has yet been offered of the possibility of inoculating any kind of tumor from one animal to another. There has sometimes been a slight apparent commencement of growth at the spot inoculated, but it has always before long undergone reabsorption.

It must be particularly noticed that the above characters which Virchow laid down as distinguishing malignant from innocent new growths, so far from being peculiar to any special kind of tumor, belong also to other pathological processes, such as inflammation and tubercle. These exhibit "local progression," and they sometimes pay no more respect to the natural boundaries of the tissues than does cancer itself. The question of their "recurrence after removal" is seldom or never raised. But each of them "spreads to lymphatic glands." And inflammation, at any rate, assuming the form of pyæmia, undergoes "generalization" or "metastasis" through the blood. Surely, then, it is more probable that malignancy should belong to several kinds of new growths, perhaps in different degrees, than that it should be a fixed property of one particular species, isolating it from all the rest. Now, that is precisely the doctrine which Virchow taught, and is perhaps the most important part of his views. Other observers independently pointed out the impossibility of drawing an absolute line of distinction. Sir James Paget described, in 1853, under the name of "*recurrent fibroid*," a growth characterized by an inveterate tendency to return *in situ* after removal, without infecting distant structures. Dr. Wilks, in his "Lectures on Pathology," published in 1859, made a separate group of "semi-malignant" tumors, including, besides the "recurrent fibroid" of soft structures, the "osteo-sarcoma" of bone. And he specially recorded instances in which "myeloid" or "enchondromatous" growths, which were then generally regarded as innocent, appeared metastatically in the lungs. But by these writers cancer was still left as an independent affection, consisting of several varieties, of which the chief were scirrhus, or hard cancer; encepha-

loid medullary, or soft cancer; epithelioma, melanosis, osteoid cancer; and colloid, or gelatinous cancer. These forms of "cancer" were completely redistributed by Virchow, according to their histological characters. He showed that most instances of "encephaloid cancer," "melanosis," and "osteoid cancer" really belonged to the *connective-tissue* series of growths, in common with Paget's "recurrent fibroid" tumor and many others. Scirrhus he associated with epithelioma, both forms being characterized by cells of an *epithelial* type. Colloid cancer is now known not to be an originally special kind of growth, but the result of a peculiar degeneration.

Virchow's theoretical views have since been substantially adopted by all pathologists. But there are still some physicians and surgeons who think that in clinical practice it is advisable to use the word cancer for malignant tumors in general, without regard to their histology. In this I cannot agree with them. Histological characters appear to me to indicate the true relations of tumors, and therefore to be fundamental characters in their case, although I hold a different opinion in the case of tubercle. Nor does it seem that any practical advantage is to be gained by using "cancer" as a term of prognosis. We shall presently find that among new growths affecting the interior of the body, the seat in some particular tissue, the age of the patient, and other circumstances, enable one very generally to determine with accuracy what is likely to be the pathological nature of the disease, before we see or handle the affected part. To ignore all these points—which apply to the *histological* classification of new growths—is deliberately to deprive oneself of some of the most important means of tracing out the real origin and course of the disease. In describing the various kinds of tumors I shall frequently have occasion to illustrate my meaning. But one example which has recently occurred to me is perhaps worthy of special mention. An old woman died in Guy's Hospital with cerebral symptoms, and I discovered three tumors in the brain or in its membranes. From their appearance I felt convinced that they must be secondary to a primary growth elsewhere. But I searched every part of the body in vain, until at length I happened to notice that the extreme lower end of the rectum had not been taken out with the rest of the intestine. This, when removed, was found to contain a large ulcer with prominent fungating edges, which evidently was the lesion for which I was looking, although it had given rise to no symptoms during life; even the administration of enemata had failed to lead to its discovery. A microscopic examination of the secondary cerebral tumors afterwards showed that these were "columnar epitheliomata." Now, if I had been able to examine them previously, my task would have been very much more easy, for I would have known almost exactly where to look for the primary affection which gave rise to them.

It seems to be impossible to frame a satisfactory classification of tumors, or, indeed, of any group of diseases. But since I must follow some order in my description, I shall adopt the following arrangement, which is very like one proposed by Lücke, in Pitha u. Billroth's "Handbuch d. Chirurgie":—

I. Cystic Tumors.

II. New growths proper.

A. Of connective-tissue type.

(*a*) Normal or fully developed.

1. *Fibroma.*
2. *Lipoma.*
3. *Enchondroma.*
4. *Osteoma.*

(β) Embryonic.
5. *Myxoma.*
6. *Sarcoma.*

B. Of the type of lymph-glands.
7. *Lymphoma.*

C. Of epithelial type.
8. *Papilloma.*
9. *Adenoma.*
10. *Carcinoma.*

D. Of the type of higher tissues.
11. *Angioma.*
12. *Neuroma.*

Not all of these, however, require to be described, since I only propose to discuss such tumors as fall frequently under the notice of physicians; and, in this chapter, such as are found in various parts at once, so that they cannot fitly be left to a later part of this work. Thus, I shall altogether omit cystic tumors and those conforming to the type of the "higher tissues," and shall only say a few words of others.

New Growths of Connective-tissue Type.—1. *Fibroma.*—This is made up of fibrous tissue, which may be either arranged in loose meshes, or felted, sometimes very tightly. In the former case its substance is soft and succulent; in the latter it may be exceedingly hard, so as to creak when cut through. I shall have occasion to mention the occurrence of fibromata in various organs, as giving rise to more or less serious symptoms by pressure, or in other ways. Here it may be worth while to observe that in the kidneys it is not uncommon for several small growths of this nature to be found, which appear as soft white masses, and which are apt to be taken for secondary tumors when there is a growth in some other part the malignancy of which seems doubtful. I have more than once been saved from a blunder only by the microscope; in all other respects these renal fibromata are unimportant.*

2. *Lipoma*—This is a tumor consisting of adipose tissue, like the subcutaneous fat, but circumscribed, and generally enclosed in a well-marked capsule. When they occur in the interior of the body fatty tumors do not produce symptoms so as to come under medical observation unless they are so large as to press upon important organs, which is very seldom the case. But Dr. Frederick Taylor ("*Path. Trans.*," vol. xxvii) met with a case in which a lipoma grew in the post-pharyngeal space of a child, and caused death by suffocation. In the abdomen such growths have occasionally reached an immense size, so that they have been mistaken for ovarian tumors, and have been excised.

3. *Enchondroma.*—Tumors made up of cartilage are usually known by this name, although chondroma would seem to be the more correct form. Histologically they differ widely in different cases; the matrix may be either hyaline or fibrous, and the cells may present various characters. As primary growths they scarcely ever come under the notice of the physician. Virchow speaks of multiple enchondromata of the lungs, situated chiefly near their roots, and probably bearing some relation to the cartilages of the bronchia; but these, he says, are generally found quite accidentally in the post-mortem room.

4. *Osteoma.*—This is the technical name for growths which consist of

* English writers have, of late, been accustomed to separate, under the name of *Myoma*, a common variety of tumor containing much fibrous tissue, on account of the supposed presence in it of more or less numerous bands of smooth muscle. But all German pathologists seem now to be agreed that these growths, which are seen especially in the uterus and in the prostate, should be replaced among the fibromata; and Rindfleisch declares that the opinion that they contain muscular fibres is really a mistake.

osseous tissue, but it is not very often used. For when a tumor projects outward from a bone it is called an exostosis, and when it extends inward (as into a frontal sinus) it is sometimes called an enostosis; and most other bony growths contain soft structures as well, which are regarded as the more essential parts of them. I hardly know in what position an osteoma is likely to be observed in strictly medical practice unless it is as an intracranial exostosis, causing the symptoms of a cerebral tumor.

There is, however, a very remarkable kind of growth, for which the name *malignant osteoid tumor* seems to me the most appropriate, and which is of much interest to the physician, because it not infrequently comes under his notice rather than that of the surgeon. By Sir James Paget it was called "osteoid cancer." Virchow termed it "osteoid chondroma," a most unfortunate appellation, since (as he himself pointed out) the structure of the non-calcified part of the growth is not that of cartilage, but resembles rather that which belongs to periosteum. Moreover, as might have been anticipated, great confusion has arisen from its being supposed to be identical with an "ossifying enchondroma," which is a tumor really consisting of cartilage, the deeper and older parts of which have undergone conversion into bone. Malignant osteoid growths are made up of an exceedingly dense, firm and tough material, which is a pale grayish color, and which cannot be teased out. Under the microscope it may appear more or less distinctly fibrous. Sir James Paget describes the fibres as "crisp and stiff," or as moderately broad, "with uneven, thorny edges," and arranged in bundles "looking like faggots." Embedded in this substance are cells, which are rather small, of round or oval shape, without capsules. When calcification takes places, the salts may be deposited in hard, granular masses, but in some parts more or less perfectly formed bone may be seen. The calcified growth has a peculiar dull white, chalky, or mortar-like appearance. It cannot be cut, and must be sawn through, but it may be "rubbed or scraped into a fine, dry powder." Malignant osteoid tumors are most frequent in young subjects. Of nineteen cases collected by Sir James Paget, five occurred in persons between ten and twenty years old, nine in those who were between twenty and thirty. They are generally attended with severe pain. A very favorite seat of them is the lower end of the femur. I have seen one affecting the humerus close to the shoulder. This kind of growth forms a very large, hard mass, surrounding the whole circumference of the bone, slightly if at all nodulated on the surface, and gradually sinking down to the level of the rest of the shaft. Thus it might seem to be outside the bone, but on section the medullary cavity and the cancellous tissue are found to be completely occupied by the opaque, hard substance above described. When a flat bone is attacked by it, the growth is said by Sir James Paget always to project from both surfaces. I have seen a remarkable case in which the two iliac bones were affected symmetrically, each having a large, bossy prominence projecting from both the dorsum and the venter, which might have been felt during life. There may, indeed, be a large number of such tumors growing from different bones in the same patient, and the clinical aspect of the case may be that of a paraplegia from compression, or that of a cerebral tumor. Or, again, the main symptoms may be thoracic, from an immense mass lying at the root of the lungs, or from numerous nodules scattered in their substance and beneath the pleuræ. The secondary growths in lymph glands and viscera may have an appearance identical with that of the tumors of the bones themselves. They may be calcified in almost their entire extent, only a very little dry, tough, white, fibrous-looking substance remaining round their edges. It was this circumstance which so surprised the older pathologists, who almost imagined

that they had before them cases in which osseous tissue itself possessed the property of malignancy.

5. *Myxoma.*—The true relations of this form of tumor were first recognized by Virchow. Older synonyms for it are "collonema," "sarcoma gelatinosum," and "fibro-cellular tumor" (Paget), but in practice it was probably often confounded with "colloid." It consists of a semi-translucent material, sometimes so soft as to quiver like a jelly, of a bluish or yellowish tint, emitting a sticky fluid when squeezed or scraped. This comes from the intercellular stroma, and owes its viscidity to the presence of mucin. The addition of acetic acid to a thin slice of the growth renders it white and opaque, from precipitation of the mucin.

The cells are generally scattered at considerable intervals. They may be round, but they are often stellate, with long processes ramifying in the stroma. Virchow pointed out that this structure is identical with that of the mucous tissue, which in earlier fœtal life occupies the place of the subcutaneous fat, which at birth is found in the umbilical cord, and which constitutes the vitreous body of the eye. Myxomata are seen chiefly by the surgeon, but the physician may meet with them in various situations, as, for example, in the brain. Sometimes they are found in large numbers upon the nerve trunks, constituting a part of the tumors which were formerly incorrectly grouped together under the name of *neuromata.*

6. *Sarcoma.*—I have already remarked that Virchow referred to the *connective-tissue* series of growths as consisting of embryonic cells belonging to that order of tissues, certain tumors which present a high degree of malignancy, especially those known as "encephaloid" and "melanotic" cancers. He grouped them with other tumors of innocent character, or which at most exhibit a tendency to return *in loco* after extirpation, and he adopted for them all the name of sarcoma. His views with regard to them have since been universally accepted; in this country the more readily because no confusion could arise. For the word sarcoma, although it dates back to Galen, and had been employed by many subsequent writers down to the time of Abernethy, had fortunately fallen into complete disuse except in the compound form of osteo-sarcoma.

A point on which Virchow laid some stress as distinguishing sarcomata from growths of which the cells were epithelial, was the absence in the latter of any intercellular substance or matrix. And this still holds good, in so far, that where the cells of a tumor are embedded in a structureless or granular material, the conclusion may be drawn that they are not epithelial; but the converse is not universally true, for the elements of certain spindle-cell and other sarcomata are often tightly packed together without anything between them. Chemically, the matrix of a sarcoma generally contains, according to Virchow (Kr. Geschw., ii, 217), albumen, casein, or mucin, as well as gelatin.

The cells of a sarcoma may be of three kinds:—

(*a*) *Round Cells.*—These vary in character in different cases. Sometimes they are undistinguishable from leucocytes. In other instances they are much larger, and they are then often exceedingly delicate, so that the addition of water to a microscopical specimen may cause the disappearance of all but the nuclei, which then seem to be free.

(*b*) *Spindle Cells.*—These are the bodies which, by French writers, had been called *fibro-plastic*, and which had in England been recognized by Paget and others as characterizing the class of tumors which he termed "recurrent fibroid." They present many differences of size and appearance, but they all agree in having an elongated shape, tapering gradually into a point at each end, or having their ends prolonged as delicate, sinuous fibres. Their nuclei are always elliptical, and sometimes narrow

and oat-shaped. They are commonly arranged side by side, in bundles, or, as it is termed, are "fasciculated."

(*c*) *Giant Cells.*—Under this name are now described certain bodies, which, however, are rather irregular masses of protoplasm than cells, and which contain round or oval nuclei to the number of ten, twenty, thirty, or even more. From similar elements being found in the medulla of bones, especially during fœtal life, as Kölliker and Robin pointed out, they were until recently termed "myeloid" cells, or by French writers *myéloplaxes.* Indeed, the growths in which they occur, and which are comparatively infrequent, were by Paget, Wilks, and others, described as a special kind of tumor. Virchow, however, insisted on the fact that such growths always likewise contain spindle cells, and that therefore their true place is among the sarcomata.

But different kinds of cells by no means constitute the whole of the formed elements of a sarcoma. It also contains blood vessels, which are sometimes so large and receive so abundant a supply of arterial blood, that it may pulsate, so as to be mistaken for an aneurism. The walls of the finer ramifications of these vessels are usually themselves of embryonic character, consisting of spindle cells applied to one another in such a way as to enclose a blood channel between them. It is, therefore, not surprising that they are apt to allow blood to escape into the substance of the growth, where it may coagulate, so that on section the mass looks like a mere clot. Such tumors, of course, increase in size with extraordinary rapidity; and they were formerly known as *fungus hæmatodes,* a name now happily almost forgotten. In other instances, there are so few cells between the vessels that there may be the greatest difficulty in recognizing the presence of any new growth. Thus, in 1877, I made an autopsy in the case of a lad, aged fifteen, who had been under Mr. Cooper Forster's care for a fracture of the femur, which seemed clearly to have been caused by external violence. Repair did not take place; an incision led merely to the escape of blood, and amputation was followed by the death of the patient. I found the injured bones and muscles soaked in blood, and at first I had no suspicion of there being any tumor elements. But presently I noticed that the blood-stained appearance of the muscles was limited definitely by convex margins; and the microscope showed masses of delicate spindle-cell tissue in them. In the lungs there were five or six scattered secondary nodules, the size of marbles, and reddish in color, projecting above the level of the pleural surface. These, however, felt quite soft; and when cut into, each of them collapsed, leaving a cavity surrounded only by a very narrow margin of indefinite-looking tissue.*

Again, instead of being embedded in a merely granular material, the cells of a sarcoma may be supported by a formed stroma, consisting of connective tissue. This, however, is hardly to be regarded as equivalent to the ordinary intercellular substance, for the fibres have no doubt been developed out of a corresponding number of the cells, and have taken their place. The growth is, in fact, a mixed one, a *fibro-sarcoma.* Sometimes, as Billroth especially has pointed out, there is a regular alveolar structure, exactly like that which characterizes the carcinomata. Of late years, since it has been thought that cancers proper can arise only where there is epithelium, such "alveolar sarcomata" have acquired great importance, as enabling pathologists to account for apparent exceptions to this rule.

Still greater complexity is caused by the combination, in the same tumor, of a variety of other structures belonging to the connective-tissue series, such as cartilage, mucous tissue, bone or fat. It is a question whether the occurrence of certain rare cases in which a growth, ordinarily innocent, undergoes

* For further details of this case see the "*Guy's Hospital Reports*" for 1880, p. 17.

metastasis to distant organs must not be explained by the presence in it of more or less numerous embryonic cells, the diffusion of which by the blood-stream causes the infection. According to this view all such tumors are "mixed," and should be designated accordingly. I have myself met with an instance in which fibromata of the uterus had led to the formation in the lungs of similar growths, in which the appearances regarded by almost all pathologists as indicative of the presence of smooth muscular fibres were as conspicuous as in the uterine tumors themselves. And in another case, that of an infant fifteen months old at the time of death, there were in the liver, secondary to a mixed sarcoma of the neck, a number of dense, flat, button-like nodules, which consisted almost entirely of well-developed fibrous tissue, although at the margins some spindle-cell elements were, with a little difficulty, discovered.

Another circumstance which greatly modifies the appearance of sarcomatous growths is the occurrence in them of degenerative changes, of which caseation is the chief.

It is important to inquire whether, without having recourse to the microscope, one can distinguish the different varieties of sarcomata from one another. And I think the reply must be that this is often impossible. Different specimens vary greatly in consistence and in color; but there is no constant relation between their characters in these respects and their histological structure. However, I am under the impression that the softest and whitest among them—those which would be termed *par excellence*, "encephaloid" or "medullary"—consist rather of round than of spindle cells. The statement is commonly made that encephaloid tumors are almost always sarcomatous, thus implying that exceptions to the rule do sometimes occur. But I think that tumors of epithelial type never have so homogeneous, white, glistening an appearance as is presented by many sarcomata; and that the only cases in which they are equally soft are those in which they grow into the interior of a free space, as into the enlarged pelvis of a kidney, or into the channel of a vein, where they meet with scarcely any resistance. On the other hand, the giant-cell sarcomata are often recognizable, without difficulty, at the first glance: they are blotched, or uniformly tinted with a dull crimson, contrasting here and there with a grayish hue.

But in many instances the primary seat of a sarcoma enables an accurate guess to be made as to its histological characters. Thus a growth which starts in the submucous tissue of the alimentary canal or in one of the solid viscera is but little likely to be made up mainly of spindle cells. The fasciculated sarcomata belong especially to the periosteum and to the fasciæ. Giant-cell sarcomata, on the other hand, arise usually in the cancellous substance of bones, and spread out the compact tissue over them. They also often appear in the form of an *epulis*, a growth projecting into the mouth from the alveolar processes of the jaws.

Melanotic Sarcoma.—Of all the sarcomata, that which is most definitely localized in its origin is the *pigmented* or "*melanotic.*" This constitutes the larger part of what was formerly known as "melanotic cancer." Rindfleisch, indeed, seems to imply that even when a pigmented growth possesses an alveolar structure it is yet likely to be a sarcoma, belonging to Billroth's "alveolar" form. But most writers allow that true carcinomata are sometimes melanotic, and I have myself examined one specimen (from the skin of the neck) in which the cells lining the walls of the alveoli were distinctly columnar in form, and arranged side by side, so that their epithelial character could not be doubted. Still, one may, for practical purposes, continue to describe pigmented growths under the sarcomata.

As a rule, they start from some part of the body which normally contains pigment, generally from the skin or from the choroid coat of the eye,

sometimes, according to Virchow, from an adrenal body, or a vesicula seminalis.

A curious fact, which was illustrated by a case in my own knowledge, is that the rectum sometimes becomes the seat of a primary melanotic tumor. And white or gray horses, which have a deficiency of cutaneous pigment, are extremely liable to be affected with a pigmented growth, situated about the anus or upon the tail, although this is said to be of a comparatively innocent character. Many cases of melanotic sarcoma in the human subject are developed from pigmentary moles, which may have existed from birth, and have, at any rate, remained for years without causing the slightest inconvenience to the patient. Such a mole may begin to grow, or it may itch, provoking the fingers to scratch it; and then, within a very few months, a fatal diffusion of secondary growths from it may occur. Some observers, including Mr. Hutchinson, even think that the development of multiple melanotic sarcomata throughout the body may, in the absence of any more obvious starting point, be accounted for by the discovery upon one of the toes or elsewhere of a flat pigment mark, which may yet show no sign of having taken on active growth. But I should, for my own part, be disposed to place such cases rather under the head of a general diffused sarcomatosis.

It is to be observed that the distribution of pigment in melanotic tumors is exceedingly capricious. Whole nodules may be uniformly black as ink, but, perhaps, others in the same case are purely white and medullary, and yet others may be of a gray color, or streaked or marked with black patches and lines. Where there is no alveolar structure, melanotic sarcomata usually belong to the spindle-cell variety. In many instances they are extremely numerous, and they may be thickly scattered in regions and tissues which are comparatively seldom affected by new growths. Thus there may be many of them along the course of the intestine, with ulcerated surfaces projecting into its interior. And others may be found in the spleen, in the thyroid, in the muscular substance of the heart, and, in fact, in every part of the body.

Mixed with the tumors and nodules, there are sometimes black spots or patches, which look as though they were mere deposits of pigment, without any new growth. I have observed this, for example, in the mucous membrane of the stomach, of the renal pelvis, and of the bladder; and I have seen parts of the lungs stained black in a way which seemed to me to be peculiar. Lücke, indeed, says that a careful examination always reveals the presence of cells in spots thus affected. But Thiersch, relates a case of melanotic carcinoma of the skin in which, at certain points, nothing could be discovered but a diffused brown staining of papillæ, the texture of which was perfectly normal. And it is certain that pigment is often excreted in the urine in large quantities, altogether independently of the occurrence of melanotic growths in the kidneys or in the bladder. Two instances of this were brought, by me, under the notice of the Pathological Society, in 1876. It had previously been described by Eiselt and by other foreign writers. Urine so affected may have an olive-green hue when voided; it becomes darker on exposure to the air, and the addition of nitric acid turns it quite black. It may throw down a deposit consisting partly of minute granules, partly of rounded, brown, translucent bodies resembling nuclei, partly of casts of the uriniferous tubules, brown or black with pigment. The coloring matter, which is known as *melanin*, can be separated in a pulverulent form, and when suspended in water, it remains unchanged for years. Certain of the tumor masses may be colorless, even in cases attended with melanuria. It therefore seems clear that the pigment is originally manufactured, not in the circulating blood,

but in the substance of the growths, which are themselves black. It is probably derived from hæmatin, but no transitions in orange or reddish-brown tints are to be recognized. One can only suppose that it is formed in the tumor in such large quantity that a surplus becomes reabsorbed, and is carried to the kidneys by excretion. The clinical recognition of melanuria may sometimes be of great help in the diagnosis of an obscure internal tumor, and it must not be confounded with the blackening of the urine which may appear when carbolic acid, creasote, or tar in any form, has been applied to the skin or swallowed.

Multiple Sarcoma.—To the physician it is a point of great practical importance that the development of sarcomatous growths in the interior of the body is sometimes accompanied with symptoms such as by no means suggest the real nature of the case. In some instances, the chief thing of which the patient complains is pain, which may either be fixed in certain parts or widely diffused, and may vary in seat from time to time. More or less rapidly advancing emaciation and anæmia complete the clinical features of the disease, the pathology of which sometimes remains obscure to the very last. The following cases are examples :—

In 1880, a man, aged forty-six, was under my care in Guy's Hospital, who had originally been admitted for what was regarded as a gouty affection of the right hand and of the left great toe. This had set in about a year before his death. At a later period, no evidence of gout could be discovered. He continued, however, to complain of pains, which were called rheumatic, in his right arm, in both shoulders, in the loins, and (especially during defecation) in the lower part of the spine. He also suffered severely from headache, which, it was thought, might, perhaps, have resulted from a previous injury. The urine was at one period albuminous, and the case was therefore regarded as one of Bright's disease of gouty origin. Subsequently, the urine became normal, and some of those who saw the patient at that time suspected that he was exaggerating his symptoms. However, he grew more and more wasted and bloodless, and his pains became fixed in the iliac fossæ, especially on the right side. The most careful search was made for tumors, which it seemed could hardly escape detection in so emaciated a subject. A few weeks before death hæmaturia occurred, and this, of course, led to the suspicion of primary malignant disease of the kidney. At last he became unconscious, with rigid flexion of the right arm, and in this state he died. The autopsy showed that sarcomatous growths had occurred to an enormous extent, but that there was nowhere any tumor discoverable by manipulation. In each iliac fossa, a layer of sarcomatous tissue was spread out beneath the periosteum, and there was a large quantity diffused over the surface and in the interior of many other bones. One tumor, which was removed, and sawn vertically through, had in its cancellous tissue many rounded masses as large as walnuts. On the under surface of the dura mater there were a number of button-like nodules indenting the brain. One kidney had two small tumors affecting the mucous lining of its pelvis. The growth consisted of large cells of the most irregular form, embedded in a fibrous matrix.

Another case, is that of a boy, aged sixteen, who came under my observation when I was clinical clerk to Dr. Rees, in 1854. He said that he had been strong and robust until eleven weeks before his admission, when he began to suffer from pain, at first in the loins, and afterwards in the shoulders and limbs. There was great tenderness of the whole surface of the body. He had also experienced a sensation of tingling in the area of distribution of the ulnar nerve to each hand. He was a very delicate-looking lad, with a clear complexion, a pink flush on each cheek, a moist

skin, a white tongue, and a pulse of 144. The first diagnosis was of subacute rheumatism; subsequently of chronic inflammation of the spinal membranes. He lay for about two months, during which time he became emaciated to the most extreme degree. Towards the last, masses of enlarged glands could be felt through the walls of his contracted abdomen. He continued to suffer excruciating pain on the slightest movement, and there was exquisite tenderness of the skin. One day he became insensible, and had a convulsive attack, after which he lived only three hours. A large mass of medullary growth was found in front of the spine, eroding the vertebræ, and involving the nerves as they emerged from the spinal canal.

Sometimes the growth of multiple sarcomata leads to a still more rapidly fatal illness. In vol. xxv of the "*Guy's Hospital Reports*," I have recorded certain cases which were attended with purpura and with hemorrhages from mucous surfaces. One of the most curious is that of a man, aged twenty-five, who came under Dr. Pye-Smith in 1879, for what was supposed to be an attack of rheumatism. He said that on Whit-Monday, June 2d, he had got wet through; and from that time up to his admission, two months later, he had complained of pains in the left shoulder, in the chest, and in the hips. He had been confined to bed for nine days with profuse sweating; the temperature varied from 102° to 102.3°. The skin had an unpleasant, sour smell, and a systolic apex murmur was detected on one occasion when he sat up. He went on well for twelve days, when a purpuric rash came out on the chest and the abdomen, hæmaturia occurred, and his eyelids and his scrotum became greatly swollen. Five days later, on August 14th, he died. At the autopsy the scattered spots of purpura still remained visible. Some of them were flat, but others were slightly raised and indurated, and a few had a central pale elevation with a narrow ring of purple discoloration around it. These proved to consist of small, round or irregular cells infiltrating the little lobules of subcutaneous fat. In the kidneys there were several white or pinkish sarcomatous nodules. In the lowest two inches of the ileum there was what appeared to be the primary growth, a homogeneous-looking yellow mass, of considerable thickness, involving all the intestinal coats.

Another case occurred in the person of a cab-driver, aged thirty-eight, a patient under Dr. Moxon in 1877. Up to five weeks before admission he had always, he said, been a strong man. He then "caught cold, from being exposed to draughts." After this he suffered from pains in the back, legs, arms and chest, chiefly in the joints, and flying from one joint to another. He was restless and hot at night; he had profuse, sour sweats; his urine was high-colored and became thick on cooling. Five days before he came into hospital he was attacked with severe pain in the back, hæmaturia set in, and purpuric spots came out on the neck, the groins, and the legs. Afterwards he had epistaxis, his gums became sore, and bled. His temperature had been taken before admission, and was found to range from 100° upwards. On admission he was much blanched and extremely weak, with no perceptible pulse. The nose began to bleed almost immediately, and on the following morning he died. The kidneys were found to contain a large number of white tumors, and there was in the right vesicula seminalis a firm growth, to which they were apparently secondary.

A third case was that of a man, aged twenty-eight, admitted under the care of Dr. Wilks in 1872. He said that for some weeks he had had bad headache and neuralgia, that his teeth had been loose and painful, and that his gums had been spongy and had bled. For ten days he had been suffering with severe pains in the elbows, the knees, and the shoulders, but these seemed to be diminishing in intensity. He was an anæmic man, with a brown tongue, foul breath, and teeth and gums caked with dried blood.

His pulse was 128, his temperature 100.2°, his respirations 28. His gums and his nose continued to bleed, and eight days after his admission, raised purpuric spots appeared on the abdomen. On the following day he became unconscious and died. A whitish, firm growth was found in the anterior mediastinum, probably affecting the thymus; and there was a large quantity of a similar material in the subserous tissue of the peritoneum and in the mesentery.

A fourth case, somewhat like the others, came under my observation in February, 1876. The patient, a bank clerk, aged twenty-five, said that he had got very cold about a fortnight before Christmas, 1875; for three days he was chilled through, and he was never well afterwards. He visited me in the morning, and I then found his temperature 101°. His mouth and his tongue were stained with blood, his gums were slightly spongy, he had purpuric spots on the legs. It appeared that he was not in the habit of eating any vegetables, and I supposed the disease to be scorbutus. But it soon became apparent that this was an error; he rapidly grew extremely anæmic and feeble, hemorrhage from the bowels set in, the lymph glands in various regions of the body became enormously large, and at the end of a few months he died. There was no autopsy.

As I have remarked in my paper on this subject, different views may be taken as to the relation between the symptoms which presented themselves in these various cases, and the underlying malignant disease. It may be that the purpura, the spongy state of the gums, the epistaxis, the fever, and the rheumatoid pains are mere results of a profound alteration of the blood, analogous to that which exists in idiopathic anæmia, in splenic leucæmia, or in scorbutus. But another possible explanation of the purpura may be that a minute development of sarcomatous tissue, with vessels made up of embryonic cells, occurs at each spot which is the seat of an effusion of blood; or, perhaps, that sarcomatous cells, or nuclei, or even leucocytes in an abnormal condition, become lodged in the capillary walls, and produce softening of them, after the manner of emboli.

In some other instances, scarcely less obscure at the bedside, the diffused development of sarcomatous growths is indicated by other symptoms beside those mentioned in the last few paragraphs. One is the formation of nodules in and beneath the skin, which can be felt and seen. Such nodules, indeed, were present in the case of the clerk, above cited; but I committed the mistake of supposing that they belonged to an accidental "*molluscum fibrosum.*" It would, I believe, be possible to guard against a similar error in the future by remembering the peculiar discoid shape of molluscous growths, which generally have a flat under surface and a convex upper surface, uniting in a distinct edge or border. In 1876, Dr. Moxon had a patient under his care in whom the chief symptom was wasting, until sarcomatous nodules appeared in and beneath the skin of the chest and of the limbs, and cleared up the diagnosis.*

Another symptom of the formation of multiple sarcomata, especially when they affect the bones, is a gradual enlargement of certain parts of the skeleton. Thus, in one of my cases the calvaria was found to be very thick, and its whole substance was homogeneous and opaque; the bodies of the vertebræ, the ribs, and one *os innominatum* were also found to have their cancellous tissue converted into a dense material. Similar changes were observed in two instances recorded by Dr. Goodhart in vol. xxix of the "*Transactions*" of the Pathological Society; and Dr. Cayley

* I have since seen a case which was believed to be of a similar kind, until at the autopsy the subcutaneous tumors were proved to be cysticerci. Their real nature might, however, have been recognized, if one had thought of it, by their peculiar shape and size; they were firm elliptical bodies, very like a French olive in outline, but somewhat smaller.

relates in the same place the case of a man who came under Mr. Nunn for chronic enlargement of the lower jaw; his left clavicle was also much thickened, and both tibiæ curved forward. This patient died of malignant disease of the lung and of the liver. But the most remarkable instances are those which were brought, by Sir James Paget, under the notice of the Royal Medical and Chirurgical Society, in 1876, under the name of "Osteitis deformans." In these the diffused osseous change seemed to precede the development of any local tumor by many years. Thus, one case was that of a gentleman who, at the age of forty-six, became subject to aching pains in the lower limbs; the left tibia grew nodular, uneven, and broad; the skull gradually enlarged, so that its circumference increased, in the course of twenty years or more, from 22½ to 27¾ inches; the spine became curved and shortened, and stiff. He died, at last, of a malignant tumor of the left radius. The calvaria was found to be about four times the normal thickness; its texture was in parts finely porous, in parts compact. Another case, very similar in its character, ended, after many years, in the formation of a medullary tumor of one humerus. Probably there is an analogy between this simple affection of the bones, leading, ultimately, to fatal malignant growths, and the diffused ichthyosis of the tongue which terminates in cancer. To the pathologist it may probably continue to appear necessary to regard the successive stages of each disease as distinct. But, from a clinical point of view, it is important to keep before one the significance of a thickened tibia or clavicle in an obscure case, that may possibly be one of sarcomatous growth; and it may be of great value in diagnosis to elicit the fact that a patient's hat has become too tight for him, or to observe an alteration in his attitude, the chin projecting forward and downward, the arms hanging, and the legs being shortened and bowed outward, as in Paget's cases, and in one which had been recorded some years before, by Wilks.

I have already remarked that the occurrence of a large number of melanotic sarcomata in the same patient sometimes appears to be independent of any infective process—that, in fact, they are multiple rather than malignant. The same thing may, I believe, be said of other forms of sarcoma, as in the above cases, and likewise of the lymphomata which are next to be described. Not only is it often impossible to discover any one growth which can be regarded as standing toward the rest in the relation of primary to secondary; but the tumors themselves may fail to present Virchow's first indication of malignancy, that of "local progression," without respect to the natural configuration of the parts affected. For instance, I have seen a sarcoma of an undescended testis, weighing eight pounds, which had on its outer side a detached piece, bearing the very same relation to it, in position and in size, which the epididymis has to the healthy organ. And in another case, one ovary, although of normal shape, was about twice the length of the opposite ovary, and its substance was opaque, white, and made up of rounded sarcomatous cells. Another point, which is perhaps worthy of notice, is that in young adults who die of sarcomatous growths, the thymus is often found presistent, if not rejuvenescent.

7. *New Growths of the Type of Lymph Gland Tissue.*—Among the primary forms of tumor, in Virchow's classification, is one to which he gave the name of *lymphoma*, and of which he defined the structure as being identical with that of a lymphatic gland or of a solitary follicle; the leuco-cytogenic, or "adenoid tissue of His." It is made up of cells, which lie in the meshes of a stroma. The cells are leucocytes, and can be more or less readily removed by penciling a thin section of the growth. The stroma, which then becomes visible, is characterized by being "reticulated;" that

is to say, its fibres not merely cover one another, but branch and unite together, many of the points of union being themselves nucleated, so that they may be fairly compared with fixed connective-tissue corpuscles, triangular, or stellate in form.

The majority of the lymphomata, enumerated by Virchow, really lie beyond the definition of tumor, as we now understand it. He included among them not only tubercle, but even the transitory growth to which enteric fever gives rise in the follicles of the intestine; just as under the term "Granulation tumors," he described the various lesions which are produced by syphilis, leprosy, and glanders. In fact, he seems almost to have aimed at gathering together, for the purposes of his great work, all the more important structural changes which belong to the science of pathology. This, however, is incompatible with the order which I have found it necessary to adopt, although, as I have already admitted, it would be far from easy to define the word tumor scientifically.

Even when the lymphomata have been reduced within very narrow limits, they still constitute a vague and unsatisfactory class of new growths. Dr. Green, in his "Introduction to Pathology," says that they include three varieties; "simple lymphoma," "lymphadenoma," and "leukæmia." Two of the examples which he cites of "simple lymphoma," are cases of enlargement of one or more of the lymphatic glands in some particular region of the body, and those of overgrowth of the intestinal follicles, forming polypi. But I think it would be very difficult to say how many of the cases in question may not really be of an irritative or inflammatory origin, or else may not belong to the early local stage which sometimes occur in Hodgkin's disease. And as for "leukæmia," I shall hereafter have to discuss whether the lymphoid growths found in that affection are not merely accidental, the result of the leucocytes into which the blood is overcharged being deposited in the interstices of the tissues. There remains, therefore, only the third of Dr. Green's varieties, that which he terms "Lymphadenoma." This word is one which has tended greatly to confuse the whole subject. By many French observers, including Cornil and Ranvier, it is used to include all forms of tumor of which the structure is identical with that of "adenoid tissue." On the other hand, few German pathologists employ it at all. And we should, I think, be strictly correct in stating that its true position in the nomenclature of disease is that of being a French equivalent to the German term Lymphoma. Dr. Green, however, like several other English writers, applies it in a more limited sense, to correspond with what I shall describe elsewhere as "Hodgkin's disease." In that morbid condition there are, indeed, growths which spring up in immense numbers, and sometimes with extreme rapidity, not only in the lymphatic glands and the spleen, but also in almost all other organs and tissues; and they may possess at least the first of Virchow's characters of malignancy, spreading from one part to another without regard to the natural boundary lines between them. But it is nevertheless impossible to reserve for them any such name as "lymphadenoma," or even to describe them under any one pathological designation. For, as we shall hereafter find, their historical characters vary very widely in different instances, so that we must regard "Hodgkin's disease" as a clinical name for a group of cases in which the same organs are affected, and of which the symptoms and course are similar, but not as anatomically denoting any one special kind of tumor.

This brings me to another term, "Lymphosarcoma," which also needs to be explained. Like "lymphadenoma," it has been employed in somewhat different senses by different writers. Some seem to apply it to any sarcomatous growth having its starting point in lymphatic glands; they forget

that the seat of a sarcoma is in itself a matter of indifference, and certainly does not require to be marked by any special name. On the other hand, Virchow is disposed to designate as lymphosarcomata all lymphomatous tumors of any size, of which the elements show no tendency to caseate, and which undergo progressive and sometimes very rapid enlargement; and he would include under this head most instances of Hodgkin's disease, and all the lymphomatous tumors of the mediastinum. But it is certain that many of the cases in question, even where a large number of organs are involved, exhibit no histological characters that can be said to differ from those of a pure lymphona. And it seems to me that the only logical course is to reserve the name of lymphosarcoma for such growths as appear to be transitional between a lymphoma and a sarcoma.

New Growths of Epithelial Type.—As I have already remarked, we owe to Virchow the very important doctrine that the "cancer cell," which was believed by histologists to be heterologous, *i. e.*, unlike any of the elements of the healthy body, is, after all, identical with the cells of epithelium. He pointed out this fact explicitly in a paper in the first volume of his "*Archiv*," published in 1847; and at the same time he indicated the region in which the most perfect representatives of cancer cells are to be found, namely, upon the mucous surface of the ureters and of the bladder, especially in infants. He also made a striking comparison between the alveolar structure of a cancer and that of the lung, in a case of lobular pneumonia, where the pulmonary vesicles are filled with exudation which is just turning into pus. It is a remarkable fact that, at a much later date, pathologists, still ignorant of his teaching, actually committed the mistake of diagnosing malignant growths of the urinary passages or in the lungs, on microscopical evidence alone, in cases where no disease was present.

It is much to be regretted that Virchow did not carry out his usual method of nomenclature, and stamp with his authority the practice of applying the term epithelioma to all *tumors of epithelial type*. Unfortunately it was already in use, as a designation for a very limited group of cancers, characterized, however, not by the presence of ordinary epithelial, but by that of horny epidermic cells. But I believe that this inappropriate limitation of it will hereafter be given up, and that "Epithelioma" will universally be employed, as many observers do even now, in the wider sense which seems naturally to belong to it.

Epithelial growths, however, are not all of them included under cancers. There are certain innocent forms of them, two of which respectively bear the names of *papilloma* and *adenoma*. These bear precisely the same relation to one another which an intestinal villus does to a Lieberkühn's tubule. The former consists of a solid protrusion outward of mucous membrane or of skin, upon which epithelial cells are arranged; the latter is a hollow inversion of mucous membrane or of skin, with an epithelial lining. They each come under the notice of the surgeon rather than of the physician; but I may briefly mention certain instances of them.

8. *Papilloma.*—Of this we have examples in common cutaneous warts, and in the horny growths which are sometimes seen upon the face, neck, or chest. Another common variety occurs on the genital organs. Yet another form is sometimes met with in the larynx, especially at an early period of life. Lastly, there is an affection to which the vesical mucous membrane is liable, and which is known as "villous tumor" of the bladder, or sometimes (on account of the alarming and dangerous symptoms to which its gives rise) as "villous cancer." It consists in the formation of one or more tufts of long, soft, delicate-branched filaments, each containing a wide vascular loop, and covered with a series of layers of large epithelial

cells. I believe that this kind of new growth is never found to spread to lymph glands, nor to produce metastatic nodules in distant parts. But Billroth still continues to place it among the carcinomata, on the ground that masses of epithelial cells lie in meshes at the bases of the villi. I have myself observed one case in which the resemblance to the structure of a cancer was certainly very close.

9. *Adenoma.*—Of this form of tumor, having its seat in the skin, an instance may be found in Molluscum contagiosum. Other examples also are met with, corresponding some with the sebaceous, some with the sudoriparous glands. Upon mucous membranes the most common variety is one which constitutes a large number of so-called "polypi," growing from the surface of the intestine, of the cervix uteri, or of the nasal fossa. In the female breast adenomata very frequently occur.

10. *Carcinoma.*—It is to be observed that the structure of the two kinds of new growths last described is by no means always identical with that of the part in which they arise; thus a nodule of molluscum contagiosum differs considerably from the healthy skin in its histology, and a villous tumor of the bladder from the vesical mucous membrane. And I think that now it is impossible to maintain in its integrity the dictum of Waldeyer, which is cited by Lücke with approval, that the carcinomata can be absolutely distinguished from other epithelial growths by their being "atypical." Probably the real fact is that, as in so many other departments of pathology, no positive line of demarcation exists. The carcinomata, indeed, may be said to combine in many cases the distinctive features of a papilloma with those of an adenoma, in so far that they consist partly of outgrowths covered with epithelial cells, partly of inversions lined with them. Thus a cancer of the skin often begins in a growth resembling a wart. Many cancers of the stomach or of the intestine, or of the gall-bladder, present very abundant villous processes. At the os uteri, nothing is more common than for a cancer to begin as what is termed a "cauliflower growth."

In briefly sketching the histology of the carcinomata, it will, I think, be the most convenient plan to begin with what may be termed "*simple carcinoma*," the common type of cancerous growths affecting glandular organs and mucous surfaces in general. Afrerwards I must describe separately two varieties which occur, the one in connection with skin and with mucous membranes lined by laminated pavement-epithelium, the other with mucous membranes lined by columnar epithelium; these may respectively be called "*keratoid carcinoma*," and "*columnar carcinoma.*"

(*a*) *Simple carcinoma* presents a great many varieties of structure according to its exact seat. But it is always made up of oval or rounded alveoli, which contains masses of cells like those of epithelium. The walls of the alveoli usually consist of fibrous tissue, and they are more or less abundantly supplied with blood vessels. In very soft, rapidly growing cancers, however, they may be composed of spindle cells; or, in other words, their tissue may be sarcomatous. Otherwise the softness or hardness of a carcinoma depends chiefly upon the proportion between the thickness of the alveolar walls and the size of the alveoli themselves. In many instances the substance of the growth is as easy to break down as that of a healthy spleen, or even of a spleen from a case of fever; and then one finds that the alveoli are large and that their walls are made up mainly of blood vessels, and have but little fibrous tissue in them. But sometimes it is so hard as to be spoken of as "stony;" the microscope then shows that it consists almost entirely of interlacing dense white bands, the alveoli being very small, and perhaps so few in number that their presence may easily be overlooked. It is to such growths that the old name of

"scirrhus" is still applied. They are commonly very slow in their course, and they contract and shrink, so that the general appearance is often that of a cicatricial relic, rather than of a tumor; and if they occupy the circumference of a hollow canal (such as the intestine or the common bile-duct), they greatly narrow it. I shall hereafter have repeated occasion to insist upon the caution which is required in accepting statements as to the occurrence of fibrous non-malignant growths in such situations.

As to the extent to which the walls of the alveoli of a simple carcinoma are a new formation there is still some uncertainty, and perhaps different cases differ in this respect. The analogy of keratoid carcinoma, which will be described further on, suggests that they may be in great part made up of pre-existing tissue elements, modified in their arrangement by the pressure of the masses of epithelial cells which are in contact with them on all sides. On the other hand, it is certain that in growing cancers the alveolar walls generally contain numerous leucocytes, from which a new fibrous tissue may well be supposed to be developed. And, as Waldeyer remarks, when a carcinoma extends along the interior of a thrombus within the channel of a vein, its alveoli can be nothing else than entirely new formations. I must confess, however, that in some cases of this kind the alveoli have appeared to me to be but very little marked.

In thin sections of a carcinoma the alveoli look like closed spaces, but in reality they freely communicate with one another in different planes. Thus, when one gently squeezes the cut surface of such a growth, or when it is scraped with a knife, the cell masses, with more or less of an albuminous fluid, escape readily, forming what has long been known as the "milky" or "cream-like" *cancer juice*, or appearing as a soft, curdy, solid substance. The cell masses themselves—the "*cancer bodies*," as Waldeyer proposes to call them—may sometimes be withdrawn in continuity from two or more alveoli so as to have a branching or reticulated appearance. The cells of which they are made up present every variety of shape, "polymorphism" being, indeed, one of their characters. They have commonly very large, round or oval nuclei, and sometimes two or even more of them. The protoplasm is abundant, it is often granular, and it has no definite cell wall.

(*b*) *Keratoid Carcinoma.*—This is the form of growth which is commonly known in England by the inappropriate name of "Epithelioma," said to have been originally given to it by Hannover, in 1852. Foreign writers, following Lebert, often term it "cancroid." What characterizes it is the fact that some of its cells undergo a chemical change identical with that which constitutes *cornification* in the superficial layers of the epidermis. The horny cells are, as might be expected, situated always toward the centre of the cancer bodies in which they occur. They become fixed together, and the pressure to which they are subjected moulds them into globular bodies, more or less laminated in structure, but often having a transparent centre, which looks like a single large cell, although, no doubt, this is not really its nature. These bodies were called, by Lebert, "epidermic globes." In this country they are usually, but not very suitably, designated "birds'-nest cells." It must be clearly understood that in themselves they afford no proof whatever of the presence of a malignant new growth. On the contrary, they may be found under various circumstances, as, for example (according to Thiersch), "in lupus, in the hyperæmic borders of cicatrices, in sebaceous tumors, and in the epithelial covering of the gums." Their real importance lies in the fact that when they are seen in a new growth they show it to be not merely a carcinoma, but one which has its origin either in the skin or in some mucous membrane having a laminated epithelium. This form of cancer is exceedingly

frequent about the orifices of the body, especially the lips, the anus and the vulva. The mucous membranes upon which it may occur are those of the mouth, tongue, pharynx, larynx, and œsophagus, that of the lower part of the rectum, that of the urethra and of the bladder, that of the vagina, and of the os uteri. In many of these situations, however, it is common to meet with growths which, although the presence of horny globes compels one to call them keratoid carcinomata, yet contain the globes in small numbers, and which, in their general appearance and structure, approximate very closely to the "simple" form of cancer. Such transitional varieties are met with in the bladder, and they are very common in the cervix uteri. In the œsophagus they have appeared to me to increase in relative frequency from the pharynx downward, until at the cardiac orifice of the stomach a point is reached at which the liability to cornification within the interior of the cancer bodies ceases altogether. Another proof of the close relation between the two forms of cancer is sometimes afforded by an examination of the structure of the secondary nodules in distant parts, from a case in which the primary growth is keratoid. In some instances I have seen a nodule in the kidney or in the lung present horny, laminated globes, which were as well formed as those in the original seat of disease in the tongue or in the œsophagus. But in a recent case of keratoid cancer of the lip, in which the cervical glands contained typical globes, I found that some large secondary masses in the liver, which were softening centrally into cavities, had rather the structure of a simple carcinoma, the indications of cornification of any of the cells being very slight and imperfect. The keratoid form of cancer is, however, much more apt to infect the lymph glands which correspond with its primary seat, than to be carried to distant parts by the blood stream.

In the more characteristic cases of keratoid carcinoma, the material which can be scraped or squeezed from the cut surface of the growth is firm and granular-looking, so that it is very like the secretion of a sebaceous gland. It often comes out in the form of long, worm-like masses.

Histologically a keratoid carcinoma differs from the simple variety in some other respects, as well as in containing the "globes" or "birds' nests." Not infrequently some of the cells have prickle edges, exactly as in certain layers of the normal epidermis. Another peculiarity is that the "cancer bodies" are much more plainly seen to form continuous, bud-like, branching processes, which grow more or less vertically downward from the surface, penetrate the deeper structures and push them aside. Indeed, in its typical forms keratoid carcinoma may be said to present no definite alveoli. The substance that intervenes between the different cancer bodies was all of it pre-existent, and it had undergone little or no change, except that it is commonly infiltrated with leucocytes.

It seems now to have been clearly proved that the so-called "*Rodent ulcer*," which occurs chiefly on the face of persons advanced in life, and which is characterized clinically by its very slow course, is only a variety of keratoid carcinoma. And in all probability the same may also be said of the affection termed "*Cylindroma*" by Billroth, in which certain peculiar hyaline bodies are found. Each of these kinds of growth concerns the surgeon rather than the physician.

(*c*) *Columnar Carcinoma.*—In 1852, Bidder, of Dorpat, described in "*Muller's Archiv*," a case in which the cells of a soft cancer of the pylorus were identical with those of "columnar" or "cylinder epithelium." Many other instances of the same kind have since been recorded, and it has commonly been called a "cylinder" or "columnar epithelioma," a name which, of course, must be changed into that of columnar carcinoma, if we are hereafter to use the term epithelioma in the wide sense given to it in the

present chapter. In all probability the area of distribution of this form of tumor, as a primary growth, has now been completely mapped out. It includes the whole alimentary canal, from the cardia downward to within a short distance of the anus, the biliary passages, and the gall bladder, and (according to Cornil and Ranvier) the nasal fossæ, the upper part of the uterine cavity, and perhaps the ovaries. Of course, secondary nodules may occur in various parts. I have found them in the liver and the brain, and other observers in the lungs and in bones. The appearance of a primary columnar carcinoma is commonly that of an irregular shallow ulcer with a soft, raised, slightly projecting border; but I am not aware that it could be identified by its naked-eye character alone. It may yield an abundant juice, full of the columnar cells, which are often still adherent to one another by their sides. A thin section generally shows that they are very regularly arranged around the borders of long, branching channels or alveoli. But, as Cornil and Ranvier admit, some of them may be polymorphous. And I think that I have clearly seen transitions between this and other forms of cancer, not only in examining different primary growths, but also sometimes in comparing together primary and secondary growths from the same case, the tendency being for the latter to approximate toward the simple variety of carcinoma.

Histogenesis of Cancer.—The development of Carcinoma, generally, has of late years been investigated by many histologists with infinite pains, but the conclusion at which they have arrived is, after all, a matter of inference rather than of direct observation.

That leucocytes are unable of themselves to produce epithelium is almost established by what is known of the healing of wounds and ulcers from their margins, and by the success of Reverdin's practice of transplanting cuticle. One scarcely seems to need the corroboration afforded by the fact that tumors of epithelial type arise primarily only in those structures which contain epithelial elements. This fact, indeed, rests upon an experience which is not altogether unequivocal. German writers still cite a case of Virchow's, recorded in 1850, in which the tibia is said to have been affected by a primary "cancroid," the skin being healthy, and Otto Weber is quoted as having, in 1859, related a case in which there was a similar lesion of the lower jaw. One may, however, fairly hesitate as to the value of such observations made at a time when their theoretical significance could hardly have been appreciated. Far greater weight would seem to belong to the fact that Waldeyer and other recent investigators have failed to meet with a single instance of the same kind. It would, indeed, be easy to account for the occasional occurrence of exceptions to the rule. Apart from the difficulty of discovering in all cases the growth which is really primary, it might be argued that it is possible for a lesion starting from the skin to extend downward and to spread into the deeper structures, so as to appear to have begun in them. Paget, for instance, speaks of having "seen two examples of primary epithelial cancer in the lymphatic glands;" one was in a sweep, whose groin contained a large mass, notwithstanding that the penis and the scrotum appeared to be unaffected. But it may be doubted whether the microscope might not have thrown fresh light upon the matter. Thiersch records a case in which a nodule seemed for a time to be subcutaneous, but really had had its origin in the sweat glands. And I may take this opportunity of observing that some carcinomata seem definitely to arise from the cells belonging to the glands of the skin, or of a mucous membrane, rather than from those which cover the surface. Further, it is conceivable, as was suggested by Remak in 1854, that a fragment of germinal epithelium, embedded in other tissues from fœtal life, may sometimes afterward develop

into a cancerous tumor, instead of forming an innocent dermoid cyst. According to Thiersch, it is no uncommon thing for cancer to arise in the wall of such a cyst after it has existed for a number of years; and Waldeyer cites an instance in which a similar change occurred in a congenital sacral tumor.

Even recently, however, some pathologists have maintained that although epithelial cells are formed only in contact with pre-existing epithelium, they yet arise out of cells which are not themselves epithelial. Klebs speaks of an "epithelial infection," by which, for example, in a healing ulcer, the rete mucosum of the skin at its margin is supposed to convert the cells of the adjacent granulations into epithelial cells. But, as Waldeyer remarks, there are striking instances in which a cancer fails to impress its own characters, even upon epithelium with which it happens to come into relation. Thus, whereas the secreting cells of the liver are directly concerned in the development of some of the primary carcinomata of that organ, they are pushed aside by secondary nodules, the germs of which are derived from elsewhere, and take no part in their formation.

In 1867 Köster propounded the view that the cells of cancerous growths in the skin are formed from the endothelia of lymph channels. This, however, has met with no support; Waldeyer says that he has repeatedly seen cancer bodies lying in immediate contact with perfectly unaltered endothelium, and although he admits that they often penetrate into lymph channels and occupy them, he maintains that it is far from being always the case. On the whole, if we are not to cling to the notion that the epithelial cells are developed by germination or fission from pre-existing cells, it would seem to be more easy to accept the view that they arise out of ordinary leucocytes. For, as I have already had occasion to mention, it is a fact that the tissues in immediate contact with a primary cancer are very commonly thickly infiltrated with leucocytes; and, if they do not contribute to the new growth, their presence requires to be explained in some other way. It is not unlikely that they are the results of a reactive or inflammatory process, of which a more intense degree is seen as ulceration of the growth. Waldeyer points out that the cancer cells themselves are altogether passive when ulceration occurs; they break down and escape with the discharge. But there can be little doubt that the putrid and offensive character of the ichor which exudes from an open cancer is due to the extension inward of a septic agency along the epithelial columns and through the alveoli, which, as we have seen, communicate with one another.

Caseous Decay.—Carcinomata are liable to certain retrograde changes, beginning with their substance. Like sarcomata and almost every other kind of hard growth, they are apt to undergo "caseation." Whether this is altogether spontaneous—an indication that the cancer bodies have reached their natural term of life—or whether it is due to interruption of blood supply, has not, so far as I know, been ascertained. But it often affects all but the narrowest possible growing margin of a tumor, especially of a secondary nodule. And, not uncommonly, where a caseating tumor is seated in the interior of a solid organ, such as the liver, its centre softens down so as to produce a cavity filled with a straw-colored albuminous liquid.

Colloid Transformation.—But the most remarkable degeneration of cancers, and one which never occurs in sarcomata, is the "*colloid.*" Chemically there is, I believe, an exact analogy for it in the substance of the thyroid body when enlarged. Colloid material differs from mucus in not being precipitated or rendered opaque by acetic acid, and also in containing sulphur. Cancers which undergo this change sometimes occur in the breast, but scarcely anywhere else, except within the abdomen. At the

pylorus one can sometimes trace very clearly the gradual conversion of a simple carcinoma into colloid. Until the nature of the colloid degeneration was understood by pathologists, there was no greater puzzle. The abdomen was sometimes found filled with enormous gelatinous masses, in which the microscope showed no definite structure, but which yet penetrated organs and destroyed them in such a way as clearly to show that it possessed malignant characters. In 1847, Virchow's idea was that it was nothing else than a persistent condition of the gelatinous exudation out of which, at that time, he imagined all cancers to develop themselves. Long afterwards it was described as a separate and a very anomalous variety of carcinoma. But of late the doctrine that it is the result of a peculiar degenerative process has met with general acceptance. There can be no doubt, however, that much new material is laid down from the blood in a growth which is undergoing this change, for its alveoli have so enormously increased in size that they are often visible to the naked eye. Indeed, formerly, when the structure of carcinomata was not known, the terms "alveolar cancer" and "reticulated cancer" were often employed as synonymous with colloid. The epithelial cells, however, themselves become swollen out into glistening, structureless globes. And it is only toward the centre of some of the alveoli that a few unaltered cells may still remain visible. Colloid growths have a yellow tint, which serves to distinguish them from the myxomata which formerly were confounded with them. Immense masses of cancer, having undergone this degeneration, sometimes fill the abdominal cavity, lying in great part loose and unattached to the serous membrane. In such cases it may be only after careful search that one discovers any portion of the growth in an unaltered state.

Heredity.—Hitherto I have spoken of tumors as if they had only a local origin. But there is another side to their ætiology, which is commonly expressed in England by the statement that they are, or may be, *constitutional*. The proof of this lies mainly in the fact that malignant growths undergo transmission by inheritance. The experience of Sir James Paget is very striking; in his hospital days he found that the proportion of cases in which a family tendency could be traced was one in six; subsequently, in private practice, he made it one in four, and still more recently it has become one in three. It is true that these results might, perhaps, lose much of their apparent significance, if we were to inquire as to the proportion of healthy persons, chosen at random, who would be found to have had one or more relations affected with cancer, supposing their family history to be scrutinized with sufficient care. But some of the cases recorded by Paget and by others are far too striking to be set down as mere coincidences. Thus a lady, who died with cancer of the stomach, had had seven children and about thirty grandchildren who grew up. Some of them were still living; but a daughter had already died with cancer of the stomach, two granddaughters with cancer of the uterus, a granddaughter with cancer in the breast, a grandson with cancer in the bladder, a grandson with cancer in the rectum, a grandson with cancer in the axillary lymph glands. A point of considerable importance is that where the disease is transmitted by inheritance it often appears in the descendants at a much earlier age than in the first patient. Thus, a young lady of twenty-four died of "epithelial cancer" of the pharynx; her mother had been attacked by cancer when between forty and fifty years old, her grandfather between sixty and seventy, a great-aunt at about forty, her great-grandmother at eighty or older. A schoolfellow of my own died of cancer of the rectum before he was forty years of age; his father and his grandfather were believed to have also suffered from cancer, but at advanced periods of life.

In the *St. Bartholomew's Hospital Reports*, for 1866, Mr. Morrant Baker analyzed 103 of Paget's cases, and carefully investigated the question whether the appearance of cancer in successive members of the same family could be attributed to the mere propagation of a local defect, or (in other words) of a tendency on the part of some one organ to a special morbid change. But he found that it was hardly more common for the disease to occur in the same spot in two members of the same family in turn than for its seat to be quite different. A scarcely less important point is whether the inheritance is limited to a particular kind of new growth in each case. In regard to this Cohnheim observes that sometimes a mother suffers from adenoma of the breast, and her daughter afterwards from cancer of that organ. And among Paget's observations there are several in which it seems clear that carcinoma occurred in one member of a family and sarcoma in another. So, again, Sir William Jenner mentions the case of a man who had cancer of the tongue, and whose child, twenty-two years before, had died, at the age of two or three years, with disseminated malignant growths, which one may presume to have been sarcomata.

But, in truth, there are grounds for stretching to the widest possible extent our conception of the inheritance of a tendency to tumor formation. In the course of the discussion of the Pathological Society, in 1874, Mr. Hutchinson remarked that persons who had common warts in large numbers generally had had relatives affected with cancer. And I know that Dr. Goodhart shares with me the impression, based upon our observations in the deadhouse at Guy's Hospital, that it is very common to find all sorts of innocent tumors in the bodies of those who have died of malignant growths. On the other hand Paget has insisted on the frequency with which the liability to sebaceous cysts, or to certain multiple osseous tumors, is transmitted by inheritance.

Thus, it seems clear that the hereditary propagation of cancer is, after all, only part of a widely-spread diffusion of new growths of various kinds in certain families rather than in others. And the larger half of the ætiology has obviously still to be made out.

It seems to be certain that, to use the words of Sir James Paget, the growth of cancer often quickly follows "deep anxiety, deferred hope, or disappointment," in such a way as to suggest that mental conditions may play a part in its causation.

The Origin of Cancer.—According to Cohnheim, there is but one way in which it is conceivable that a new growth should arise in an adult, and that is by a portion of embryonic tissue having become arrested in its development during fœtal life, and having remained shut off until its dormant capacities have ultimately roused into activity. Such an hypothesis was, I believe, first propounded by Virchow, to account for the remarkable fact that enchondromata arise in connection with bones, but never from permanent cartilage; he supposed that a fragment or the original cartilaginous representation of the bone might remain unossified, and might ultimately form the starting-point of a tumor. Cohnheim maintains that cancers are particularly apt to arise at spots which (as in the case of the orifices of the great mucous channels) are the seat of somewhat complicated processes in the development of the embryo, so that involutions of the external germinal layer may be supposed likely to occur there. The ultimate formation of the new growth he attributes to a failure of "physiological resistance" on the part of the normal tissues around. A somewhat similar notion was put forth by Thiersch in explanation of the liability of aged persons to keratoid carcinoma; he attributed it to the atrophied and inelastic condition of the fibrous texture of the skin at

advanced periods of life, whereby he supposed the extension downward of the epidermis to be facilitated.

There can be no doubt that Cohnheim's theory is readily applicable to certain kinds of tumor. It is probably true, so far as the dermoid cysts are concerned; and it may perhaps account for the frequency with which growths arise in the uterus or in the breasts of unmarried women in the later years of life, since in such persons the organs in question no doubt contain germs which fail to receive their normal physiological stimulus. As regards the breast, indeed, Dr. Creighton has worked out a similar idea with extreme care and in great detail.* Having studied fully the normal process of evolution or "unfolding" which the mamma undergoes when preparing for its secretory functions, he finds that the growth of tumors in it may be regarded as a modification of that process, under what he terms "spurious stimulation," occurring at a time when the organ is in a quiescent condition. But it is obvious, though Cohnheim does not seem to think so, that such theories afford no explanation whatever of the mystery of tumor development. How a fragment of tissue, after lying dormant for years, can produce a carcinoma or a sarcoma, still remains entirely unexplained.

Moreover, there are some facts which seem to me to show conclusively that the theory of dormant embryonic masses is at least not universally true. I refer to the production of tumors by injuries, and to their appearing in spots which have for a long time previously been the seats of various local and accidental lesions. As regards the traumatic origin of cancers, Cohnheim, indeed, takes up a most uncompromising attitude. He cites Boll's statement that in twelve per cent. of the cancer cases operated on by Langenbeck there had been an injury before the growth was developed; but he declares that such a mode of causation is nothing less than an impossibility. He believes that his experiments on animals have enabled him to study all the congestive or inflammatory processes which are capable of resulting from injuries, and that such observations are final. But the following cases related by Paget seem to me to prove the contrary:—

A boy was accidentally wounded in one eye, which was sound. Within a few days a medullary tumor grew from the eyeball; it was removed three weeks later, but it quickly recurred, and destroyed life. Another boy fell and struck his knee; swelling followed, which was at first supposed to be inflammatory; but it increased, and proved to be a large medullary growth around the lower end of the femur. A steady man, at his work, slipped, and strained or broke his fibula; the injury led to pain and swelling, and in the course of eight weeks there was found to be a growth of large size outside and within the shaft of the bone.

Many instances can be cited in which a malignant tumor appears to arise out of some chronic local lesion. Thus it is well known that a keratoid cancer sometimes appears in the cicatrix of a burn on the arm or on the hand, where such a growth is otherwise rarely seen. A similar affection of the tongue often follows ichthyosis, or white syphilitic patches, at the end of several years. In the common bile duct, a carcinomatous growth is very frequently traceable to irritation of its mucous membrane by gall stones. And I have seen more than one instance in which a malignant growth in the posterior wall of the urinary bladder has seemed to have been caused by the repeated introduction of catheters.

I am not sure, however, whether most of these facts are so absolutely incompatible with Cohnheim's theory as he himself seems to suppose. One must admit that injury or irritation of a part gives rise to the formation of a new growth only in very exceptional cases; and it is surely not

* "Contributions to the Physiology and Pathology of the Breast." 8vo, London, 1878.

impossible that a dormant fragment of embryonic tissue, if its presence there may be assumed, should find in the injury, or the irritation, the stimulus needed to arouse it into activity.

The cases which it is most difficult to reconcile with such a view seem to be those of cancer in the scars of burns, and (on account of their frequency) those in which cancer of the tongue follows ichthyosis, or in which cancer of the bile ducts is set up by gall stones.

SYPHILIS.

HISTORY OF THE DISEASE—ORIGIN AND INCUBATION—PRIMARY LESION—INFECTING AND SOFT SORES—PREMONITORY STAGE—SECONDARY LESIONS OF SKIN, THROAT AND MOUTH, EYES, ETC.—TERTIARY LESIONS OF SKIN, TONGUE, PALATE, BONES AND TESTES—DIAGNOSIS—INFECTION—PATHOLOGY—PROGNOSIS—TREATMENT.

Considering how modern is the distinction between measles and small-pox—diseases unlike one another, and each remarkably definite in its character—one can hardly be surprised that neither the medical writings of antiquity, nor even those of the middle ages, contain descriptions of syphilis as an affection *sui generis*. What first drew attention to this disease was its epidemic prevalence in Italy at the end of the fifteenth century, when Charles VIII, of France, invaded and occupied Naples. The general supposition then was that it was a new malady; but some years later the suggestion was made that it had been brought from the West Indies by the sailors of Columbus, who arrived in Europe about the time when it broke out. However, there are grounds for the belief that the disease had been observed in France, in Germany, and in Italy, as well as in Spain, before the expedition of Charles VIII (1494-5), and when Columbus had not yet reached Palos (March, 1493), or at least had only recently landed his men there. And critical inquiries seem to show that in all probability syphilitic affections were by no means unknown during previous centuries, although they were confounded with other maladies, and particularly with leprosy.

The Italian epidemic of 1494 and the subsequent years was extraordinarily severe. Lancereaux says that almost a twentieth part of the population were attacked, and that although few died, fewer still were entirely cured. But the disease soon lost its pestilential character, and by the middle of the sixteenth century its type resembled that with which we are now familiar. A point worthy of notice is that its diffusion was at first ascribed to climatic influences, in ignorance of its real mode of propagation. Smaller epidemics of syphilis have since been observed, which have sometimes been entirely misunderstood, and described under a variety of names; and some affections long regarded as peculiar to certain regions have only recently been determined as modifications of syphilis. Under the former head I may mention a disease which attacked one hundred and eighty persons at Brünn, in Moravia, in 1578, one which raged in Canada in 1780, and the "Scherlîevo" of Fiume, at the beginning of this century; under the latter the "Sibbens," which prevailed in the west of Scotland during the eighteenth century, and the "Radesyge" of Sweden and Norway.

At the present time the disease is found in almost every part of the world, but with different degrees of frequency, according as the conditions are favorable or otherwise for its usual mode of propagation. But among the inhabitants of Iceland it is said never to have established itself, although it has repeatedly been introduced by sailors. And, according to Dr.

Livingstone, in the natives of pure blood in the centre of Southern Africa it does not persist in any form, but gets well spontaneously. The severity of syphilis in different countries is variable. In China and in Japan it seems to be generally mild, and Lancereaux cites French physicians, who had studied it in these countries, and have suggested that its virulence has gradually become attenuated by its wide diffusion through the population in successive generations. Upon the same authority it is stated to acquire a disproportionate degree of intensity when it passes from a Chinese to a European. Similar assertions have been made with regard to its transmission from one race to another elsewhere. But its worst forms always appear in seaport towns, where vice and intemperance prevail together; and it is also aggravated by unfavorable hygienic conditions of all kinds.

The term "Syphilis" seems to have been invented by Fracastorius, who, in 1521, published a poem in which he related how *Syphilus*, a shepherd, was stricken by Apollo with the new disease, which was even then not recognized as venereal. The French, from a very early period, contrasted *la petite* with *la grosse vérèole*, just as in England "smallpox," which we now regard as a single word, formerly conveyed a similar distinction. Many other names employed in various countries indicated a belief that it had been introduced by communication with foreigners. Thus, while it was "mal de Naples" to the French, it was to the Italians "mal francesce;" and, unhappily, the Sandwich Islanders originally knew it as the "English disease." At one time the most common designation for it was that of "*lues venerea*," which dates back to Fernelius (1556). But it is to be observed that the "venereal disease" was held to include both gonorrhœa, proved to be distinct by Ricord, in 1831, and the soft chancre, which many observers now regard as an independent affection. Indeed, the conception of syphilis as a general malady, comparable with the exanthemata, had no existence until about forty years ago. It is true that John Hunter described a constitutional form of the venereal disease, but he expressly taught that the action of the poison on the blood was different from that which occurred in any kind of fever. Since the modern view with regard to it has gained general acceptance, the name of Syphilis has superseded all others. That this view should be firmly held is, indeed, of the first importance. One may almost say that the physician ought, as far as possible, to thrust out of his sight the idea that the disease carries with it the stigma of impurity derived from illicit intercourse. There are many ways in which a person may fall a victim to syphilis without any breach of morality. And in exceptional instances one must be prepared to recognize its manifold varieties in patients of either sex, at any age, and in every position of life.

Course of the Disease—Incubation.—When a person is infected with the syphilitic virus there elapses, in uncomplicated cases, a considerable interval of time before any change is observed. The disease is like the acute contagious fevers, in having a period of incubation. It is very remarkable that this fact was unknown until, between 1856 and 1862, certain experimenters inoculated syphilis upon healthy individuals. Prof. Bäumler, in Ziemssen's "*Handbuch*," brings together thirty-one observations of this kind, in the very large majority of which the incubation was from fifteen to twenty-five days. Once it was only ten days: four times between thirty-five and forty-four days. In 1865 Fournier recorded a series of cases in which the affection had developed itself naturally. The incubation was more often over than under three weeks; it not infrequently reached a month or six weeks, and once was prolonged to ten weeks. In one patient of Bäumler's, in whom the exact date of exposure to the poison was known,

the incubation was twenty-five days; in another, twenty-nine days. The idea that no such period occurred was based partly upon a natural tendency to ascribe the disease to the last impure intercourse preceding its appearance, partly upon the circumstance that another virus, which produces an effect almost at once, is often transmitted in association with that of syphilis proper.

Primary Stage.—The earliest symptom of syphilis is manifested at the seat of infection; it is spoken of as the *primary* lesion, while the more remote symptoms, which appear elsewhere, are termed *secondary*. It is generally called a "chancre;" but upon the skin its typical character is that of a flat, red papule. This, which is at first very small, soon increases in size, and as it grows larger becomes indurated, so that to the touch it feels like a little piece of cartilage let into the part. After a week or ten days it may desquamate slightly; or a little moisture may ooze from its surface, and presently dry up into a thin scab; or it may continue to look shining and glazed; or, lastly, it may become excoriated, and slightly depressed in its centre. Upon a mucous membrane, the primary affection seems to begin as a very small, itching vesicle with a reddened base, which soon breaks, forming an erosion, and afterward a shallow ulcer; this, too, acquires an indurated floor as it enlarges.

The histology of these lesions has been investigated by several observers, but especially by Biesiadecki. A very abundant infiltration of nucleated cells is found, not only between the connective-tissue bundles of the skin or mucous membrane, but also in the adventitia of the blood vessels, which are narrowed by it. Biesiadecki states that there is an actual development of connective-tissue fibres toward the periphery of the indurated mass, and that this is the cause of its hardness, which by Auspitz had been attributed to the presence of a peculiar amorphous material packed in the interstices between the cells and the spaces in which they lie.

The course of the primary affection of syphilis varies in different cases. Sometimes it quickly passes away, leaving no trace of its presence, but when it is of large size it generally takes many months in subsiding, and upon the skin its site remains marked by a brown pigmented patch, with more or less superficial scarring in its centre. On the other hand, mucous membranes never show pigmentation, and all that is left by even the largest papule is a little vascularity and looseness of texture. Mr. Hutchinson has drawn attention to the fact that in some individuals an induration, like cartilage, appears again and again during a period of several years, exactly where a former syphilitic lesion was situated, without any fresh infection. Ultimately even the pigment disappears, and it may then be quite impossible to discover that the patient had ever suffered from primary syphilis.

Hitherto I have avoided speaking of the typical primary syphilitic lesion as a chancre, for I wish to bring prominently forward the view that if (as is often the case) it has the characters of a chancre, they are accidentally present rather than essential to it. At least this seems to me to be the simplest way of stating the results of investigations which have, within the last few years, been made with regard to the mutual relations of certain different kinds of venereal sores. And Bäumler actually calls the affection which I have been describing, not a chancre, but an "ulcerating sclerosis."

In 1852 Bassereau took the pains to trace to their origin in the opposite sex a number of venereal cases; and this method of "confrontation" (as he termed it) showed that, whereas sores which were followed by secondary symptoms had been derived from persons who themselves suffered under similar effects, other sores, which remained simply local, or which at most were attended with suppurating buboes, came from individuals in whom the disease had likewise failed to produce any constitutional effects. His views were soon afterward adopted by Ricord, who indicated a number of distinc-

tions between the two kinds of lesions in question, of which one became known as the "indurated," "Hunterian," or "infecting" chancre, and the other as the "soft" or "non-infecting" chancre. His account of the infecting chancre need not be quoted in this place, except that I must mention the important fact, which was now brought out for the first time, that the ordinary slight secretion of such a sore cannot be inoculated upon the bearer of it, nor upon any other individual who has already had syphilis. Bidenkap has, indeed, since found that there are occasional exceptions, it being sometimes "auto-inoculable" during the early stage of the disease, when there have as yet been no constitutional symptoms. But such observations evidently do not affect the principle on which the rule is explained, since they are strictly parallel with the well known circumstance that vaccination can be successfully performed at the commencement of smallpox before the general eruption appears. Ricord's description of the non-infecting chancre was that it begins (without any incubation) in a pustule, which, in two or three days, breaks; that there is then formed a deep, punched-out ulcer, with irregular and slightly undermined edges, a gray surface, and a soft, or at least not an indurated base; that it secretes pus freely, and that this is inoculable again and again upon the same individual, and also upon all other persons, whether affected with syphilis or not. One consequence of the difference in the inoculability of the two affections is that whereas a patient seldom presents more than one indurated chancre, several soft chancres are often seen side by side. Those who have recently investigated the histology of the latter affection have found that it is not so different as might have been expected from that of the primary lesion of syphilis. There is a similar infiltration of cells into all the tissues, including the walls of the blood vessels, but it is said that the channels of these vessels are dilated, instead of being narrowed. The cicatrices left by non-infecting chancres are often hollowed out and very irregular in form, so that they are much more conspicuous than those which finally result from typical Hunterian sores.

There does not appear to be any doubt as to the correctness of Bassereau's observations, nor as to the general validity of Ricord's distinctions between the two kinds of venereal sore. But subsequent experience has shown that the practical application of those distinctions in the prognosis of such affections is liable to certain sources of error. And the theoretical question still remains under discussion, whether the poison of the soft chancre is or is not, in its origin, independent of syphilis. It is hardly necessary now to allude to the confusion which at first prevailed in consequence of the way in which Ricord and those who followed him formulated their doctrines. Instead of speaking of the "duality" or "unity" of the chancre, they insisted on the *duality of the syphilitic virus*. The inevitable result was that they drove from their camp all those who regarded syphilis as a single specific disease, comparable with the contagious fevers. And yet for such persons there was no place among Ricord's opponents, who advocated the *unity* of the local soft sore with that which is followed by constitutional symptoms. This, however, is scarcely more than a verbal difficulty.

The chief considerations bearing upon the theoretical question as to the origin of the contagious principle of the soft chancre are the following: In 1854 Clerc met with a case in which an indurated chancre on the scrotum, followed by secondary symptoms, had inoculated itself upon the skin of the penis with which it came into contact, producing there a sore which remained free from induration. Another patient under his observation had an indurated chancre, the gray surface of which suggested that its secretion would be inoculable; the experiment was tried on the patient himself, and the result was a soft sore which reached the size of a franc piece. It is evident that these facts are easily explicable on a theory of Rollet's, to which I shall

presently refer, namely, that "mixed sores," containing both the virus of syphilis and the contagion of the soft chancre, are not uncommon. But the view which Clerc adopted was that the soft chancre is nothing else than the product of inoculation from an infecting chancre upon a person who is suffering or has suffered from constitutional syphilis. And, as if there were not already sufficient confusion, he proposed to limit the use of the term chancre to the indurated primary lesion of syphilis, and to call the soft sore a "chancroid." Shortly afterward Mr. Henry Lee showed that an indurated sore, when irritated by savine ointment or powdered savine, could be made to pour out a purulent fluid which, when inoculated upon the bearer, or upon other patients, produced, without any incubation, a sore having the characters of a soft sore and capable of being inoculated again and again. And, lastly, Pick, of Prague, found that pus from a case of scabies, or pemphigus, or acne, was also capable, when inoculated upon syphilitic subjects, of generating a similar sore, although it failed to affect healthy individuals in the same way. From these experiments Clerc's hypothesis, of course, derives far more support than from his own observations, because the alternative theory of Rollet can be altogether set aside. And if it is really the case that the sores obtained by Lee and Pick are identical with soft chancres, I think that one may be justified in concluding that such chancres are a kind of by-product of syphilis, and that their contagious principle is generated *de novo* in persons suffering under that disease. As I shall presently point out, there is reason to believe that in flat condylomata, themselves due to syphilis, there often arises a virus which is capable of transmitting them, independently of all other symptoms of syphilis, to healthy individuals. And such facts are fairly comparable with those which seem to show that in various morbid states of the human body animal poisons may be developed which may set up erysipalas, or even perhaps diphtheria, in other patients. But I cannot agree with Mr. Hutchinson in speaking of soft chancres as "abortive inoculations," since what creates all the difficulty in explaining their origin is that their contagion possesses a virulence of a certain kind which is wanting in the secretion of a true infecting chancre. That it is not a mere question of the presence or absence of pus is shown by Ricord's observation that the matter from the interior of a suppurating gland produces a soft sore when inoculated, whereas this is not the case with the matter which is formed in the connective tissue outside it.

From a practical point of view, however, the origin of the soft chancre is of minor importance in comparison with the question whether it contains the virus of syphilis, that is, whether it remains local and can only propagate itself, or whether it is liable to be followed by constitutional symptoms. In principle I believe that the first of these statements is the correct one, but unfortunately it is open to exceptions, which interfere with the application of it in the prognosis of individual cases. I have already alluded to the theory of "mixed chancres," promulgated by Rollet, of Lyons, in 1858. Now, if a person is infected at the same time with the poisons of the soft chancre and of syphilis the necessary result must be the immediate production of what appears to be nothing but a soft chancre, which will not assume an indurated character until the lapse of three or four weeks, corresponding with the incubation of syphilis. Such sores, I believe, constitute an actual majority of the primary syphilitic lesions, themselves derived from chancres, which are seen in the hospitals of large cities. It is, however, a question whether the rapid spreading of the base and sides of a soft sore, which sometimes occurs, may not lead to the destruction of any syphilitic poison that may be present in it, and thus save the patient from further ill-effects. In this way it might be possible to account for an occasional failure in the transmission of syphilis from a person

suffering under that disease to one who has never had it, notwithstanding the occurrence of a chancre in the latter individual. Bäumler states that there are only seven experiments on record in which the artificial inoculation of unquestionably syphilitic secretions upon healthy persons has not communicated the constitutional affection. On the other hand, it not unfrequently happens that a male patient derives syphilis from a woman in whom a soft sore is the only discoverable primary lesion; and the best observers have been obliged to allow that they have seen instances in which a sore that has at any one period been indurated has been followed in the same individual by secondary symptoms. Mr. Morgan, of Dublin, has especially insisted on the fact that in the Lock Hospital of that city, he scarcely ever saw indurated chancres in the very class of women from whom the men who come to the institution derived their venereal diseases. But this proves less than might at first sight appear, now that we know that the secretions of moist secondary eruptions contain the specific virus. And Mr. Cooper Forster has stated that at Guy's Hospital he generally succeeded in finding a characteristic primary affection in female patients when he carefully looked for it. It is usual to explain away all difficulties in the matter by saying that the induration is sometimes very slight and parchment-like (*parcheminie*), so that it can be detected only by taking the sore between the finger and thumb in a particular manner, which is impracticable in the case of a female with a sore situated within the vaginal orifice. But this is in reality equivalent to the admission that the primary lesion of syphilis does not invariably present the distinctive characters which are assigned to it. Conversely, the typical induration is sometimes closely simulated by inflammatory thickening of the floor of a soft chancre, as the result of irritation by caustics.

Thus it appears that, in practice, although it is easy to say that certain sores are almost sure to be followed by secondary symptoms, it is seldom, if ever, possible to assert that any sore will *not* be so followed. It must, consequently, be unadvisable to attempt to limit the use of the term chancre to sores from which the syphilitic virus is absent, as has been proposed by some writers, including Mr. Forster. We had much better keep to its original meaning, and apply it to all *ulcers*, whether syphilitic or not, which are directly due to venereal infection. But I think that when an indurated papule constitutes the primary lesion of syphilis it should not be called a chancre.

Constitutional symptoms of syphilis are sometimes seen in persons in whom no primary affection is known to have occurred. One source of fallacy in these cases is that a primary lesion sometimes assumes the appearance of a secondary one. Thus, a parchment-like glazed papule on the glans penis may be undistinguishable from the elements of a general eruption among which it lies, while on the labium the affection may be so modified as to simulate a "broad condyloma" or "mucous tubercle." In other instances, perhaps, it is so inconspicuous that the patient never notices its presence, and it quickly subsides, leaving no mark. But some observers admit a true "*syphilis d' emblée.*"

Premonitory Stage.—Early syphilis is further characterized by *swelling and induration of the lymphatic glands* which bear an anatomical relation to the seat of the primary chancre or papule. So constant is this symptom that Fournier failed to detect it in only three out of 265 cases in males, and in the same number among 223 cases in females. The glands which correspond with the genital organs are, of course, those in the groins. An indurated sore upon the finger leads to enlargement of glands in the axilla, or at the bend of the elbow, or in both places. One upon the lip affects

those near the lower jaw. They reach the size of filberts, or may even be a little larger, but they never approach the dimensions of the suppurating bubo which accompanies a soft chancre. They feel hard, are freely movable, and are seldom tender or painful. There is generally no reddening of the skin over them. They scarcely ever suppurate. Sometimes along the dorsum of the penis an inflamed lymphatic vessel can also be felt, like a cord.

The "indolent bubo" of syphilis, as it is termed, appears a few days later than the primary lesion. It commonly includes a chain of glands, and may be present in both groins at the same time. It runs an exceedingly slow course, so that it often does not begin to subside until six months have elapsed. Thus it may be very useful in diagnosis, by indicating the probable seat of a chancre or papule, the marks of which might otherwise have been overlooked. One must, however, remember that syphilis is not the only possible cause of a similar affection of the inguinal glands. Mr. Cooper Forster has insisted on the frequency of such an occurrence in those who habitually irritate the skin of the buttocks in rowing. I think I have also seen it in young men accustomed to take much walking exercise. Whether glands in other regions are liable to become enlarged apart from syphilis I do not know.

The appearance of the primary papule, or chancre of syphilis, is followed by an interval during which we may suppose that the virus is undergoing multiplication in it and in the adjacent glands. And as the lesion itself now and then subsides in the meantime, the period is sometimes spoken of as the "second incubation." Its length is generally six or seven weeks; but it is liable to wide variations, as is shown by the fact that where the disease has been inoculated experimentally it has been found to range from twelve days to twenty weeks. Probably it is shorter in persons who are weakly, or whose habits are dissolute or intemperate, than in those whose general health is good. During its course some patients look and feel well. But others—it is said, particularly women—complain of malaise and depression, and acquire a pale and haggard aspect.

Secondary Stage.—The *constitutional* symptoms are variously ushered in in different cases. In some there is well-marked fever, the temperature rising suddenly, or gradually, until within a few days it may reach 104°. With the appearance of an eruption it sometimes declines; but it may run on for several weeks, assuming, as Bäumler has shown, an intermittent type resembling that due to malaria. The proportion of cases attended with febrile disturbance is stated by Güntz, who has specially studied the question, at 20 per cent.; but Bäumler thinks that it is really higher.

Another and a very characteristic symptom of this period is what French writers term a "bitemporal neuralgia;" the occurrence of more or less violent pains, which shoot upward along each tenple. They are not felt at all periods of the day, but come on, often with surprising regularity, in the evening or at night. A similar tendency to nocturnal exacerbations, indeed, belongs to all other painful syphilitic affections. Ricord used to ascribe it to the warmth of the bed, and said that in persons whose occupations compelled them to sleep during the day, the time at which the pains returned was reversed. But whether or not this is the fact, there can be no doubt that his explanation of it is incorrect. For the hour at which the bitemporal neuralgia sets in is often early in the evening, while the patient is still up. And Bäumler is probably right in thinking that it coincides with an increase of fever, and is in some way dependent upon augmented vascular excitement. Pains in the back and limbs may be present. There may even be swelling of joints. I have seen a distinct, thought slight and painless, enlargement

of some of the articulations of the fingers; and Bäumler speaks of patients seeking advice for an affection of the metacarpo-phalangeal joint of the thumb, but leaving it to the physician to discover that they have syphilis.

Syphilis of the Skin.—Unlike all other specific poisons, the virus of syphilis produces not merely one or two definite cutaneous affections, but an immense variety of them. They are known as "syphilides" or "syphilodermata" (more properly syphilodermiæ); but in consequence of the way in which they shade off into one another they almost defy description, and are classified with the greatest difficulty. With regard to the causes which lead to the evolution of one of these eruptions, rather than another, all that we at present know is that a patient who is in a bad state of health is most likely to have those forms which suppurate and ulcerate. It is by no means the fact that the differences between them are directly attributable to constitutional tendencies towards particular non-syphilitic cutaneous diseases; as, for instance, that a person liable to ordinary psoriasis is specially apt to be affected with a squamous syphilide, or one who has lichen with a papular one. It is, therefore, better to avoid using such names as "syphilitic lichen," "syphilitic roseola," or "syphilitic psoriasis." Indeed, however difficult the diagnosis may be in individual cases, syphilitic affections in the aggregate present characters which show that they are really distinct from the non-syphilitic eruptions which may simulate them.

There are certain features which belong, more or less, to all the syphilides. One of them is a peculiar color, which is commonly said to resemble raw ham, or to be "coppery;" according to Bäumler, the former comparison has been traced to Fallopius, who wrote about three hundred years ago; the latter only to Swediaur, at the commencement of the present century. The cause of the tint is probably a chemical change in disintegrating blood discs which have been extravasated into the tissues, and therefore it is not present at first; while, on the other hand, a very similar appearance is sometimes displayed by non-specific eruptions, of chronic course, especially in the legs, where the venous circulation is apt to be embarrassed. A second character of syphilitic cutaneous affections is their multiplicity or "polymorphism" in the same patient, and at the same time. Sometimes macules, papules, pustules, scaly patches are so intermingled that we cannot say which of them preponderates. Or there may be little more than crusts and indefinite patches, to which no positive character can be assigned. In my opinion, it is only in such cases that one can be justified in setting down an eruption as syphilitic, for no better reason than the fact that it does not correspond with any known skin disease; but I believe that this method of diagnosis was formerly often adopted, even when the characters of the affection were obviously peculiar. A third point, which is somewhat special to the syphilides, is their tendency to arrange themselves in circles or semicircles, or less completely annular forms. Lastly, unless their development is very rapid, they are seldom attended with itching; in many cases, the patient experiences nothing whatever to draw his attention to the skin.

However, I think that in most cases a correct diagnosis must be based, not so much upon the recognition of characters common to all the syphilides, as upon an accurate acquaintance with each of them individually. It is, therefore, absolutely necessary that we should in some way classify them. Now, there is one main division which but seldom fails to be applicable, namely, between those which are of *early* and those which are of *late* occurrence. The former, to which some writers limit the name of *secondary* affections, may relapse several times but the first appearance of any one of them is very

rarely postponed beyond twelve months from the time of infection. The latter, which are often called *tertiary*, seldom appear within the first year, and they may break out for the first time after an interval of many years. As the fundamental difference between them, Mr. Hutchinson insists upon the bilateral symmetry of the early syphilides, which is absent in the case of the late ones. It is, in fact, impossible to strip a patient suffering under one of the common secondary eruptions, without seeing the close correspondence of its pattern on the two sides of the body, and on the opposite limbs. Other features are the large number of the spots or patches, their isolation from one another, their comparatively superficial seat in the skin, their little tendency to ulcerate, and the fact that, as a rule, they leave no cicatrices behind them. On the other hand, the so-called tertiary eruptions, besides being non-symmetrical, consist of elements which are comparatively few in number, but which generally run together; they affect the deeper layers of the integument; they destroy the tissues, and are followed by scars. These distinctions are not absolute, nor do they apply equally to every form of syphilide belonging to the early and the late group respectively; but the exceptions to them are few.

The chief early syphilides are the following:—

1. *The Macular or Exanthematic Syphilide* ("syphilitic roseola"). It consists of rather ill-defined, pale or dark, rose-colored spots; irregular in form; of small size, or approaching that of a threepenny-piece, or even larger; scarcely if at all raised above the surface; generally disappearing completely under pressure. Brisiadecki and Kaposi, have each investigated their histology, and have found the capillaries in them surrounded by cells, and an excess of nuclei in their walls. The external coat of the larger vessels also showed round and spindle-shaped cells. Kaposi further detected wandering cells in the substance of the papillary layer of the cutis.

This form of syphilide is most constantly seen upon the sides of the chest and abdomen, but sometimes it covers the whole of the trunk, and appears on the neck and face; on the limbs, it affects the flexor rather than the extensor surfaces. As a rule, it takes a week or more in coming fully out, but sometimes it is developed so rapidly that the case may be mistaken for one of measles. Or an error in the opposite direction may be committed, and a rash regarded as syphilitic, which is really due to copaiba taken medicinally. On the other hand, where there are but few spots, the affection most likely to be mistaken for an exanthematic syphilide is Tinea versicolor. Such a pale and scanty eruption often fades within a fortnight. One which is dark colored and abundant, may remain visible for several weeks. It may then, after a time, assume a squamous or papular character; otherwise, it sooner or later disappears, even when no treatment has been adopted. Probably this syphilide never comes out for the first time at an interval of more than a few months after infection, but relapses of it may occur at any time during the first, or even (according to Kaposi) during the second year. The maculæ, however, are described as being then larger and darker, and often annular in form.

2. *The Follicular Syphilide.* Under this name, I group together two eruptions, which have generally (except by Bäumler) been separated from each other, the first being placed with the papular syphilide, under the name of "miliary syphilitic lichen," the second with the pustular syphilides as "syphilitic acne." The present arrangement, however, is much more natural, since the affections in question bear no close relation towards the other forms, whereas they are intimately allied to one another.

One variety consists of small, pointed, dry elevations or papules, each corresponding with the mouth of a hair sac. They are generally more or less scaly at the summit, and their bases may be surrounded by a minute white

collar of desquamating cuticle. They may either be scattered irregularly or collected into groups or clusters. Histologically they appear to be characterized by an exceedingly superficial infiltration of cells into the papillæ close to the orifice of the hair sac, and by an extrusion of this infiltration downward along its walls. This eruption is exceedingly slow in its course, developing itself by successive crops at considerable intervals of time, and subsiding only after several weeks or even months. Sometimes the papules pass into vesicles, or become pustular; but even when this is not the case they always leave minute white cicatrices.

The other variety consists, from the first, of pustules, which, like the papules, are small and pointed, and which have swollen, reddish-brown bases, and are each traversed by a hair. They may be present in immense numbers, not only upon the face, but also upon the chest, and back and limbs. They dry up into little yellowish-brown crusts.

It is worthy of notice that the fine punctured scars which (contrary to the rule as regards early syphilides in general) result from these follicular affections are often exceedingly useful in the diagnosis of the disease at a later period.

3. *The Papular Syphilide* ("lenticular syphilitic lichen"). This consists of red, shining elevations, firm and solid to the touch, often presenting a marked copper-like tinge. In size they vary considerably, some being scarcely bigger than millet seeds, others as large as peas or even beans. Thus the larger and more typical of them can scarcely be termed papules, according to the usual practice of dermatologists, being what would rather be called "tubercles" or "nodules." They develop themselves very rapidly, reaching their full magnitude in a few days. They do not, however, generally all come out at once, but rather in successive crops over a period of some weeks or even months. Histologically, each of them consists of an infiltration of cells into the superficial layers of the cutis, so dense, that the boundary line between the papillæ and the rete mucosum is almost, if not quite, obliterated. The cells themselves are of unequal size, and are supported by a stroma with fine meshes. Towards the sides and the base of the papule the infiltration is sharply limited, the deeper strata of the cutis containing no cells, and not being even œdematous. As Kaposi remarks, this strongly suggests that the pathology of the affection differs from that of a mere inflammation.

The eruption is sometimes scattered irregularly over the whole body, sometimes the papules are grouped together in clusters. They may be more numerous on the neck and on the forehead than elsewhere; in certain cases they are thickly crowded in the naso-labial grooves, at the angles of the mouth, and about the genital organs. They generally remain for some weeks and then subside, leaving dark stains, which in their turn slowly disappear. But not unfrequently they desquamate, in which case the papules may be made to pass into what will be described as the squamous syphilide, and in other instances their summits soften down, and become covered with yellow or brown crusts.

In consequence of the thickness of the cuticle of the palms and soles, these parts show, not raised papules, but flat, round, horny plates, each with a reddish-brown border. And after a time the plates may become detached, forming so many little ulcers, or the adjacent surface may become rough and scaly, and cracks or fissures may be formed. Thus a very complicated affection arises, constituting the chief form of what has long been known as "syphilitic palmar and plantar psoriasis." At this stage of the disease it is generally bilateral.

Another modification of the papular syphilide is seen on parts of the skin which are delicate in structure, and which habitually touch other

parts; and also upon the mucous membranes of the various orifices. In either case the lesion which results is known as a "mucous tubercle" or "*plaque muqueuse*," or as a "flat condyloma," the epithet being needed by way of distinction from the "pointed condyloma," or "wart" of the genital organs, which is non-syphilitic in origin. This affection consists of one or more broad, raised patches, each from the size of a lentil to a half-crown, with a sharply-defined edge, and a surface which is sometimes dry and warty-looking, but much more often moist and coated with a dirty gray secretion of a peculiarly nauseous, pungent odor. Histologically they are characterized not only by the usual cell infiltration of the cutis, but also by a great overgrowth of the papules and by a branching out of their summits. Flat condylomata are sometimes present in large numbers, not only about the genitalia, but also along the perineum and around the anus, in the groins, at the umbilicus, in the folds of the axillæ, beneath the breasts, in the neck, between the toes, at the angles of the mouth, and elsewhere. They may become confluent, so as to affect an extensive surface almost uniformly. Where this is the case it is sometimes possible to mistake the affection for an infiltrating eczema. Not infrequently they are so placed upon opposed parts of the skin or of a mucous membrane as strongly to suggest the idea that they spread by a local infection. But, if this is the fact, it is one of great theoretical interest, since a patient who already has syphilis is believed to be absolutely protected from the further influence of the virus, so that we are led to the idea that flat condylomata must possess an independent contagion peculiar to themselves. We shall hereafter see that they are often the means by which syphilis is transmitted to persons previously healthy. In such cases they generally give rise to a typical, primary, indurated papule, which is followed, after the usual interval, by constitutional symptoms. But, on the other hand, it is certain that in some cases the affection which they set up in non-syphilitic individuals is itself undistinguishable from a flat condyloma, and cannot be proved, either at the time or afterward, to contain the syphilitic poison. As much as this is admitted by Kaposi, but he passes it lightly by with the remark that secondary symptoms sometimes fail to appear even after an indurated primary sore. But so common is the occurrence of flat condylomata, apart from all other indications of syphilis, that some observers (including several of my surgical colleagues) have actually attributed them to mere irritation of the surface by dirt and moist secretions. Moreover, in some countries they have been known to prevail endemically in such a way that their connection with syphilis has been altogether a matter of inference. These considerations seem to me to point strongly to the conclusion that, although the affection owes its origin to syphilis, it yet acquires a contagious principle of its own, which is capable of surviving in full activity after the original syphilitic virus has in some way disappeared or become exhausted. As I have already remarked, this tends strongly to corroborate the doctrine that the soft chancre arises and spreads in a similar manner.

4. *The Pustular Syphilide* ("syphilitic ecthyma and rupia"). This is rather a collection of several different varieties of eruption, than a single, well characterized form. It consists of pustules of all sizes, each of which is generally seated upon a firm, red base. They are sometimes present in immense numbers, especially upon the face and the trunk. They come out rapidly and with much febrile disturbance, but in successive crops, which may be prolonged over many weeks; and they may relapse even after a year has passed. They dry up into brown, or dark green, or black scabs, and leave large stains, which ultimately pass into shallow, flat, white cicatrices. But in many cases their inflamed bases continue to spread long after their summits have

thus scabbed over. Under such circumstances, as the crusts increase in size and in thickness, the older parts of them are continually being pushed upward by the aggregation of fresh material beneath; the necessary consequence is that they assume a conical shape, and, in fact, look very like limpet shells. Some of them may thus acquire an enormous size. This particular variety is commonly called rupia; there is, I believe, no similar affection of non-syphilitic origin. It is to be observed that it resembles the late syphilides, not only in being attended with more or less deep ulceration of the cutis, but also in being unsymmetrical. Indeed, it has often been described among the tertiary eruptions.

5. *The Squamous Syphilide* ("syphilitic lepra or psoriasis"). Contrary to what used once to be taught, this, as an independent eruption, is infrequent. We have seen, however, that the macules and papules of syphilis often, after a time, become scaly. Now, if such spots go on spreading still further, there arises an affection which may closely resemble psoriasis. The features which have generally been relied on as distinguishing it are the dirty gray appearance of the scales, their rather scanty development and small size, the copper-like tint of the patches, and the absence of a definite localization upon the points of the elbows or knees, or upon the extensor surfaces of the limbs generally.

A particular variety which has been called *the early circinated syphilide* is characterized by thread-like, scaly rings, from the size of a split pea to that of a shilling; they may be formed in very large numbers, especially on the face.

6. *The Vesicular Syphilide.* That syphilis very rarely produces an eruption of vesicles is well known. Mr. Hutchinson, however, speaks of a form which is attended with clusters, like those of shingles, but which is bilateral and widely distributed over the body. And Hardy gives three varieties, which he terms respectively cyanotic, varioliform and herpetiform. I am not myself acquainted with any of them.

7. *The Pigmentary Syphilide.* This also is a very rare affection, which was first described by Hardy. In the museum of Guy's Hospital we have models and drawings taken from a case which was under Dr. Barlow's care in 1856. The patient, a woman, had brownish-black maculæ scattered over the chest, shoulders, and arms. The commencement of the syphilis dated from eight months previously. Under treatment the stains faded to a considerable extent. Hardy speaks of this syphilide as being almost peculiar to the female sex, as occurring principally on the neck and front of the chest, and as consisting of irregular spots, of a *café au lait* color, of the size of half-franc or franc pieces, situated close to one another, or running together.

Concomitant Early Affections.—One of the points in which syphilis resembles the acute exanthemata is its strong tendency to affect the *fauces*, apart from all other mucous surfaces, in conjunction with the skin. Indeed, the throat sometimes suffers before any cutaneous eruption can be discovered; and in other instances an inspection of it reveals morbid changes, which had caused neither pain nor discomfort, so that the patient had no suspicion of their presence. The acute angina of early syphilis appears as a diffused redness, and not in the form of distinct maculæ, contrasting in this respect with the throat affection of measles. There is often a good deal of swelling, particularly of the tonsils. The follicles become enlarged and prominent; or they may rupture, giving rise to shallow excoriations. In more chronic cases flat condylomata, assuming the appearance of gray patches or ridges, are present on the tonsils or the palatine arches; or they may be yellow ulcers, with sharply-defined red borders. But the most curious affection of

all is one which is seen, not only upon the fauces, but also upon the hard palate, the inside of the cheeks, and the lips. It consists of scattered, milk-white spots, which have been termed *plaques opalines*, and which are perhaps best compared with the effects of the application of nitrate of silver, upon a mucous membrane. Their shape may be round, oval, or indefinite; they vary in size, and may run together so as to cover a large surface with an irregular pattern. Sometimes they are slightly puckered, and parts of their surface may be reddened, with only a little white opacity here and there. All their varieties are deserving of careful study, for they are very characteristic of syphilis, although I can hardly agree with Kaposi in thinking that no similar affection ever occurs in those who have not had that disease. They run an exceedingly slow course, and may break out again and again, not only during the early stage, but also long afterward.

In many cases the *eyes* suffer. The affection most commonly observed is an iritis, attended with the formation of yellowish-red nodules near the edge of the pupil. This is generally bilateral. Mr. Hutchinson says that in his experience it arises within the first six months of the disease, if at all. It is generally associated with one of the more severe forms of eruption. Later attacks are said to be always relapses; they are often limited to one eye at a time. Or retinitis may occur, in which case the fundus of the eye has a hazy appearance, when seen with the ophthalmoscope; the disc is reddened and swollen, and its margin is indistinct; there may be many small extravasations of blood. The recognition of this marked change is very important, because of its insidiousness; Mr. Hutchinson speaks of the patient as noticing "nothing, except that his sight is very dim; he has no pain, no congestion of the front of the eye, no intolerance of light."

The *lymph glands* in various parts of the body become swollen at this stage of syphilis; we are especially accustomed to look for enlargement of those which lie one above each elbow, and of those that are seated at the back of the neck, close to the occipital bone. Whether this is dependent upon any previous affection of skin or mucous membrane is at present doubtful.

Another symptom is *falling off of the hair;* it may come away with the comb so freely that the patient is apprehensive of becoming prematurely bald. I have, however, never myself seen anything like a complete alopecia from this cause; and I am disposed to think that the cases which have been so interpreted have really been examples of Alopecia areata. The fine hairs from the limbs are shed, as well as those of the scalp.

As to early syphilitic lesions of the viscera, nothing positive is as yet known. Mr. Hutchinson has ably insisted on the strong probability that such affections may occur, and he mentions a transitory albuminuria as not uncommon. I myself had one patient whose urine was albuminous (and who, I think, had dropsy) at a time when it could hardly be supposed that the vessels had already become lardaceous. I have also occasionally seen jaundice, which in one case quickly subsided, but which in another instance ended in acute atrophy of the liver. To this subject, however, I shall return hereafter.

Later Symptoms.—After the lapse of the six months, or the year, or two years, during which one or more of the early syphilides has developed itself once or oftener, the disease often becomes altogether latent. In many cases, indeed, it finally ceases, and the patient henceforth remains perfectly free. But in other instances he continues to be troubled, at varying intervals, with slight manifestations of it. Perhaps small, scattered pustules appear on the scalp, which are scratched by the comb, and cause him infinite annoyance, scabbing over again and again. Perhaps the palm of one hand now and then becomes scaly and fissured in its centre. Perhaps some of the nails

grow rough, and thick and discolored. Affections such as these may go on for a very great length of time.

But in some cases, possibly many months or even years after the subsidence of the early syphilides, there appears an eruption presenting definite characters of its own, and yet differing from any that would have arisen during the secondary stage of the disease. I have already indicated what are the chief features common to such late or *tertiary* syphilides, namely, that they are less symmetrical, generally cohere together, affect the deeper layers of the integument, destroy the tissues, and are followed by scars. Their special characters vary widely in different cases.

In 1869 a woman was under my care in hospital who had been infected by her husband twenty years previously, and in whom the disease assumed the form of thin, reddish-brown, glazed patches, covering the greater part of the face; they were not at all raised, and there was only the slightest possible desquamation from them; in fact, they were scarcely more than maculæ. In other cases, thick, white scabs appear upon the circumference of a bold, red ring, or festooned line. Such an affection, perhaps, deserves to be specially designated as squamous. Or again, immense patches of skin may become thickened, raised, of a reddish-brown color, and rough, with a bran-like scurf; so that, if the disease had not been syphilitic, one would have named it a dry eczema. But, as a rule, what constitutes the essential feature of the late eruptions of syphilis is the presence of lurid, reddish-brown nodules; these are like those of the early papular syphilide, except that instead of being scattered, they now cohere in rings or patches, which themselves are often fused together, so as to cover a large surface or to form straggling, festooned lines. It is also, I think, a peculiarity of such late tubercles, that they invariably leave cicatrices, even when they have not ulcerated. But in most instances they either become covered over with crusts, or become eaten away, so as to form small, deep ulcers, with vertical edges. It is no uncommon thing to see patches, a foot or more in diameter, some parts of which have already cicatrized, while other parts show recent nodules, or serpiginous lines of scabs, spreading over the healthy skin around them. All these varieties are worthy of the most patient clinical study, for although they are absolutely characteristic of syphilis, the patient often has no suspicion of their nature. They have a special tendency to affect the face (when they are seen on the forehead, nose, and lips, rather than the cheeks), the nape of the neck, the shoulders, the back, and the extensor surfaces of the limbs.

As an illustration of the importance of the correct diagnosis of such affections, I may mention the case of a gentleman, æt. 55, who came to me for the most obviously syphilitic ulcerating patches, one on each shoulder, which had been steadily getting worse for a year. His medical adviser had put no question to him as to his having had any venereal disease, and all that he could tell me was that when a young man he had had some complaint of this nature, attended with swelling of the testis. He had been married for many years; his wife had had three miscarriages but no children. Iodide of potassium cured one of the patches in a fortnight, and the other within six weeks.

The later period of the disease is also characterized by lesions of the throat and mouth, most of which differ from those of the secondary stage. *Plaques opalines* may, indeed, continue to form on the cheeks or lips. But the surface of the tongue now for the first time becomes affected in a variety of ways. Sometimes more or less extensive patches appear unnaturally smooth and glossy. Sometimes there arise oval grayish ulcers, especially on its centre or edges. Sometimes its mucous membrane becomes greatly thickened, with deep grooves intersecting it in all directions. All these changes render

the contact of salt or pepper exceedingly painful to the patient, and make him glad to confine himself to the blandest possible diet.

In other cases, again, the soft palate becomes affected with deep, sharp-cut ulcers, which very rapidly perforate it, and eat away a considerable part of its substance. Such ulcers, when they heal, leave well-marked cicatrices. Some years ago a woman died in Guy's Hospital whose velum had long before been extensively destroyed on one side, so that the uvula was held in its place by two thread-like processes of mucous membrane, which looked as if they could not possibly have escaped being torn through during deglutition; the preparation is now in our museum. Sometimes the palate may become adherent to the pharynx, so as to cut off the communication between the cavity of the nose and the air passages below.

Gummata and Nodes.—That I have not yet mentioned gummata as occurring in the skin and mucous membranes will probably surprise many of my readers; for some writers characterize the tertiary period as the "gummatous stage" of the disease.* In the subcutaneous tissue, or in the substance of the tongue, such tumors often acquire a very considerable size; and the skin or the mucous membrane over them may at length become ulcerated through, so as to expose a gray, degenerating mass, of the most typical kind. But it is, I think, impossible to say that tertiary eruptions in general deserve to be called gummatous, to the exclusion of the early ones. I have already remarked that the scattered lenticular papules which may constitute one of the first manifestations of constitutional syphilis have a structure which seems not to be merely inflammatory; and recent observers have shown that the nodules which are found in iritis are small gummata, instead of consisting merely of fibrinous exudation. Again, Mr. Hutchinson has mentioned a case in which definite gummata were found in both testes and also in the spleen, although a secondary rash was still out on the skin of the patient, who died of "syphilitic disease of the heart —myocarditis with gumma."

The gummatous affections of internal organs are, indeed, by far the most important of the effects of syphilis, since they produce various symptoms, and not rarely destroy life. But I shall leave them to be described among the local visceral diseases, since I could in no other way bring their clinical relations into sufficient prominence. In this place I shall content myself with insisting on the necessity of carefully examining the bones and the testes whenever a syphilitic taint is suspected.

It is believed that those bones which, like the skull-cap, clavicles, ulnæ, and tibiæ, are but thinly covered with soft parts are, more than others, liable to be affected. By running one finger along the surface of these bones it is easy to discover whether there is any swelling of them, or whether the patient shrinks from pressure. The enlargement caused by syphilitic periostitis generally rises gradually from the surrounding surface, but I remember an instance in which the frontal and the parietal bones presented bold hemispherical tumors that at first appeared as if they must be malignant growths, especially as one upper eyelid was enormously swollen and in part gangrenous; the effect of treatment with iodide of potassium in this case was extraordinary. When such osseous affections are recent, they often feel soft and semi-elastic, and at an autopsy one may find that there is a gray succulent material, more or less extensively caseating,

* The term *gumma* is one of considerable antiquity. Fallopius, in the sixteenth century, spoke of tumors of bones as having been called "*gummata gallica*," on account of their containing a matter resembling *gummi eliquatum*. Wagner has recently proposed to substitute the name "syphiloma" for gumma; but I fail to see any advantage in this suggestion.

which can be shelled out of the substance of the bone, which had been excavated to receive it. At a later stage the soft substance may be absorbed, and there may be left a central depression, with a thickened zone around it. But upon the bones of the limbs, I think that the whole of the gummatous growth is more often converted into a uniform dense, raised, bony mass.

The testes, when affected with syphilis, often present firm, yellow gummata, of irregular form, which are embedded in the proper substance of the organ or in the epididymis, and which can often be easily detached during life. But in many cases a diffused fibrous thickening, with atrophy of the secretory structure, is all that one discovers at an autopsy. This, no doubt, suffices to account for the fact that so many of these patients are unable to beget children. But I suppose that one could hardly in the living subject recognize such an affection by palpation, distinguishing it from the flaccidity which may accompany any serious disorder of the general health. Nor am I satisfied that it is in itself a proof of syphilis; it is certainly found in the post-mortem room in many cases in which there is no other evidence of that disease. Still more doubtful, perhaps, is the significance of closure of the extremities of the Fallopian tubes in the other sex.

Diagnosis.—From the foregoing description of syphilis it must be sufficiently evident, not only that the recognition of a disease of which the symptoms and effects are so numerous and varied must often be very difficult, but that to attempt to lay down any rules for its diagnosis would be of very little use. Sometimes the first glance at a patient reveals to a skilled observer the nature of the case beyond a possibility of doubt; and he is bound to treat it actively, particularly in a married woman, without asking questions which might cause endless domestic misery. In other instances, even when all inquiries can be pushed without the least reserve, one is unable to hazard a positive opinion until one has watched the effects of remedies for a considerable time. It is important to remember that the body should always be stripped as thoroughly as possible, and that every part of it should be looked at before one pronounces as to the character of a doubtful eruption. Some of the most puzzling cases are those in which several cutaneous diseases are present at the same time. I well remember one instance in which a macular syphilide was almost hidden among the nodules of an acne induration and the papules and pustules of scabies.

Contagion.—With regard to the more usual ways in which syphilis is transmitted I need say very little, but it is perhaps worth notice that among married women belonging to the lower classes the disease is often traced back to a confinement, when in all probability the patient really derived it from her husband after her convalescence, he having become infected as the result of adulterous intercourse while she was in childbed.

There seems to be no doubt that the surface of the skin must be abraded, or fissured, to allow of the penetration of the virus, and the most recent writers think that this is true even of mucous membranes. Few facts in medicine are better established than the erroneousness of the doctrine of Ricord, that primary syphilitic lesions alone possess the power of propagating the disease. Clinical experience has furnished numerous instances of contagion from flat condylomata, and experiments have been made which show that it is possible to convey the virus to healthy persons by inoculation with the blood of syphilitic patients, or with matter from pustules of a secondary eruption or from an ulcer of the tonsils. Whether pus from a late or tertiary affection of the skin contains the poison, seems to be still

doubtful. There are also undecided questions as to the contagiousness apart from admixture with purulent discharge of the natural secretions of the salivary, mammary, or lachrymal glands; of the semen, without impregnation of an ovum; or of an inflammatory exudation from a mucous membrane, as, for instance, the gonorrhœal pus in one who also has syphilis. With regard to this last point it is suggested that if pus cells are wandering leucocytes from the blood, they must almost certainly contain any virus which the blood itself contains. But it is generally held that the saliva and the milk are not in themselves capable of conveying the disease. That mixed fluids from the mouth may form the vehicle by which the virus passes from one person to another is shown by cases of transference in the act of kissing; in glass-blowing, when the same metal tube is employed by different workmen in turn; in smoking, if one cigar or pipe is used by several individuals; or (as in a case recently related by Dr. Baxter) in cleaning the teeth with another person's brush. Nor must we forget that we may unintentionally infect our patients if we omit to thoroughly cleanse a Eustachian catheter, or a laryngoscopic mirror, after using it in a syphilitic case. The disease has also been transferred by one person biting another in the hand. Lastly, accoucheurs and midwives have acquired primary sores on the finger when they have overlooked a slight abrasion upon it during their attendance on a woman with flat condylomata, or other specific affection of the genitalia.

Pathology.—I have already hinted at the analogy between syphilis and the specific fevers, an analogy which has been particularly insisted on by Mr. Hutchinson, and I must now briefly discuss certain questions with regard to its pathology, some of which are of great practical consequence, while all of them have important theoretical bearings upon the study of contagious diseases in general.

Concerning the nature of the primary indurated papule or chancre, two opposite opinions are entertained. Some think that it is itself a local expression of an infected state of the blood. Others, following John Hunter, suppose that the virus does not reach the blood until after it has multiplied itself at the seat of inoculation, and has passed along lymphatic vessels, so as to be discharged with their contents into the great veins. In support of the second view, attention has recently been drawn to the fact that, in the ordinary indolent inguinal bubo, the glands which lie along the iliac vessels become swollen, as well as those which lie superficially in the groin. This is said to be shown by three preparations at the Lourcine Hospital, in Paris, each of which was taken from a woman who had died of some intercurrent disease while suffering from sores upon the vulva. But, as Bäumler remarks, if such were the mode of transmission of the syphilitic poison, one would expect that under ordinary circumstances the constitutional symptoms should be longer delayed than when, the seat of the primary affection being on the lip or on the finger, only a single group of glands would have to be traversed, and I think that even if the *irritation* which causes the glands to swell does spread from one gland to another, it by no means necessarily follows that *infection* of the blood takes place by the same slow and circuitous route. The analogy of vaccination seems to suggest that it is quite possible for an indurated papule or chancre to be the direct result of inoculation of the specific virus *in situ*, and yet for the blood to be already poisoned from the moment when such a lesion begins to be developed. The rarity of multiple primary syphilitic sores, and the fact that the secretion of a primary sore is scarcely ever inoculable upon the patient, point strongly in the same direction. One might have expected to settle the point at issue by excising indurated chancres, or destroying them with caustics at their

very commencement, but at present the evidence as to the effects of such operations is conflicting.

Another question concerns the relation between the secondary and the tertiary stages of syphilis. Dr. Wilks has long thought that visceral lesions are most frequently met with in cases where cutaneous eruptions and other conspicuous symptoms of the disease had been absent or but slightly marked. But, although I am inclined to adopt this opinion, I do not know that it has been positively proved to be correct ; and, if it were, we should still have to ask whether it is not the omission of early specific treatment in the cases in question which determines the occurrence of remote effects. An opposite doctrine is maintained by Mr. Hutchinson, namely, that the tertiary phenomena—being, as we have seen, unsymmetrical—ought properly to be regarded rather as sequelæ of syphilis. His expression is that they are "regrowths," in morbid structures left behind from the secondary period. Of the points which he makes in support of his suggestion, the most important seems to me the fact that the liability to outbreak of tertiary symptoms sometimes continues after the patient has produced a family of healthy children, and when one can therefore hardly suppose that his blood still retains the virus.

It must not be imagined that this matter is one of merely theoretical interest. A patient affected with syphilis is apt to be exceedingly pertinacious in his inquiries as to whether the disease will leave a permanent taint. He has heard of instances in which, after the lapse of many years, tertiary symptoms have appeared ; and he has acquired a notion that the syphilitic poison differs from the contagious principles of all other specific diseases, in being never eradicated. But I think we are justified in assuring him that the distinction is one of degree, rather than of kind. In the immense majority of cases, a person who has had syphilis is, after a few years, free from it in every sense in which it can be said that one who has had scarlet fever or smallpox is free from those diseases. In each instance the only appreciable difference between the individual who has been attacked, and other people is, that he is no longer capable of taking the infection. And since we are not accustomed to speak of the exanthemata as remaining uncured during the whole lifetime of the patient, or until he falls exceptionally ill with them a second time, I do not see why we should adopt a modern fashion and use language of this kind with regard to syphilis. It is well known that a person very rarely has an indurated chancre and afterwards a secondary eruption and sore throat on two different occasions ; but several instances of such an occurrence have now been recorded. In a case of Mr. Hutchinson's the interval of health was only three years ; it has generally been longer. The second attack has always been very mild.

Opinions probably differ very widely as to the extent to which the presence of a syphilitic taint modifies other diseases. Some surgeons have held that it may interrupt the natural course of almost every morbid process ; that a broken bone may fail to become united, and a wound refuse to heal, until the virus is neutralized by the administration of specific medicines. But I very much doubt the fact of such occurrences, unless, indeed, the general health of the patient is profoundly impaired. On the other hand, it is well established that an injured part may gradually take on the characters of a syphilitic sore ; but then it is also the case that psoriasis or eczema may develop itself upon spots which have been bruised or lacerated in a person liable to one or the other of these diseases.

Prognosis.—This must be discussed with the treatment of syphilis, if we are right in believing that the administration of medicines has immense

power in cutting it short. To adults, indeed, it is scarcely ever directly fatal. Bäumler mentions, as the only two ways in which it can destroy life, hemorrhage dependent upon spreading ulceration (*phagedæna*), and exhaustion appearing when the early symptoms are very acute and severe. The only case that I know to have come down to the post-mortem room at Guy's Hospital, during the last twenty-five years, in which death was attributed to syphilis, apart from any visceral lesions, is that of a man, aged thirty-one, who lay in one of the wards for seven months, with a rupial eruption on the skin, and complaining of pain and swelling in his joints. The most extreme wasting took place; his knees and his left arm were rapidly contracted; his wrists and ankles were enlarged. Towards the last he was drowsy and listless, and at the autopsy the brain appeared to contain an excess of fluid. Indirectly, however, as we shall see in the chapters on diseases of the brain, larynx, lungs, liver, kidneys, syphilis is the cause of a very large mortality. If, therefore, I say that the prognosis is almost always favorable, it is because I hold that by proper treatment we can not only cure the early symptoms, but prevent their remote after effects, which are so dangerous.

Treatment.—Mercury has been employed as a remedy for syphilis during nearly four hundred years, but unfortunately the experience which appeared to justify its use was long vitiated by a most obvious source of error; the natural course of the disease, when unmodified by drugs, had never been carefully studied. Moreover, the metal was generally given so freely as to cause profuse salivation, with the most pernicious results. Thus, when certain English army surgeons, in the early part of the present century, introduced a "simple treatment," in which mercury found no place, they had the immense advantage that their patients remained free from many evils to which persons suffering under syphilis were then commonly subjected by their medical attendants. And what made the difference the more striking was that the real nature of syphilis as a specific disease being then altogether unknown, the same methods had been employed indiscriminately for all venereal sores. Is it matter for surprise that those who advocated this great revolution in medical practice should have overshot their mark, and attributed to mercury not only the injurious effects which it really does cause, but also many of the remote consequences of the disease itself, such as are now known to occur when no medicine has been administered?

It is now an established fact that it is possible for not only the primary lesion of syphilis, but also the various secondary and tertiary eruptions to subside spontaneously without any specific treatment; indeed, this result sooner or later occurs in the large majority of cases, when the patient is placed under favorable conditions as regards rest of body and mind, good diet, and pure air.

Nevertheless, the experience of the last few years tends irresistibly to the conclusion that a patient who has syphilis derives immense advantages from a course of mercurial treatment, provided that it is judiciously managed. Among those who are best qualified to form a judgment upon this matter there is at the present time hardly any difference of opinion; and what adds to the force of this consensus is that it has been arrived at by a variety of methods, and that some of those who now most warmly advocate the use of mercury were originally in doubt, or even active in opposition.

1. It is certain that mercury, properly administered, is in no way injurious to the patient's general health, if his constitution is sound and fairly robust. Even in the case of a person with a turbercular tendency, the drug may almost always be given with perfect safety, although one should

perhaps be more cautious with it. Nor would the actual existence of visceral disease prevent my prescribing it, except that one naturally would refrain from treating a mild form of syphilis at all, if the patient were obviously about to die in a few months, of phthisis, cirrhosis of the liver, or renal dropsy. It is generally thought that syphilitic affections attended with much ulceration should, if possible, be treated by other remedies rather than mercury, but Mr. Hutchinson is now inclined to doubt the correctness of this opinion. The cachexia resulting from syphilis, so far from being a reason for avoiding the use of mercury, is generally quickly curable by it.

2. Mercury has a most marked effect upon the induration of a primary infecting papule or chancre. The treatment of such affections belongs to surgery rather than to medicine, and I shall not further touch upon it. But I may quote Mr. Hutchinson's statement, that the remedy almost invariably arrests the process of induration if given when it is just beginning; while at later periods it no less surely, though more slowly, effects the reabsorption of the indurating material.

3. Mercury, if administered systematically during the existence of a primary indurated sore, will probably often prevent altogether the development of secondary symptoms. This is perhaps more important than any other point connected with the treatment of syphilis, and it deserves the most careful verification. At present the best evidence in favor of it is the statement of Mr. Hutchinson, in a paper read before the Hunterian Society, in 1874, especially when contrasted with his previous utterance on the same subject in Dr. Reynolds' "System of Medicine." Formerly he thought that secondary symptoms were for the most part inevitable, whatever treatment might be adopted. But in a series of eleven cases of vaccino-syphilis, in which he began to give mercury two months after contagion, and about a fortnight after the appearance of the specific induration, not only did every patient quickly get rid of chancre and glandular enlargement, but only about half of them ever showed secondary symptoms; these, moreover, were almost always of an exceedingly mild type, and did not appear until many weeks later than they would have done if no medicine had been taken. Now, where syphilis has been intentionally inoculated upon healthy persons constitutional effects have scarcely ever been wanting; and the same may be said of cases of vaccino-syphilis in which early mercurial treatment has not been adopted.

4. Mercury renders the secondary symptoms of syphilis mild and of brief duration in proportion as its administration is begun early and is carried on regularly and without intermission for a considerable time. Upon this point also Mr. Hutchinson has recently spoken very decidedly. Formerly he cited instances in which a patient, while actually salivated for iritis in one eye, became attacked with the same affection in the other eye, as tending to establish an opposite conclusion; but now he says that in such cases the failure is probably due to the fact that the "salivation is premature," that is, that the remedy has not yet excited its full influence upon the rest of the body, notwithstanding that it has affected the gums so severely. "The secret of success is to avoid any interruption of this kind." And he insists upon the fact that cases in which the secondary symptoms of syphilis are exceptionally severe are almost always cases in which mercury has not been given. My own experience leads me to confirm this statement very strongly.

5. Mercury, if administered efficiently during the primary and secondary stages of syphilis, will probably diminish the frequency and severity of tertiary symptoms. Here, indeed, we come to a point as to which it is and must be difficult to obtain direct proof, for there are no certain data as to the proportion of cases of syphilis which result in tertiary symptoms when

no treatment is used; nor do we yet know to what extent their occurrence may depend upon the severity and character of the secondary symptoms. But there is at least a strong presumption that if the drug can prevent the earlier and more constant of the phenomena of syphilis, it will not have less power over the more remote and exceptional effects of the disease.

If these statements should prove to be correct, it would evidently be the bounden duty of a medical man to prescribe mercury to all patients suffering under primary or secondary syphilis who should place themselves unreservedly under his care. And I entirely agree with Mr. Hutchinson that we cannot insist too strongly upon the necessity of continuing the remedy regularly for many weeks after the entire subsidence of every visible symptom of the disease.

There has been some discussion as to whether mercury should be called an "antidote" to the syphilitic virus. Mr. Hutchinson is disposed to give it this title. But, for my own part, I should prefer to avoid using any expression which would imply that the cure of syphilis must depend upon, and be preceded by, the disappearance of the specific contagion of the disease. I think it is at least as likely that mercury acts directly upon the living tissues, depriving them of their susceptibility to the particular kinds of morbid action which syphilis induces. The notion of an antidote is, we believe, inapplicable to such a case as the cure of psoriasis or pemphigus by arsenic; and I do not think that it could properly be extended to include the cure of gout by colchicum. But surely the use of mercury for syphilis has a far more obvious relation to these therapeutic measures than toward the treatment of cases of poisoning, whether by mineral or by vegetable substances.

With regard to the relative advantages of the various preparations of mercury, differences of opinion prevail. But I think it is clear that we should employ in preference those which are mild; it is rarely advisable to prescribe *calomel* or *blue pill*, in frequently repeated doses, except, perhaps, when it is necessary to produce a rapid effect, as when iritis or retinitis is present. Nor can I believe that the hypodermic injection of the *bichloride*, or of *calomel*, promises advantages which counterbalance its pain and its tendency to set up inflammation. I have generally been content to administer the *bichloride of mercury* by the mouth, in doses of one-sixteenth to one-twelfth of a grain, three times a day. Another preparation which may often be used with advantage is the *hydrargyrum cum cretâ*, in doses of two or three grains; when the gums appear likely to be too quickly affected, further inconvenience may be obviated by letting the patient at the same time take the chlorate of potass; but, as a rule, it is not desirable to mask the effect of the mercury in this way. Sometimes, when no mercurials taken internally could be borne, the inunction of *blue ointment* has succeeded perfectly. But best of all, in really difficult cases, is the *calomel vapor bath* of Mr. Lee. About fifteen grains may be volatilized by a spirit lamp over a water bath, the apparatus being placed beneath a chair on which the patient sits, naked, and wrapped round with blankets. The bath should last from a quarter of an hour to twenty minutes.

Except during the cold seasons of the year, the patient may go out, and even continue at his occupation, while he is carrying out a mercurial treatment. He should, however, keep early hours and take plenty of rest; his clothing should be made of flannel, and other warm materials; he should have good food, but should generally abstain from stimulants. Pure air is important to him; there are greater advantages in residing at the seaside than at Aix-la-Chapelle or other continental spas.

Even when syphilis has reached the tertiary stage, mercury should, I think, as a rule, be prescribed, if the patient has not already gone through a

systematic course of it. But in the majority of cases the remedy which is now most serviceable is *iodide of potassium*. This is, indeed, often employed with advantage, even during a protracted secondary period; but over the early macular and papular eruptions it has little if any power. With regard to the dose of it, there has been a good deal of difference of opinion. But of late it has become the general practice to give from ten to thirty grains three times a day. It is generally prescribed with spiritus ammoniæ aromaticus and some bitter tincture or infusion. But some persons take it more readily in milk, which they keep on a table beside them and sip from time to time.

Even when iodide of potassium does not finally eradicate the disease, it often exerts so marked an influence over the symptoms, that the patient will go on with it more or less continuously for years. In such cases it is generally desirable to interrupt its use from time to time; and important services are then rendered by the chlorate of potass. I have repeatedly seen obstinate forms of cutaneous eruption removed by this salt, given in doses of ten grains, three times daily. Another medicine which may now be prescribed is the dilute nitric acid, and still more valuable is, I believe, *sarsaparilla*. I have used the fluid extract with results which appeared to me to be incontestable. Of the value of guaiacum, which three centuries ago had so high a reputation, I know nothing.

It is not generally worth while to apply remedies directly to the eruptions of syphilis, but some of the more localized tertiary affections are very rapidly brought to subside when they are covered with the diluted blue ointment, and calomel should always be dusted upon mucous patches (*i. e.*, flat condylomata). The severe nocturnal pains produced by nodes may often be relieved by blistering the skin over them.

Congenital Syphilis, unlike the ordinary form of syphilis in many respects, is a modification of the disease, which, instead of being acquired by direct infection, is transmitted to the patient from one or both parents. Some modern writers prefer to call it "hereditary" or "inherited," because it does not usually manifest itself by well marked symptoms until some weeks after birth. Recently, however, the discovery has been made that the bones in the fœtus are often affected by syphilis, and I do not think that there can be any doubt that the malady, even if latent, is really present from the very commencement of extra-uterine life, just as it is in the adult during the period of incubation, or in the intervals between successive eruptions. The term hereditary may well be reserved for diseases such as gout and phthisis, which usually begin at a later age, the tendency to them being all that is handed down.

At one time it was thought that infection to the fœtus *in utero* must necessarily come from the mother; even when the father had been the original sufferer, the idea prevailed that she always received the virus first and gave it to her offspring. But observation proved that in a large majority of cases the mother neither showed any sign of the disease nor knew that she had ever had a symptom of it. All writers admit that the semen may convey syphilis directly to the ovum; and the assertion is frequently made that the father is the parent from whom the disease is most often derived. But instances must be rare in which it would be possible to declare positively that at no period since her marriage had a woman gone through even a slight form of syphilis, especially as it is not certain whether the semen may not also infect her, independently of the occurrence of impregnation. But little stress, therefore, can be laid upon the well-known fact that a man sometimes transmits the disease to his children notwithstanding that he did not marry until long after the disappearance

of all secondary eruptions, nor until a period when, if any further symptoms were to arise, they would be regarded as tertiary and as incapable of being directly contagious. It would seem, however, that such cases are in the highest degree rare and exceptional. If one is consulted about the propriety of marriage on the part of a patient who has had syphilis, one should perhaps never declare it to be impossible for the offspring to be tainted. Until two years have passed after the complete subsidence of the secondary symptoms of the disease marriage must be altogether forbidden. But lapse of time seems, as a rule, to quickly diminish the risk. Thus, as Mr. Hutchinson has pointed out, each succeeding child of the same parents is less likely to suffer than the previous one; sooner or later the taint wears out, and the children subsequently born remain free. Sometimes, however, one infant may escape notwithstanding that both older and younger ones are attacked.* There are, moreover, modifying circumstances which may cause the later children of a married pair to suffer more than the earlier, even when the syphilis had been contracted long previously. According to Mr. Hutchinson, such a result sometimes happens when the general health of the infecting parent becomes more and more broken down from year to year; but the more frequent cause is supposed to be the fact that a healthy woman impregnated by a syphilitic husband gradually becomes herself poisoned, so that she forms a second source of infection for the children whom she afterwards brings forth. For there is reason to believe that the offspring are much less likely to escape, and that they are more often affected severely when both parents are tainted than when only one is. The full extent and subtlety of the contamination which a woman may indirectly derive under the conditions just referred to, were first pointed out by Colles, of Dublin, in 1837. He formulated the remarkable law which now bears his name, that "a newly-born child, even although it may have symptoms in the mouth, never causes ulceration of the breast which it sucks, if it be its mother who suckles it, though it is still capable of infecting a strange nurse." In other words, although the mother may have shown no sign of syphilis, she has yet undergone a modification of the disease, bearing somewhat the same relation to the ordinary form that vaccinia does to smallpox, and no less protective in its action. The *choc en retour* (as French writers have termed the infection of a woman through her fœtus) is not, however, always unattended with symptoms. She not infrequently becomes pale and thin, her hair may fall off, perhaps certain lymphatic glands become swollen, or there may be some swelling of bones. Mr. Hutchinson has observed that symptoms may first appear at the menopause, in the form of so-called "psoriasis palmaris," sores on the tongue, or indurations in the legs. He suggests, as an interesting point for inquiry, the question whether a woman infected in this way *per fœtum* can transmit the taint to children subsequently born to a healthy father.

The effects of syphilitic infection on the fœtus vary greatly in different cases. Very often it dies *in utero* and is thrown off after an interval, in a more or less decomposed condition, with its cuticle peeling in large flakes, as the result of maceration in the dark and fetid liquor amnii. Thus abortions and miscarriages, especially if several times repeated, afford very valuable indications of a syphilitic taint; so that not only is it important, whenever a women suffers from any complaint that could be attributed to this cause, to inquire as to the mode of termination of her former

* The case is, of course, different when a husband or a wife contracts syphilis after having already had children; their next infant will then be the one most severely affected. It is generally said that a woman acquiring the disease during her pregnancy may convey it to the fœtus at any period up to the seventh month.

pregnancies; but also, when a married man is the patient, to make similar investigations with regard to his wife.

The occurrence of a specific lesion of the *placenta* has long been suspected, but until recently nothing definite was known about it. In 1863 Dr. Wilks cited in the "*Guy's Hospital Reports*" some observations of Mr. Wilkinson King, in which the chorion had been found thickened and coriaceous, and the amnion lined by a false membrane, as the result, it was supposed, of syphilis; in almost every instance abortion had taken place at the third month. Virchow, however, afterward described the decidua and the maternal part of the placenta as the structures liable to morbid changes in this disease. If his view had been correct, it would have followed that the changes in question must be absent whenever the fœtus derives its infection solely from the father; but there is reason to believe that most of the hard, yellow masses in the placenta which have been taken for gummata have been mere residues of accidental hemorrhages. At any rate, gummata spreading from the maternal into the fœtal part of the placenta were only once found by Fränkel, of Breslau, who in the "*Archiv für Gynakologie*" for 1873 has recorded a series of investigations with regard to this subject. In sixteen cases, however, he discovered a peculiar affection of the villi themselves, a fact of great interest, since it is obviously compatible with an entire absence of infection from the mother. The lesion in question consists in a dense growth of round or spindle cells in the substance of the villi, attended with a more or less complete destruction and disappearance of their vascular loops, and ending ultimately in a process of fatty degeneration. Villi so altered are less easily isolated from the structures in which they are embedded; they are swollen and opaque, and have irregular outlines and bulbous extremities. Sometimes the whole placenta is uniformly affected; it is then remarkably large and heavy (even to the weight of two pounds), close, tough, and of a pale grayish-yellow color. In other instances, only certain portions of it are diseased; these appear as opaque, wedge-shaped masses, surrounded by zones of congestion; in the healthy portions there are often hemorrhages. Once Fränkel found that the cell-growth had extended from the villi into the maternal part of the organ. He is in doubt whether the premature expulsion of the fœtus, which in his cases took place at varying periods from the sixth month onward, is dependent upon the placental lesion directly or rather upon the death of the fœtus. In some instances gestation appeared to have gone on to its natural limit, and when the morbid change was partial the child was sometimes born alive. The earliest period at which the affection of the villi was detected was at the sixth month; it often seemed to be only just commencing. Thus we have still to learn whether a similar lesion exists when abortion occurs at the third or the fourth month.

Once or twice the fœtus showed gummata in the *liver*, but in the great majority of cases what alone characterized the placental disease as syphilitic was a peculiar morbid condition of the *bones*, which was never absent. This affection, first described by Wagner, of Berlin, in "*Virchow's Archiv*" for 1870, but independently observed by a French physician, Parrot, at about the same time, seems to begin at a very early period of fœtal life. Like that which constitutes rickets, it has its chief seat at the meeting lines between the shafts of the long bones and their epiphyseal cartilages, and, in the case of the ribs, between their anterior extremities and the cartilages which tip them. The meeting lines in question become much thicker than natural, uneven and irregular. But here the resemblance ceases. There is in syphilis comparatively little increase of the normal "zone of proliferation," whereas the "zone of incrustation of cartilage" is enormously exaggerated. It forms a thick layer, dense and homogeneous,

but friable, white, and opaque, like mortar; long processes project from it into the substance of the cartilage beyond. As the affection advances, this layer becomes separated from the shaft by a soft, or even semi-fluid, grayish-red or yellowish material, consisting of granulation tissue, which may shade off into pus. Another feature is the formation of new bone (osteophyte) around the outside of the shaft in the neighborhood of the epiphysis.

Wagner terms this lesion a "syphilitic osteochrondritis." It is widely diffused throughout the body, but it is said to be always most marked where the growth of osseous tissue is naturally most active; and what is interesting is that precisely the same distribution is stated to obtain in the case of rickets. Thus the lower end of the femur is the favorite seat of both diseases, while the corresponding part of the humerus is least often affected by either. Parrot, however, says that in syphilis the osteophyte is most abundant just where the other changes are least developed; and this corresponds exactly with a remark long ago made by Mr. Hutchinson, to the effect that periostitis in congenital syphilis is more often met with just above the elbow joint than anywhere else.

In a few exceptional instances, when an infant infected with syphilis is born alive, the soft material between the shafts of the long bones and their epiphyses increases to such an extent as to detach them from one another, and even to form fluctuating, purulent swellings beneath the periosteum. There is then a complete loss of power in the limbs, attended with so little pain that it has actually been mistaken for a paralysis. The child's hands are described by Parrot as lying pronated by its side; its legs are extended, and when it is lifted up they hang helpless, and swing backward and forward. After death, almost every one of the principal epiphyses may be found separated from the bone to which it belongs.

Henceforth there ought to be little difficulty in diagnosing cases of this severe kind, but it is otherwise with the ordinary form of the disease, in which there is seldom a sufficient degree of enlargement of the ends of the bones to justify one in speaking positively about it, at least in very young infants. Indeed, Köbner has shown that even after death the microscope is often required to reveal the presence of the lesion.

Nor, as a rule, does a child infected with syphilis manifest any other symptoms of the disease when it is first born. The only exception is, perhaps, a peculiar and very rare form of bullous eruption, which is known as *pemphigus neonatorum*, and which may either be present at the time of birth or come out a few days later. In 1851 there was a discussion upon this affection in the Academy of Medicine in Paris; Paul Dubois declared that it was syphilitic, while Cazeaux maintained the contrary. The question, so far as I know, has never been cleared up; but probably an examination of the bones in future cases would easily settle it. The bullæ are flaccid, and contain opaque serum or pus; their favorite seats are the soles of the feet and the palms of the hands. I have never myself seen a case. This eruption generally proves rapidly fatal.

The *thymus* has been found in a state of suppuration by Dubois; there was no abscess, but the organ when squeezed emitted drops of yellowish pus, easily distinguishable from the opaline liquid which it may contain when in a normal state. Another morbid condition, first pointed out by Depaul, as occurring in the bodies of syphilitic children, consists in the presence of yellowish-gray indurated nodules in the *lungs*, softening in their centres into cavities. Lastly, Gubler has described a peculiar change in the *liver;* the organ is not only enlarged, hard, and elastic, but its cut surface shows a number of small, white, opaque grains on a uniform yellowish ground. Dr. Wilks showed a specimen of this affection at a

meeting of the Pathological Society in 1866; it came from an infant one month old. I once saw suppuration of the thymus in a child who had lived for some months. But, generally speaking, if congenital syphilis ends fatally, except in early infancy, it is by some accidental complication, such as broncho-pneumonia, or diarrhœa, or marasmus from improper food, and the internal organs then seldom present characteristic lesions. Acute peritonitis, or pleurisy, or meningitis has sometimes been found to be the cause of death.

The more common symptoms of congenital syphilis generally begin to show themselves toward the end of the first month of extra-uterine life or in the course of the second month, rarely after the end of the third month. The earliest is, in most cases, a *nasal catarrh*, producing what is popularly called the "snuffles." This is attended with the discharge from the nostrils of a fluid, at first thin and serous, but afterwards viscid, so that it dries up into crusts which obstruct the passage of air. Consequently the child is no longer able to breathe while it is at the breast; it takes the nipple into the mouth only to drop it again, and, as Diday pointed out, it fails to get enough food, and rapidly loses flesh. Indeed, although syphilitic infants are sometimes at birth well grown, plump, and fat, and may even remain so throughout the whole course of the disease, the rule is that they are from the first, or soon become, pale and wasted; they have a dull, opaque, yellow, and wrinkled skin; to use a favorite expression, they "look like little old men." Soon after the catarrh there appears an *eruption*, of which the favorite seats are the nates and the face, but which may cover the whole of the body and limbs. It consists of maculæ, blotches, or flat papules, which may be bright red, brownish, or copper colored. They are sometimes isolated, sometimes so confluent that they have been described as having an "erysipelatous" character. It is often difficult to distinguish between syphilis and the effect of the nurse's negligence in allowing the parts about the anus to remain soiled with execreta, or in drying them insufficiently after having washed them. As a rule, the red blush produced by mere irritation is ill-defined and fades away at the edge, whereas at the corresponding part of a specific rash there are to be seen a number of isolated, sharply-outlined spots. The papules themselves may have smooth and glazed surfaces, or they may be dry and horny, or raised here and there into flaccid bullæ, or superficially ulcerated. At the corners of the eyes, about the angles of the mouth, in the folds of the neck or of the groins, or around the anus, the papules often become covered with a moist, opaque layer, and assume more or less the appearance of flat condylomata. But I am inclined to agree with Mr. Hutchinson, that really typical "mucous patches," occurring chiefly at the anus, are comparatively seldom seen in children less than eight or ten months old. Sometimes the eruption takes a pustular form; the greater part of the body may then become covered with moist scabs, separated by cracks, from which a sero-purulent fluid is constantly oozing. In the interior of the mouth ulcers are often present, or there may be a diffuse *stomatitis*. The latter affection involves the gums and dental sacs; Mr. Hutchinson has even seen it lead to necrosis and exfoliation of the alveoli. The mucous membrane of the nose may likewise ulcerate, and discharge a sanious fluid, perhaps containing fragments of bone from the septum, which becomes perforated.

Another but a rare symptom is *iritis*. Of this Mr. Hutchinson, some years ago, cited twenty-three cases, the majority of which occurred in girls. It was usually seen at about the age of five weeks. Sometimes it was limited to one eye, sometimes it affected both eyes. It was attended with irregularity of the pupil, alteration of color in the iris, and the exudation of white, yellow, or red lymph. But it required to be carefully looked for,

as the cornea was generally clear, and had but a very faint pink zone around its margin.

I have already remarked that although, in accordance with Colles' law, a child suffering under congenital syphilis is incapable of infecting its mother, it may give the disease to any other woman who suckles it. Diday even records cases in which women advanced in years appear to have contracted syphilis by kissing such infants; and the use of a spoon that had previous been employed in feeding them has been followed by the same terrible consequences. Probably the source of the virus is always the secretion of an ulcer about the lips or within the mouth. Hence Diday lays great stress on the importance of searching for such ulcers, when a child born of syphilitic parents is to be placed with a wet nurse. But as it is practically impossible to be sure that the back of the throat is healthy, French writers advise that as soon as any cutaneous eruption, or even a nasal catarrh is discoverable, one should, for the nurse's sake, insist that she should no longer suckle such an infant. If one knew a woman who had had syphilis she might be engaged for the service; but otherwise recourse must be had to the feeding bottle. The primary lesion on the nipple of a nurse is described by Diday as a small red papule, slightly desquamating on the surface. When once infected, she, in her turn, may convey the disease to other infants. That the milk is not then the vehicle of the poison is shown by an observation of Mr. Henry Lee's. A woman, who was nursing a syphilitic child with one breast, acquired an ulcer upon that breast, and afterward an eruption; but her own infant, for whom she reserved the other breast, went on sucking for six weeks and remained healthy.

I do not find any evidence that syphilis contracted by an infant from the wet nurse differs from the congenital form of the disease in its symptoms, as might be expected.

Treatment.—We have seen that congenital syphilis seldom proves directly fatal, except soon after birth. This, at least, is my decided opinion. In very mild cases the eruption often subsides in a few weeks when left entirely without treatment; and in the more severe forms of the disease specific medicines have immense power. I have generally been accustomed to prescribe the *hydrargyrum cum cretâ*, in doses of a grain, or two grains, two or three times a day, according to the age of the infant; a minute quantity of Dover's powder, or of carbonate of soda may be given with it, according to circumstances. Many prefer, however, to use the diluted *unguentum hydrargyri* externally; it may be applied on the inside of a flannel band which is sewn around the knee, as Sir Benjamin Brodie recommended; or ten or fifteen grains of it may be rubbed into the child's armpits once or twice daily, for a few minutes at a time. Or the *liquor hydrargyri perchloridi* may be ordered in doses of ♏xx to ♏xxx, three times a day. The effect of these various measures is generally to remove the visible signs of the disease in from one to three months. Sometimes relapses occur; and in such cases, or where the improvement is slow, it may be advisable, in addition to the mercury, to administer the *iodide of potassium* in doses of two or three grains. Mucous patches about the anus may be dusted over with calomel.

Later Effects.—The subsidence of the usual symptoms of congenital syphilis is occasionally followed by the development of others, which are obviously analogous to those that constitute the "tertiary" stage of syphilis in the adult. Thus nodes may appear. Mr. Hutchinson says that the bone most affected by them is the humerus at its lower end, and that they often reach such a size as to impair the movements of the elbow joint. I have seen extensive periosteal swellings upon the tibia and upon the ulna in a girl who must have been eight or ten years old; she originally came to Guy's Hospital on account of large ulcerating or rupial patches,

covered with thick brown crusts, on her trunk and limbs. Under iodide of potassium they healed with marvelous rapidity, but she nevertheless kept returning to me for two or three years. Another form of eruption of which I have seen several instances in not very young children, consists of raised red rings or zones, somewhat like the "circinated squamous syphilide," of adults. At the Evelina Hospital I once saw a girl, eight or ten years old, with a sloughing ulcer which had destroyed the soft palate, and with great enlargement of the metacarpal bones of one hand. Mr. Hutchinson cites the case of a boy, aged eight, almost the whole of whose calvaria was involved in disease at first regarded as "strumous," but which proved to be syphilitic; he was the son of a clergyman, but his mother had contracted the disease from a former husband, an officer in the army. I have once or twice met with nodular swellings, doubtless gummata, upon children's testes. With regard to affections of the cerebral arteries, or of the brain itself, I shall have something to say hereafter. Of lardaceous changes in the viscera, as the result of congenital syphilis, I at present know nothing.

The most interesting of the remote effects of the hereditary disease are, however, some which differ altogether from those of ordinary acquired syphilis. Mr. Hutchinson has shown that syphilitic children often grow up with a special physiognomy. One peculiarity is that on each side of the forehead the frontal eminence is protuberant. This he formerly regarded as the result of an antecedent hydrocephalus from chronic meningitis. But M. Parrot has recently maintained that in addition to the intra-uterine osseous lesions already described, syphilis gives rise, during later infancy and childhood, to a morbid change in the bones bearing a somewhat close relation to rickets; and Mr. Hutchinson now accepts such a change as accounting for the appearance in question. A character on which M. Parrot has further insisted is the presence of four eminences upon the bones forming the sides of the anterior fontanelle; from their resemblance to the buttocks he calls such a calvaria "natiform." He also claims for congenital syphilis the affection known as Craniotabes, which has hitherto been deemed rachitic. But, although Dr. Barlow and Dr. Lees have supported his views, it appears to me very doubtful whether Parrot's cases ought not to be regarded as examples of the accidental presence of rickets along with syphilis; and the more so, as he is disposed altogether to ignore the former affection as an independent malady. Further observations will no doubt soon settle this question.

The remaining points originally detected by Mr. Hutchinson are far more distinctive than the shape of the forehead. One is a peculiar, broad, and sunken state of the bridge of the nose, resulting from long-continued swelling of the parts within, or from loss of support by exfoliation of the septum. Another is the presence of radiating linear cicatrices at the angles of the mouth, running outward toward the cheeks. A third is a thick, pasty, opaque condition of the skin generally, the hair being scanty, and the nails sometimes broken and disposed to split into layers. But most characteristic of all is a peculiar change in certain of the permanent teeth, and particularly in the central upper incisors, which for this purpose Mr. Hutchinson terms the "test-teeth." Not only are they much smaller than usual, but they are "notched" and "pegged;" the former peculiarity consisting in a deep crescentic excavation of the free edge, the latter in a gradual tapering of their sides toward this edge. It is supposed that this alteration in their form is the result of their having been disturbed in their growth by the stomatitis which is so marked a feature of the disease during infancy. When they first protrude from the gums, the notch is not present; in its place there is a row of minute projections; these soon afterward

break off. Similar changes may sometimes be observed in the case of the other incisors. It is important to note that the above single deep notch is alone indicative of syphilis. Horizontal grooves are common enough in persons who are altogether free from any congenital taint.

The appearances just described enabled Mr. Hutchinson to identify as syphilitic an affection of the eye, the real nature of which had not previously been suspected, and which was always known as "strumous corneitis." This disease, *interstitial keratitis*, as it is now called, occurs chiefly in children between eight and fifteen years old, but sometimes in adults up to the age of twenty-five or twenty-six. Mr. Hutchinson has only once seen it as the result of acquired syphilis. It is more common in girls than in boys. It seldom remains limited to one eye, but commonly attacks the two eyes in succession at an interval of a few weeks. It begins as a dotted haziness near the centre of the cornea, which spreads until almost the whole of it is densely opaque, like ground glass. The patient often complains but little of intolerance of light. There is not generally, at first, much congestion of the conjunctiva or of the sclerotic. Ulceration never occurs, but at a certain stage the cornea may become so vascular as to be uniformly pink or salmon-colored. When the affection is at its worst, vision is often reduced to a bare perception of light. But in the course of a year or eighteen months a surprising degree of improvement takes place. The opacity very slowly clears up, until perhaps only a few hazy patches remain; and the patient's vision may ultimately be but little impaired. Sometimes an iritis occurs as a complication, and sometimes a choroido-retinitis.

Another morbid condition which Mr. Hutchinson has seen in the subjects of congenital syphilis is a bilateral deafness, dependent apparently on some disease of the deeper parts of the ears. It comes on at about the same age as does interstitial keratitis.

Two questions in regard to the after effects of congenital syphilis still require to be briefly considered.

One is, whether a person who has passed through it in infancy is less liable than others to acquire the disease later on in life. Mr. Hutchinson has recorded some instances in which venereal sores have been contracted under such circumstances, and one in which an outbreak of constitutional symptoms occurred; and his opinion is, that the protection conferred by the fact of a patient's parents having had syphilis probably takes the form of rendering the acquired disease milder, rather than of preventing it altogether.

The other question is, whether it is possible for the taint to be handed down to a third generation so as to produce definite symptoms. In answer to this, Mr. Hutchinson says, that he has about eight times had opportunities of examining the children of persons undoubtedly the subjects of congenital syphilis. With one exception they appeared to be healthy. The exceptional case was that of the infant of a woman who came to him for interstitial keratitis, and who had also notched teeth and a characteristic physiognomy. She said that nothing ailed her baby; but on inspection it turned out to be covered with coppery blotches, and to have condylomata at the anus and snuffles in the nose. Mr. Hutchinson had had the child's father under his care for a long time, and was convinced that he had never had any venereal disease. He was therefore inclined to believe that the virus had descended from the infant's maternal grandparents.

TYPHUS.

HISTORICAL SKETCH—INCUBATION—COURSE; FIRST WEEK; SECOND WEEK; CRISIS; RECOVERY; DEATH AND POST-MORTEM APPEARANCES—COMPLICATIONS AND SEQUELÆ—ÆTIOLOGY; CONTAGION OF TYPHUS; ITS SUPPOSED SPONTANEOUS ORIGIN—PROGNOSIS—TREATMENT.

Scarcely more than thirty years have elapsed since typhus fever was finally separated from certain other diseases with which it had been confounded. And yet we can trace back its history. For its prevalence as an epidemic, and the fearful mortality which always accompanies it, enable us to feel confident that many descriptions handed down by writers of former times were based upon a study of Typhus, unmixed with any of those forms of fever from which it is now known to be distinct. The name, indeed, is of no great antiquity, for it was first applied to a malady, or to a group of maladies, by Sauvages, in 1760. Until then it had, from the time of Hippocrates downward, been employed, in accordance with its etymology to designate a confused state of intellect, with a tendency to stupor. Murchison and others think that the plague of Athens, recorded by Thucydides, was really what we now call typhus. However this may be, there is no doubt about the nature of certain epidemic fevers which prevailed in Italy, France and Hungary, in the sixteenth century, and of which accounts were given by Fracastorius, of Verona, and by many other writers. During the course of the same century occurred the first three of the famous "Black Assizes" in this country, when judges, sheriffs, and jurymen were suddenly attacked with fatal illness, as the result of infection from the prisoners brought before them for trial. One of the older synonyms for the disease, indeed, is *jail fever*. Another is *camp fever* or *military fever*, from the ravages which it has committed among soldiers and camp followers, from the time of the Thirty Years' War, and the English Civil War, down to the siege of Sebastopol. Other names, again, are *spotted fever*, *brain fever*, *putrid fever*, from some of its more conspicuous symptoms. Of late years, it has been universally called Typhus in this country and in France; but the Germans are obliged to term it *Typhus exanthematicus* or *Flecktyphus*, because they commonly speak of Enteric Fever as *typhus*, dropping the epithet *abdominalis*, which in Germany is in strictness required to complete the designation of Enterica. On the Continent, however, the disease with which we are now concerned has generally been much less common than in the British Isles. Both in Great Britain and in Ireland, it has prevailed with great severity on repeated occasions during the last two hundred years. Since the commencement of the present century, there have been epidemics of it in 1803, in 1817–19, in 1826–28, in 1836, in 1843, in 1846–48, in 1856, and from 1861 to 1870. It must be noted, however, that in some of the earlier of these epidemics there was a large admixture of cases of another form of disease, Relapsing Fever, which was not known to be specifically distinct from typhus until 1843, but the previous occurrence of which can even now be easily recognized by the small mortality which has always attended it.

Incubation.—This is of variable length. Few cases afford an opportunity of determining it, for the disease is rarely the result of a single definite exposure to contagion. But Murchison collected for the "*St. Thomas's Hospital Reports*," in 1871, no fewer than thirty-one instances more or less directly in point. In two of them the effect was immediate, and the same thing had been noticed by other observers; the patient, having been conscious of an offensive odor proceeding from a case with which he had come into contact, was at once attacked with headache, prostration, nausea, rigors; and all the other symptoms developed themselves in due course. In one instance the period was not more than two days; in two, not more than four days; in one, from two to five and a half days; in one (that of Murchison himself), exactly five days; in one, not more than six days; in two, not more than ten days. Short incubation periods have been given by several other writers; thus, Lebert, in "Ziemssen's Handbuch," states five to seven days; and some cases have been recorded in which the disease followed exactly eight days after exposure. Murchison, on the other hand, found that in the larger number of his thirty-one cases the period was longer: in four, it was exactly twelve days; in thirteen others it was within a few days of this, on one side or the other; and in four instances it was ascertained to be longer than twelve days; being in one patient not less than thirteen days; in another, not less than fourteen days; in the third, exactly fifteen days; in the fourth, not less than twenty-one days. During the incubation, the health generally appears to be perfect; sometimes, toward the end of it, there is a little malaise, with headache, pains in the limbs, and loss of appetite. Lebert says that these symptoms may be protracted over several days.

Course.—The course of typhus is best studied by a division into weekly periods. In the following account of it I shall, in the main, follow Murchison's description, but with regard to the facts all writers are agreed:—

First Week.—The onset of the disease, from which the first week is reckoned, is generally rather definite, or even sudden; more so than in enteric, though less than in relapsing fever. The patient is attacked with headache, and with pains in the back and limbs, as though these parts had been bruised. He feels chilly, or he may even shiver; after a little while he may perspire; but the chilliness presently returns and he is glad to sit huddled before the fire. He feels weary and disinclined for exertion. He is thirsty, but he has no appetite, and complains that his taste is perverted. His tongue is large, pale, and coated with fur, which is at first white, afterward yellowish. There is nausea, but not often vomiting. The bowels are constipated. The urine is scanty, dense, and high colored. He is restless, and his sleep is disturbed by painful dreams and sudden starts. Every day he becomes more prostrate; he totters in walking, his hands tremble when he attempts to use them; by the third or fourth day he gives in and takes to his bed.

From the first the expression is dull, heavy, and oppressed. The eyes are injected and suffused. The face is of a dusky or purple color, the flush being general, and not limited to the cheeks, as in enteric fever. As the disease advances, the countenance becomes more and more vacant and stupid-looking. It very seldom happens that the patient himself feels much concern about the issue of his illness. Toward the end of the week there is generally some delirium, especially at night and when he is half asleep. The advent of this symptom is generally earlier in persons belonging to the upper classes, in those who have been intemperate, and in those who are the subjects of mental anxiety and fatigue, than under opposite conditions. In some exceptional cases it sets in at the very commencement of the disease,

during the first night, and Murchison speaks of having seen cases which were at first mistaken for mania. But, as a rule, it is only after the lapse of several days that the patient's mind begins to wander, and that his intelligence and memory become confused and impaired. About the same time he generally ceases to complain of headache.

The *pulse* does not usually rise above 100 during the first two or three days; afterward it generally ranges between 100 and 120. When it is much higher the case is severe, if the patient is an adult; but in children a very rapid pulse, even at the commencement of the disease, is not an evil sign. Occasionally, however, the pulse remains below 100, or even below 90. Murchison cites instances observed by himself or by others, in which it was not above 40, or actually down to 28, for days together. Sometimes the heart's beats have been twice as frequent as the pulsations felt at the wrist. A very slow pulse is not generally a favorable sign.

The *temperature* rises rapidly in typhus. Wunderlich stated that it is generally 104°–104.9° on the first evening, and that by the fourth evening it is seldom under 104.9°, generally about 105.8°, and often higher still. But although Lebert has since observed 106.5° on the second evening, the tendency of all English clinical work with the thermometer has been to show that the average figures are very much lower than those given by Wunderlich. According to Murchison, the highest temperature attained at any period of the disease is generally about 104° or 105°; it is scarcely ever as high as 106°, except in children, and it may be below 103°. The maximum is usually observed between the fourth and seventh days, sometimes on the third day, sometimes not until the second week. During the latter part of the first week, when the maximum has once been reached, the thermometer varies little. Murchison remarks that a high range of temperature in the first week generally forebodes severe cerebral symptoms in the second. Lebert makes it a point that a rise in the evening is of comparatively little significance, when the thermometer falls each morning; what is really serious is a high temperature continuing without any intermission.

Typhus is attended with a characteristic eruption, the *mulberry rash*, as it was named by Sir William Jenner. But in rare cases this is preceded by a roseola, which is very different from it in appearance and may almost be mistaken for the eruption of scarlet fever. In the museum of Guy's Hospital we have models, illustrating this remarkable rose rash, which were taken from two women under the care of Dr. Wilks, in 1864. The parts represented are the abdomen, and the forearm with the hand; but in each instance the rash is said to have covered the patient. It was of a bright crimson color, punctated, macular, or diffused. One of the women was a nurse in the hospital, so that she was under observation from the time when she was first taken ill. The roseola in each case faded before the mulberry rash came out.*

The mulberry rash is generally first seen on the fourth or the fifth day of the fever; sometimes, as in a case admitted into Guy's Hospital, in 1874, it may be discovered as early as the third, or even the second day; Murchison says that it scarcely ever begins to appear after the sixth day. Sometimes it is altogether wanting; but observations made with great care at the London Fever Hospital in 1864, showed that this occurred in scarcely more than 2½ per cent.—among nearly 2500 cases of all ages. In children, however, absence of the eruption is much more frequent. But, on the other hand, when it does come out in them, it is often peculiarly abundant and well

* Dr. John Harley speaks of having once seen a very copious eruption of *rose papules* upon the chest and abdomen of a powerful, fair complexioned man who had severe typhus; they preceded the mulberry rash, and disappeared when it became petechial ("Reynolds' System of Medicine," vol. i, 584).

marked, so as to resemble the rash of measles; and in such cases it may be seen on the face, whereas in adults it is generally confined to the trunk and limbs. One usually looks for the typhus rash on the chest and the abdomen; but Dr. Buchanan, in "Reynolds' System of Medicine," says that the earliest traces of it are to be found in the back of the wrists, the borders of the axillæ, and the epigastrium. It consists of more or less numerous spots, of indeterminate form, the largest three or four lines in diameter, isolated or irregularly confluent. They may first be slightly raised, so that one can feel them with the finger; and as they may then be of a somewhat florid color, it is sometimes difficult, when they are few and separate, to distinguish them from the rose spots of enterica. At this time, too, they disappear on pressure. But in the course of a day or two they alter in appearance; their hue becomes darker and more dingy; they are no longer raised; and when the finger is pressed upon them they either remain unaltered or display a yellowish tint. This change is due to the escape of blood from the vessels into the substance of the cutis; in other words, the maculæ have become petechial, according to the modern definition of the word *petechia*, as a "minute ecchymosis," which we owe to Willan and his successors. They remain visible in the dead body if the case should end fatally while they are still present. In many instances they are, from the very first, of a livid or purple color, and do not fade on pressure.

Within forty-eight hours from its first appearance the mulberry rash is complete. During this time fresh spots may come out, but they are added to the old ones. There is not, as in enteric fever, a succession of crops of maculæ, one set appearing while another is fading away. With regard to this point, Murchison says that he satisfied himself by observations upon a large number of cases, surrounding every spot with a circle of ink, so as to identify it. In addition to the distinct maculæ, there is also a faint, irregular, dusky red mottling, which one would imagine to be more deeply seated, and which, therefore, has been commonly known as a "*subcuticular rash*"—an unfortunate expression, since it seems to imply that the seat of the maculæ is superficial to the cutis. After the first day or two, no increase of the eruption occurs. It is important to notice that the severity of a case of typhus is generally directly proportionate to the amount of eruption, its depth of color, and the rapidity with which it becomes livid or petechial. Murchison adds that exceptional cases in which no rash appears have a generally mild course; but, according to Lebert, they are often severe and even fatal.

Second Week.—This period is marked by a gradual aggravation of all the more obvious symptoms. The patient is now absolutely sleepless, and he usually passes into a state of continuous *delirium*. Sometimes he is noisy and violent, shouting, talking incessantly, singing, struggling to get out of bed, or fighting with his attendants. When spoken to, he generally becomes still more excited. He may throw himself out of the window, or cut his throat. Some years ago a man was brought into hospital, and was actually admitted into a surgical ward, for a suicidal wound of the neck inflicted in the course of typhus. After two or three days the delirium becomes quiet and muttering; and in the majority of cases it is so from the first. Sometimes excitement comes on as night approaches, while the rest of the day is passed in a state of stupor and prostration. But by the middle of the week there is generally complete unconsciousness. When loudly spoken to, the patient perhaps opens his eyes and stares vacantly forward; if told to put out his tongue, he may separate his jaws, and leave them gaping until they are closed for him. No other signs of intelligence can be elicited; yet Dr. Murchison, who himself went through two attacks of typhus, tells us that the imagination is far from inactive. He

took a great dislike to a nurse and to a valued friend; and because they once tied him down in bed, he kept fancying that they intended to murder him, that they were shutting him up in a dungeon, that they followed him to India, Burmah, and other countries which he had really visited in former years, and to which he now supposed himself to have escaped. Dr. Gueneau de Mussey, who caught typhus in Dublin in 1843, afterward remembered imagining that he was tied down in bed, and gradually consumed by spontaneous combustion, while women were taking water from a pond and pouring it over him. Another fancy was that he saw the front of a particular house in Paris in a state of phosphorescence, and a child suspended by the neck from a window. He also believed that he saw one of his friends killed in the street; and so strong was the impression, that during convalescence he still maintained that this friend was dead, and felt great concern for his loss.

During the second week of typhus *deafness* is very commonly present; indeed, it begins about the fifth day. It sometimes affects one ear, sometimes both. Its cause is not yet ascertained. Stokes thought that it was due to softening of the muscles of the ossicula; but to this view Murchison objects that the hearing is too soon recovered during the convalescence. For, contrary to what might perhaps have been anticipated, many cases do well in which deafness has been complete; and from the time of Fracastorius there has been a tradition that it is actually a favorable sign. For this belief, however, there do not appear to be any good grounds; it is certainly present in many cases which end fatally.

The *pupils*, in the advanced stages of typhus, are generally contracted and insensible to light. They may be as minute as pinholes, according to a comparison made by Graves. Jenner first laid stress on the state of the iris as affording a distinction between typhus and enteric fever; and Murchison says that, neither during active delirium nor in profound stupor, has he seen dilated insensible pupils in typhus. Sometimes, however, the supervention of coma is associated with dilatation of the pupils.

The muscular *prostration*, during the second week of typhus, becomes extreme. The patient sinks down in bed, and lies on his back, unable to raise himself, or even to turn on either side. Students at Guy's Hospital twenty years ago all knew one of the favorite remarks of the late experienced apothecary, Mr. Stocker, that to find a fever patient lying on his side was a good sign. The fæces are passed involuntarily. The urine dribbles away incessantly, so that unless it can be caught in a proper receptacle it soaks the sheets and produces great irritation of the skin. But before it begins to run off it may fully distend the bladder, the muscular coat of this organ being paralyzed, as well as the sphincter. One must, therefore, never omit to examine the hypogastric region at every visit, even if the nurse should say that the urine is passed in abundance. Other results of muscular weakness are loss of power to speak, to protrude the tongue, and sometimes even to close the eyelids, as the result of which the cornea may slough, and allow the contents of the aqueous chamber to escape.

At this stage of the disease *tremor of the muscles* is almost constantly present. The whole body may be in a state of agitation, especially in old people and in those who have been exhausted in brain work or by intemperance; indeed, in drunkards a state of delirium tremens often seems to be superadded to the symptoms of the fever. Sometimes the tremor is limited to the hands and tongue. Murchison speaks of having sometimes seen nystagmus and even choreiform spasms of the muscles of the limbs. Much more frequent are those jerking movements of the muscles of the forearms which are commonly called *subsultus tendinum*. Or there may

be twitchings of the face, one corner of the mouth being, perhaps, drawn up from time to time. Jenner saw two cases in which the face acquired a peculiar expression from a spasmodic action of the inferior recti muscles of the eyeballs in association with the levatores palpebrarum. In each instance the movements were excited when either of the arms was suddenly raised. Another variety of spasm is picking or fumbling at the bedclothes, for which we have the pedantic names of "floccitatio" and "carphology." In more rare cases the fingers are forcibly clenched, or the legs and thighs are bent so that the knees almost touch the chin, or there may even be trismus or opisthotonos. It has not yet been ascertained whether these last symptoms are dependent upon the presence of meningitis, which sometimes occurs as a complication of typhus.

The mulberry *rash* generally remains visible throughout the whole of this period. The maculæ may even become darker and more distinct, and may not disappear until the disease has already subsided. But the faint general mottling vanishes after a few days, and in mild cases, where this is the only eruption, nothing of it may be left at a time when the fever has still several days to run.

The *temperature* during the second week is, as a rule, rather lower than in the latter part of the first week. Sometimes, indeed, the maximum is not reached until between the seventh and the tenth day. But during this period there is generally a slight fall, to which Murchison attached importance, believing that the cases in which it was absent were severe. Henceforth, and up to the time of the crisis, there are usually slight recurring remissions of one or two degrees. A decided rise at this period indicates the supervention of some complication, generally pneumonia.

The rate of the *pulse* varies little from day to day, but its general tendency is to become more rapid. Its volume and force decline until in severe cases it is exceedingly feeble or even imperceptible. Sometimes it is dicrotic or undulatory, but Lebert remarks that this is remarkably seldom the case, whereas it is almost the rule in enteric fever. Irregularity of the pulse is not uncommon, and sometimes there are intermissions. Graves laid stress on the fact that a change from the recumbent posture to the erect or semi-erect is attended with an acceleration of the pulse, the amount of which is greater in proportion to the weakness of the patient. His successor, Professor Stokes, pointed out a still more important indication of the progressive weakness which constitutes one of the chief dangers of the disease, namely, diminution or loss of the heart's impulse with disappearance of the first sound. For several days, even when recovery is to take place, it is often impossible to feel the heart beating, and with the stethoscope only the second sound can be heard. In other cases a sytolic bruit is developed, which no doubt belongs to the class of functional murmurs.

The *breathing* at this stage of the disease is almost always hurried, being at the rate of thirty or forty in the minute. Moreover, according to Murchison, hypostatic congestion of the lungs should be regarded as a constant symptom rather than as a mere complication. It is, at any rate, present in all severe cases, and after death is scarcely ever wanting, whatever may have been the cause of the fatal issue. It commonly begins about the middle, but sometimes at the commencement, of the second week. It is indicated by diminution of percussion resonance over the bases behind, where the respiratory murmur is feeble, and by the presence of coarse râles, which may gradually spread over the greater part of the chest on both sides. The affected parts of the lungs are found, *post-mortem*, to be bulky, of a dark red or purple color, and soft; serous fluid oozes abundantly from their cut surface, and from the tubes. Indeed, bronchial catarrh

is always associated with this condition, and the patient may spit up considerable quantities of frothy, tenacious secretion, mixed, perhaps, with streaks of blood. But sometimes cough and expectoration are entirely absent.

The *tongue*, in very mild cases, may remain moist and furred throughout the whole course of the fever. But usually during the second week it is dry, rough, and brown. When the disease is very severe it contracts into a ball, and becomes covered with a dark brown or black crust; this crust may be irregularly cracked; but the fissured condition of the tongue itself, which is so commonly seen in enteric fever, is rare in typhus. The lips and teeth are covered with a dirty, blackish material, commonly spoken of as *sordes*, which consists of "an accumulation of epithelial debris, which becomes black from desiccation, or perhaps from an admixture of blood." An entire loss of appetite lasts to the end of the disease; and sometimes the patient refuses to take the nourishment which is given to him. He generally does not at this stage appear to suffer from thirst. Murchison met with some instances in which vomiting was a persistent and troublesome symptom, but such an occurrence is exceptional, and such patients have been for the most part previously dyspeptic. Neither he nor Sir William Jenner before him found the abdomen unnaturally large in the majority of cases; sometimes it was flat or even concave; distention amounting to meteorism was very rare. As a rule, there is *constipation*, but the opposite state of the bowels is not infrequent. The fæces may be of normal consistence, and even if fluid, they are generally dark in color. Murchison speaks of diarrhœa as occurring in about 5 or 10 per cent. of all cases of typhus. But in 1866 Dr. Buchanan said that it had been seen in at least one-third of the cases which had come under observation at the London Fever Hospital during some years previously; he was inclined to refer its greater frequency in the experience of some physicians, to the larger amounts of liquid food forced upon the stomachs of their patients, although he did not consider this more than a slight drawback to the practice in question. Murchison remarks that when an aperient has been required the bowels often afterwards remain open or even relaxed.

The *urine* generally remains dark in color during the second week of typhus but sometimes it is pale, and even alkaline. With regard to the amount of urea which is voided, I have nothing to add to what has already been stated in the chapter on fever. It is an interesting fact that at this period of the disease the *chlorides* fail to be excreted by the kidneys, even when the patient is made to take large quantities of common salt. The urine sometimes contains no chlorides at all, but Dr. Buchanan says that usually two or three grains are passed in the twenty-four hours, just enough to produce an opalescence on the addition of nitrate of silver. The not infrequent presence of *albumen* in the urine will be referred to presently.

Crisis.—Such is the condition of a patient suffering under typhus until about the fourteenth day, when, if he lives so long, a marvelous change takes place, constituting the crisis. Formerly what was most often noticed was that he fell into a sound and quiet sleep, from which he awoke a few hours later, rational, refreshed and cool. But at the present time the use of the thermometer enables us to watch the process much more closely. In some exceptional cases there is a rise of perhaps two degrees the day before the crisis occurs. Much more often the temperature becomes slightly lower during the same period. The crisis itself is marked by a rapid fall to normal or even slightly lower still; a difference generally of from 4° to 6°. The fall may be complete within twelve hours, but according to Lebert it much more often takes thirty-six hours and sometimes two or three days. Dr. Murchison says that in nearly one-half of the cases of typhus the date of the crisis is the

thirteenth or the fourteenth day; in more than three-fourths of them it is from the thirteenth to the sixteenth. Lebert gives a much larger proportion of cases as subsiding between the tenth and the twelfth days, some even between the sixth and the ninth days.

The existence of complications may sometimes mask the crisis, or even prevent its occurrence.

It is sometimes accompanied by perspiration, or by diarrhœa, or by the excretion of urine depositing lithates in abundance; but there is no reason to suppose that such phenomena are essential to the defervescence, as was formerly supposed. Afterwards the temperature generally remains normal, except that for a night or two there may be a slight evening rise.

Convalescence advances rapidly. The tongue at once becomes clean and moist, and the appetite is ravenous. Within three or four weeks the patient often completely recovers his bodily strength and is able to return to work.

A true *relapse* of typhus is exceedingly rare. An instance of it was recorded in 1869, by Ebstein, the interval being twenty-five days. At the London Fever Hospital no such case occurred during twenty-three years after 1855. In that year Dr. Buchanan had a nurse under his care, who, after recovering from an attack of the disease, was taken ill a week later, and went through it a second time; on each occasion there was a mulberry rash, and the fever appears to have lasted a fortnight.

Exitus Lethalis.—Many cases of typhus do not run on long enough to exhibit a crisis. The mortality from the disease at the London Fever Hospital has been, at the lowest, 15 per cent. Murchison estimates that in the metropolis generally, if slight cases and those occurring in children be taken into account, it is about 10 per cent. It differs, however, slightly in different years, and it is said to be generally higher during an epidemic than at other times. Among soldiers in camp, or in a besieged city, the death-rate has sometimes been enormous. In the Crimea one-half of the French troops who were attacked are said to have perished; and still more terrible instances are on record. In certain epidemics death has sometimes occurred on the second or the third day, or even after only a few hours' illness. Such cases have been described as examples of a special variety of the disease—*typhus siderans*. Murchison speaks of having seen several cases end fatally on the sixth or eighth day, usually from pulmonary congestion. But, as a rule, typhus does not destroy the patient until toward the end of the second week. The exact mode of death varies. Sometimes it is by "typhoid" symptoms and coma; sometimes it is through the lungs by asphyxia; sometimes it is by failure of the heart, the pulse being imperceptible, and the skin cold, livid, and bathed in a profuse sweat. In some of the last-mentioned cases the patient lies for a day, or longer, in a peculiar state, to which Jenner has appropriated the name *coma vigil*. He has his eyes wide open, so that he might be supposed to be awake, but he is absolutely insensible to all that goes on about him, and his face is devoid of expression. Sometimes death is preceded by a rapid elevation of temperature, as in a case at Guy's Hospital, in 1873, in which the thermometer registered 108·7°.

Morbid Anatomy.—Most of the appearances observed in post-mortem examination of the bodies of those who have died from typhus, instead of being characteristic of it, are rather effects of the febrile state through which the patient has passed, such as are found equally in cases of erysipelas, pyæmia, and other acute diseases. The *rigor mortis* is brief and incomplete, putrefaction takes place early, there is much cadaveric discoloration of the skin, the blood is fluid and dark colored, and it stains the lining membrane of the heart and of the great vessels. There is often but little emaciation, yet the muscles are soft and friable, and where death

has occurred at a late period of the fever their fibres are granular or waxy-looking under the microscope. A similar change is found in the substance of the heart, and probably often plays an important part in bringing about the fatal issue. The spleen is but little enlarged, weighing, on an average, seven ounces, though sometimes as much as fourteen ounces; but its tissue is very soft, and may even be reduced to a mere pulp, which escapes when the capsule is divided. Jacquot is said to have observed a case in which instant death resulted from rupture of that organ. The liver is soft and hyperæmic; in an advanced stage of the disease it is often fatty. The state of the lungs has already been noticed. There is, lastly, an entire absence of those intestinal lesions which belong to enteric fever.

Other changes which have been noted are probably altogether independent of the disease. They are common either at a certain period of life, or in dissipated and intemperate persons, such as are apt to succumb to typhus. Thus the brain has often been found atrophied, with an excess of fluid in its ventricles and upon its surface.

In other instances the kidneys have been wasted or in an advanced stage of Bright's disease. In such cases death is apt to be ushered in by convulsions, the occurrence of which in typhus should always lead at once to an examination of the urine; it may then be found to be albuminous, and may contain blood, more or less altered, and casts. Sometimes the renal affec tion appears to be quite early and recent, and probably is itself secondary to the fever. Murchison speaks of having found the kidneys weigh nineteen, twenty, or even twenty-three and a half ounces. It is to be remembered, however, that albumen may be detected in the urine of a large proportion of cases of typhus in which no cerebral symptoms occur, and which do well. It is usually in small quantities, and it is often present during only a day or two days toward the end of the disease. And even when convulsions set in, an absolutely unfavorable prognosis is by no means to be given. Recovery took place in twelve among one hundred and thirty-two cases in which this complication occurred at the London Fever Hospital, between 1862 and 1869. In very rare instances, where a fatal issue has occurred, blood has been found effused upon the surface of the brain; but this may have been the result of the convulsions rather than their cause. It is toward the end of the second week that convulsions are most often observed.

Complications and *sequelæ* of typhus are not numerous. Jaundice is exceedingly infrequent. Murchison met with only fifteen cases in which it was present. In one of them he considered that the liver was in a state of acute yellow atrophy; and in another leucine and tyrosine were found in the viscera as well as in the urine. One well-marked instance of jaundice occurred at Guy's Hospital in 1869. The patient, a man aged forty-nine, died on the fourteenth day of the fever.

Pneumonia is not very uncommon. Sometimes, when the patient appears to die of the severity of the disease, instead of there being merely hypostatic congestion of the dependent parts of the lungs, a considerable part of one of them is found to be hepatized. Either the lower, the middle, or the upper lobe may be affected in this way. In other cases inflammation of the lung sets in definitely a few days after the crisis. Of about forty consecutive fatal cases examined at Guy's Hospital, no fewer than six had well-marked pneumonia. In one instance an attack of pleurisy occurred a week after the subsidence of the fever. The left side of the chest was found to contain several pints of fluid with flakes of lymph. Murchison has several times seen gangrene of the lung after typhus.

In some rare cases acute meningitis occurs as a complication. In

addition to the cerebral symptoms which commonly accompany the fever, there is then retraction of the head, strabismus, and dilatation of the pupils. Mania sometimes sets in during convalescence, and may even compel the removal of the patient to an asylum. In the long run it seems always to be recovered from. Hemiplegia, with or without aphasia, is an occasional sequela, but in all likelihood it depends upon embolism of one of the cerebral arteries by a fragment of thrombus derived from the left auricle or ventricle of the heart; for clotting of blood in the recesses of these cavities is doubtless of frequent occurrence during the height of the disease. Murchison, however, once saw endocarditis in a case of typhus, there being large vegetations, which gave rise to infarctions of the spleen. Embolism of the arteries of the limbs is doubtless the cause of gangrene of the feet, which has now and then been known to occur after typhus. Bed sores ought hardly to arise in this disease, since the stage of coma is of comparatively short duration. Their presence sometimes accounts for the supervention of pyæmia, but in other cases blood-poisoning sets in during convalescence, without any such obvious explanation. Thrombosis of the femoral veins is not often seen after typhus. Inflammatory swellings, which have been called *buboes*, and compared with those that occur in the plague, occasionally form in the parotid and submaxillary regions, and go on rapidly to suppuration.

When a pregnant woman is attacked with typhus she not infrequently passes through the disease without interruption to the process of gestation, but sometimes a miscarriage occurs between the tenth and the fourteenth days. As a rule she afterwards does well, and the child, if not too immature, is generally saved.

Ætiology.—The most conspicuous fact in the ætiology of typhus is its contagiousness. That it passes directly to other persons from the sick is established by the clearest possible evidence. Instances of such an occurrence are commonly observed in every hospital into which cases of typhus are admitted; if into general wards, those who are already patients with all kinds of other complaints are attacked; if into special wards, the medical attendants and nurses fall frequent victims. When cases are left in private houses or lodgings, the disease passes not only to relatives and to other inhabitants of the same dwelling, but also to doctors and clergymen, whose visits are only occasional. On the other hand, the removal of a single case of typhus from the building in which it arose is often effectual in preventing any other occupants from taking it.

Lastly, the diffusion of the disease can often be traced from point to point in a town or in a district. Thus Alison relates how the son of a shoemaker in Edinburgh lay ill with typhus in a room in which his father and two apprentices were at work. Afterward both of the apprentices were attacked in their own homes, widely apart from one another, and at considerable distances from the workshop; and there speedily followed a succession of cases in the other inhabitants of these two houses, seven persons being affected in one, and twelve in the other.

An epidemic which occurred at Carlisle, in 1781, was found by Dr. Heysham to have started from a particular house in Richard Gate; one of the persons affected there was a weaver, who on his recovery communicated the disease to his fellow-weavers in a large workshop, and by them it was spread all over the town. In 1847 immigrants introduced typhus into America, and in 1867 into Australia.

With regard to the nature of the contagious principle of typhus, we are as yet altogether ignorant. There can be no doubt that it multiplies in the blood, but hitherto no observer, even with the highest microscopic

powers, has succeeded in detecting any special organisms there. It is probably exhaled both by the skin and the lungs, and it may perhaps be identical with the cause of the offensive odor which is so perceptible in the close proximity of almost all severe cases. This odor has been compared with the smell of rotten straw, or with that of mice. Murchison regarded it as *sui generis;* he says that he has known nurses in the London Fever Hospital distinguish typhus from other fevers by it alone. It is given off chiefly during the second week, and there is some reason to believe that infection is scarcely to be feared during the first week. The late Dr. Perry, of Glasgow, maintained that the disease was not contagious before the ninth day, or perhaps not until a later period still. He found that the Glasgow Fever Hospital patients who happened to have been sent in for erysipelas, pneumonia, bronchitis, or other inflammatory affections, escaped typhus so long as they remained in the wards for acute cases, but caught it when they were transferred into a convalescent ward; and he actually adopted the plan of keeping such patients in the fever wards until they were sufficiently well to go to their homes, with the result that during several months none of them were attacked. Murchison, however, although he adopts the view that typhus is most contagious after the end of the first week, thinks that the spreading of the disease in convalescent wards is due to the patients coming more closely into contact there, but especially to their wearing their own clothes, saturated with the fever-poison before admission, a view which suggested the importance of having such clothes thoroughly disinfected before they are again put on. Murchison thinks that the human body soon ceases to give off the poison after the subsidence of the fever.

It is, at any rate, certain that clothes and bedding may become vehicles for the transmission of typhus. Murchison says that laundry women are especially liable to contract the disease without direct communication with the sick. Barker and Cheyne, in their account of one of the first epidemics, relate that a child discharged from a fever hospital took to another institution a bundle of clothes which had not been disinfected; a woman who opened it perceived an exceedingly disagreeable odor, and in a few minutes became ill with what proved to be the beginning of the fever, of which there had been no previous case there. Haller, of Vienna, thought that dark colored materials were more apt to absorb the poison than light colored ones; and I well remember I used to notice, when demonstrator of anatomy, that the dissecting-room smell adhered to me more strongly when I wore dark clothes. Very few positive instances seem to have been recorded of the communication of typhus by contact with the bodies of those who have died of it; but Murchison, when he was attacked in Edinburgh, had been dissecting in a close room, in which there were many such bodies, and he had never entered the wards of the infirmary, nor seen a case of the disease. On the other hand, at St. Bartholomew's Hospital, in 1838-9, the dissecting room received seventeen bodies dead of typhus; but among six students of the hospital who alone took it, four had not dissected at all, and the other two, who had dissected, had been also exposed to contagion in the wards.

It has but seldom been noticed that the fever has been conveyed by third persons, not themselves affected with it. But Murchison relates that in January, 1867, a patient in a surgical ward at the Middlesex Hospital was attacked after she had been there for four and a half months; she had been receiving daily visits from a nurse who was in close atttendance on a patient with typhus down stairs. There have been some instances in which the disease has been supposed to have been brought into towns by bodies of men, themselves free from it. Thus, in 1861, great interest

was excited by the case of an Egyptian vessel, the "Scheah Gehaad," from which typhus was introduced into Liverpool, when thirty-one persons caught it. But, although Dr. Duncan, on the testimony of the surgeons who attended the men on board this ship, thought that they suffered from no other disease than dysentery ("*Trans. Epid. Soc.*," 1861), Dr. Parkes came to the conclusion that typhus had really been prevalent among them before they arrived in port ("*Army Med. Rep.*," vol. ii). It does not seem to me that much importance is due to the oft-quoted statement of Fodéré that the French army, during the retreat from Italy, in 1799, communicated fever to the inhabitants of fifteen towns and villages on its route, but was not itself attacked until it arrived at its destination.

On the whole, then, it appears that typhus is not nearly so apt as the contagious exanthemata, to be propagated by means of inanimate objects, or of human beings themselves unaffected by it. And another very important point is, that its poison is very easily rendered inert by free dilution with air. Some writers have stated that there is greater risk of the disease spreading from the lower to the upper stories of a house or of a hospital than in the reverse direction; and this, if true, would show that the upward currents of air that exist in all inhabited buildings are capable of carrying the infection with them. But experience has abundantly proved that it never passes undestroyed from one house to another, through the atmosphere. When the London Fever Hospital was one of a row of houses in Gray's Inn Lane, no case of typhus arose in the others; and afterward, when it occupied its second site at King's Cross, on the same plot of ground as the Smallpox Hospital, and but a few yards off, Dr. Tweedie was able to state that during eight years not one of the officials of the latter institution contracted typhus. So, again, Murchison says, that if a patient with this disease is placed in a large, well-ventilated apartment, the attendants incur little risk, and the other residents in the same house, none whatever.

The facts stated in the last two paragraphs form essential links in an argument by which Murchison endeavored to establish his conviction that typhus, instead of being always due to contagion from a previous case, is often generated *de novo* in persons placed under defective sanitary conditions, of which overcrowding is the chief. For it is true of all other acute specific diseases, as well as of this one, that a great many instances occur of which the origin cannot be traced; but the more perishable the poison, the less likely is it to be carried over long distances, and transferred from one vehicle to another, as we know to happen in the case of the contagious principles of scarlet fever, diphtheria, etc. And a further link of great importance is that where typhus is seen, independently of obvious contagion, it is almost always among the poor and destitute. One scarcely ever finds it, like the exanthemata, springing up in an unexplained way in the houses of the rich, or even of the well-to-do. The only instances to the contrary that I remember to have heard of is that mentioned by Dr. Buchanan, of two boys, living at an institution, with every accommodation, who were attacked within a few hours of one another, but who could not, after the most careful inquiries, be shown to have been in the way of catching the disease. No doubt there had been a more or less direct communication of the poison to both of them together, for the simultaneous development of the fever in them is unintelligible on any other view. The significance of the rarity of such cases is augmented by the fact that those who belong to the upper and middle classes are by far the most exposed to chance infection, because they move about so much more than the poor, come into contact with so many more people, and receive so many more letters and parcels through their hands, of which they know nothing.

To demonstrate positively the *de novo* origin of a contagious disease, is,

from the nature of the case, almost an impossibility. Perhaps, the most favorable opportunity that has ever occurred for investigating the point was afforded by the Egyptian ship, the "Scheah-Gehaad," already referred to. She left Alexandria, in November, 1860, and, after calling at Malta, arrived at Liverpool, on February 16th, 1861. But, according to Dr. Parkes, it was not proved that some of the men did not bring the poison with them when they embarked at Alexandria; they numbered 476, and included not only Arabs, but also Nubians and Abyssinians. Of the supposed instances of the spontaneous generation of typhus collected by Dr. Murchison, the most important, in consequence of the care and pains which he devoted to its investigation, is perhaps, the group of seven cases which arose in Meridian Place, Bermondsey, in March, 1859, at a time when the disease was at least very uncommon in the metropolis; for during ten and a half months previously, only two examples of it had been seen in the London Fever Hospital. But the real force possessed by this and several other similar observations lies in the fact that one single condition was traceable in every one of them, namely, that those who were attacked by the disease had been crowded and huddled together in narrow and ill-ventilated dwellings or sleeping places. They were often also destitute and famished; but that this is not so essential to the development of the disease as overcrowding, seems to be shown by the circumstance that at Dundee, in 1865, an epidemic of typhus was brought about by the inhabitants of the surrounding country flocking into the town, in consequence of work being uncommonly abundant and wages good. The occurrence of typhus in an epidemic form has, indeed, been predicted when, as in 1826 and in 1862, destitution and famine have been very prevalent; and such predictions have been verified by the result. It would be impossible to find a better illustration of the conditions under which, if at all, typhus is generated, than was afforded by the "Scheah-Gehaad;" the men on board (who were being carried to Liverpool in order to navigate back to Egypt another vessel, a man-of-war, then in that port) seem to have been crowded together in the most shocking manner; some of them were in a state of starvation; the filth and stench between decks were abominable; and, to crown all, the hatches had been battened down, on account of bad weather, and vomited matters and fæces had been discharged in every part of the ship.

No less an authority than Virchow, has endorsed the opinion that the poison of typhus can be brought into existence by the concurrence of such conditions as these; and the same view has been maintained by the late Dr. Hudson, of Dublin. But, after all, it is possible to bring the facts adduced by Murchison into harmony with the opposite doctrine, namely, that the disease, as it occurs at the present day, is invariably due to contagion from a previous case. The foundation of this reconciliation is the well-ascertained circumstance that debility and impairment of health augment the susceptibility of an individual to the typhus poison. It would seem, indeed, that every one is originally capable of taking this disease, although the fact of having once passed through it affords an almost absolute subsequent immunity. At the London and Glasgow Fever Hospitals, all the nurses who are not thus protected have been found to contract typhus within three or four months after entering upon their duties. In 1833, Dr. Tweedie stated that, with one exception, every physician who had been connected with the London Fever Hospital had been attacked with fever. But in the case of the higher officers of such an institution, there is often a much longer interval between the commencement of their exposure to contagion and the date at which they succumb to it. Thus, in 1862–3, one of the resident medical officers at the hospital in question attended a large uumber of cases for more than twelve months before he took the disease, and more

recently a person whose duties were daily in the wards and included cleansing of the dirty linen, died of typhus contracted for the first time after fifteen years' service. The opposite extreme occurred in the case of a medical man who caught it from a single visit to the London Fever Hospital.

In these cases it does not appear whether there was any special reason why the contagion should have proved effective at one time rather than another. But it is often possible to trace accidental conditions which favor its operation; one such condition is alcoholic intoxication. Murchison says he has known several instances of persons exposed for months to the poison of typhus in its most concentrated form, who were not attacked until immediately after a debauch. Habitual intemperance probably acts in a similar way; it was once noted that more than one-half of the patients admitted for this disease into the Edinburgh and Glasgow Infirmaries had led intemperate lives. Other favoring circumstances are excessive bodily fatigue, want of sleep, and overwork of the brain. Murchison, in support of the popular doctrine that a dread of typhus increases the risk of taking it, cites the case of an Edinburgh medical student who so feared it that he could hardly be induced to enter a ward in which there were any cases; he was one of the first students to be attacked during the epidemic of 1847. Again, the debility which accompanies convalescence from other complaints predisposes to typhus, and in armies it has been frequently observed to follow scurvy. Murchison altogether rejects the improbable notion, which has, however, found supporters, that typhus is less likely to occur in phthisical persons than in others.

Tweedie long ago made the remark that butchers appeared to be comparatively exempt from the liability to typhus, and Murchison suggests that the reason for it is their eating so much nourishing food. Most of the butchers admitted into the London Fever Hospital for the disease have, he says, been out of employment and destitute. Poverty and inanition are, indeed, among the most important predisposing causes. No less than 95.76 per cent. of all the typhus cases observed at the Fever Hospital during twenty-three years, more than 18,000 in number, had been inmates of hospitals or dependent on parochial relief, and many of them had been on the verge of starvation for several weeks or months. Epidemics in Ireland, in England, and on the Continent have repeatedly been associated with the failure of crops and with the widespread destitution consequent; so that although relapsing fever has of late been declared to be the true "famine fever," that name was formerly often given to typhus, and not without warrant.

Another very important predisposing cause of this disease is overcrowding. Beside its obvious influence in increasing the spread of typhus by contagion, there seems to be no doubt that it also augments the susceptibility of individuals to the poison. Thus it is quite possible that it may favor the occurrence of a first case among the occupants of a lodging-house or cellar, even though it should not be capable of generating typhus directly, as Murchison supposed.

Other so-called predisposing causes probably act only by facilitating contagion. Thus epidemics occur rather in the winter than in the summer, because the poor, for the sake of warmth, block up every hole by which air can enter their wretched dwellings during the cold season. But in some years it has happened that more cases have occurred in July, or in September, than in January. So far as is known, the variations of temperature which occur in temperate climates have no direct influence upon the prevalence of the disease. But it is as yet doubtful whether this form of fever has ever been observed in Africa, or in the tropical parts of America, although it is certainly met with in the gaols of India. If very hot countries should prove

to be exempt, it will not be possible to attribute the fact to any idiosyncrasy of the inhabitants, like that of negroes in respect of the ague poison. For Murchison says that several Africans and East Indians have been admitted into the Fever Hospital, and that the rash was characteristic.

It is worthy of notice that patients sometimes themselves attribute the disease to their having "caught cold" or got wet. Murchison says that this has been the case with a considerable proportion of the persons admitted into the London Fever Hospital. In 1856 a young man was taken into Guy's Hospital who gave as the history of his illness that he went for a long walk on damp ground, and felt that he took cold; two days later he had headache and fever, and the typhus eruption followed in due course. In that instance the poison had no doubt already been received, and the relation between the chill and the typhus was one of mere coincidence. But it is quite possible that long continued exposure to cold and wet may render a person more susceptible than he had been before.

Typhus may occur in persons at all ages, from one month to eighty-four years old. The quinquennial period at which the disease is most common is from fifteen to twenty; one-half of the cases admitted into the London Fever Hospital have been in patients from ten to thirty. The proportion of males to females among typhus patients scarcely differs from that in the population generally.

I have already remarked that typhus protects against the subsequent action of its own contagion. A second attack of this disease is indeed as rare as one of smallpox; far more so than a second attack of measles or scarlet fever. It does, however, sometimes occur, and there was a notable instance of it in the case of Murchison himself, the interval between the attacks being ten years. That writer knew of two other physicians who contracted this form of fever twice, and in one of them it proved fatal on the second occasion. A very remarkable fact, for which he vouches and of which he saw at least six examples, is that, during an epidemic, a person exposed to the poison may have what appears to be an abortive typhus fever, malaise, dry tongue, and even slight delirium, but no distinct rash; may recover, sometimes after exactly fourteen days, and then a few weeks later may go through a regular attack, attended with the characteristic eruption. Such a "*typhisation à petite dose*" in fact, seems to afford no protection whatever.

There appears to be no evidence whatever that typhus is related to any of the contagious fevers to which the lower animals are subject, although it was at one time imagined that the "cattle plague" might be an equivalent disease. Mosler injected fresh blood from patients with typhus into the veins of dogs, without any result. Zülzer, however, has since been more successful; his injections were made upon rabbits, and he found that when the blood was taken while the disease was at its height those animals died in two or three days, but not if the crisis had been passed.

Diagnosis.—This is seldom, if ever, doubtful, when one has an opportunity of watching a case throughout its whole course; but there may be great difficulty in forming an opinion upon a single visit, or if the patient is brought to a hospital a day or two before his death. At the commencement of the disease, what suggests the correct view of its nature is generally the fact that there has been exposure to the specific contagion, or at least that cases have already occurred in the neighborhood. Otherwise it could scarcely be distinguished from smallpox and the other exanthemata, and not at all from enteric or relapsing fever, some cases of which begin exactly in the same way. As a rule, all uncertainty is removed by the development of the eruption, but sometimes, and especially in children, it is by

no means easy to say whether this may not be measles rather than typhus. Lebert makes a point of the harassing cough which belongs to the former complaint, whereas the bronchitis of fever seldom causes the patient much inconvenience. Certain cases which are commonly classed under purpura might also be sometimes mistaken for typhus; they are attended with eruptions which rapidly become petechial, and with much febrile disturbance; and they no doubt depend upon blood poisoning, the exact nature of which, however, is not yet understood. Lastly, the copaiba rash has been mistaken for typhus, as in an instance alluded to by Dr. Hudson, of Dublin.

But it sometimes happens that the mulberry eruption is not so distinct as to be conclusive, or it may even be altogether absent. An error which it is very important to avoid is that of taking for cases of this kind those in which the disease is really an acute pneumonia or an acute meningitis.

As regards pneumonia, although the mistake was formerly very common, and although Murchison says he has seen many instances sent to the London Fever Hospital for typhus, I believe that the existence of this disease, when its symptoms are such as could be confounded with those of a specific fever, can always be discovered by auscultation, which no careful physician would omit before giving a diagnosis of typhus in a patient without eruption. But it may still be a question whether the pulmonary inflammation is or is not a primary disease. Murchison says that those cases of pneumonia in which the apex of the lung is first affected are particularly apt to be unattended with local symptoms, and to assume the mask of a "typhoid" condition. He remarks that if the temperature should reach or exceed 104° after the fourth day of illness the pneumonia is probably secondary.

The distinction between meningitis and typhus is one of far greater difficulty. Among the symptoms which point to the former disease are an anxious and pained, or wild and defiant expression of face, intensity of headache, the concurrence of headache with delirium (for in fever pain almost always ceases before delirium sets in), the presence of convulsions early in the case, and the repeated occurrence of vomiting.

Several further questions arise in the diagnosis of cases which come under observation at a time when the patient is comatose and with "typhoid" symptoms already developed. Pyæmia may be mistaken for typhus under such circumstances, or if there be erysipelas, jaundice, or delirium tremens, one may be unable to say whether these diseases are complications of fever or altogether independent of it. In tropical climates there may be great difficulty in distinguishing severe forms of malarial fever from typhus. And in any part of the world it may be altogether impossible to tell typhus from enteric fever, if the duration of the patient's illness should be unknown. In persons advanced in years, a mere bronchitis is sometimes attended with a dry, brown tongue, stupor, and other "typhoid" symptoms, the real cause of which may be overlooked.

But the condition which is most apt to be mistaken for typhus at this stage is uræmia, dependent upon chronic disease of the kidneys. Murchison says that patients have often been sent to the London Fever Hospital whose symptoms were all of renal origin; and at Guy's Hospital in former years the doubts with regard to such cases have sometimes never been entirely cleared up. Conversely, in 1865, a man who was in a surgical ward for stricture became feverish and drowsy, and his illness was attributed to an ascending nephritis until a mulberry rash was discovered upon him; he died, and the kidneys were found to be healthy. It must be borne in mind that the existence of a chronic renal affection, when established by *post-mortem* examination, affords no proof that typhus was not also present. According to Murchison, however, the thermometer generally affords a suffi-

cient means of diagnosis; the temperature is at or below the normal in cases of Bright's disease, unless complicated with an acute inflammation.

Prognosis.—It is very important to remember that the prognosis in typhus must be based upon a variety of considerations, apart from or in addition to the mere severity of the symptoms. Chief among these is the age of the patient. The older he is the greater, in this disease, is the danger. In children it seems never to be fatal, except by some complication. Murchison found that at the London Fever Hospital the average death-rate for all ages being from fifteen to nineteen per cent., the rate in persons above thirty was 35.39 per cent., in those above forty it was 43.48 per cent., in those above fifty it was 53.87 per cent., in those above sixty it was 67.04 per cent.

Again, typhus is especially apt to terminate fatally in persons who have been intemperate, who have suffered severely from gout, who have been exhausted by fatigue of body or mind, or who have been suffering from privation of food or from severe mental depression. The greater frequency with which these various conditions occur among adult males, as compared with females, is probably the reason why the mortality is higher in them; among children between five and fifteen it is lower in boys than in girls. Patients who have gone on struggling against the disease during the first few days often do badly; having exhausted their muscular power, they become rapidly prostrate.

With regard to the prognostic significance of particular symptoms, I need add but little to what has already been incidentally stated. Murchison remarks that a presentiment of death is a very unfavorable, but not necessarily a fatal indication. The danger in a given case is generally best measured by the intensity of the cerebral disturbance and by the degree of prostration; the earlier the date at which severe symptoms are present, the worse is the prospect. Sleeplessness continued throughout several days is of evil omen. A very abundant rash is usually a bad sign, especially if the spots are dark and rapidly become petechial. A pulse over 120 is always a serious matter in an adult; and when it exceeds 150 death is almost certain to occur. Relaxation of the sphincters before the tenth day is unfavorable; but toward the end of the second week it is not uncommon in severe cases, which may nevertheless do well. A "typhoid" state, with dry, brown tongue, sordes, and subsultus, is observed in many patients who recover. Indeed, even in what appear to be the worst cases, one should hardly ever despair. Great lividity of the face and limbs and coldness of the distal parts of the body, with profuse sweating, are among the most threatening symptoms.

Treatment.—We as yet know of no means of arresting the course of typhus, or of bringing it to a uniformly favorable termination; but a great deal may be done to increase the chance of a patient's recovery, and many of the most painful and distressing symptoms may be alleviated or checked. Murchison cites observations made, both at Philadelphia and at Belfast, from which it appears that the death-rate of this disease was greater in proportion to the duration of the fever before admission into the hospital.*

* It must not be supposed that neglect of medical assistance was the sole, even if it was the principal cause of the varying mortality in question; in many instances a fatal issue was doubtless the direct result of removing the patient at an advanced stage, whereas at an earlier period it might have done him no injury. Murchison says he has repeatedly known patients die from exhaustion caused by their being carried for several miles in a shaking vehicle. But that, with proper precautions, a moderate journey may be effected without risk appears from the fact that, among a number of patients admitted into the London Fever Hospital from the immediate neighborhood, the death-rate was, during five years, almost exactly the same as among those brought from the distant parish of St. George's-in-the-East.

When typhus attacks a person in comfortable circumstances, he may be safely left at home, provided that his apartment is spacious and well-ventilated, and that he can be properly nursed. But those who are poor should at once be carried to a hospital. The establishment of special institutions for the reception of fever cases dates from the commencement of the present century; but so recently as 1842 many London physicians, among whom was Richard Bright, were in favor of mixing such cases with others in general wards, rather than of setting apart special wards for them, or of collecting them in buildings devoted to that particular purpose. However, so far as typhus is concerned (and the same may be said of relapsing fever) this was certainly a mistake. For (1) it has now been proved by experience that if a sufficient cubic space of 2000 feet be allowed, and if ventilation be well attended to, the death-rate is not greater among typhus cases accumulated in the same ward than when they are scattered. (2) In every hospital in London where such cases have been placed in general wards there have been terrible examples of the spread of fever to other patients. To cite but a single instance: in 1862 one or two cases of typhus were admitted into one of the large medical wards in Guy's Hospital; seven other patients took the disease, and five of them died. What makes the argument stronger is that in a large proportion of medical cases, particularly in those of Bright's disease and of diabetes, the risk of a fatal termination, if typhus should be caught, is very much greater than among healthy persons. Moreover, the free ventilation, which is the chief condition of safety under such circumstances, is directly prejudicial to persons suffering from pulmonary complaints. It would be infinitely better to admit smallpox cases into a general ward than those of typhus, because protection against the former disease could easily be given by a general vaccination. Murchison has shown that the proportion of cases of typhus which have arisen at the London Fever Hospital, in comparison with the number of cases admitted, has been far less than in the general hospitals of the metropolis. (3) So far as medical men and nurses are concerned, the danger of the extension of typhus may be greatly reduced by retaining for as long a period as possible the services of those who have once passed through the disease, and by invariably selecting persons under thirty for all vacancies that may arise. Every large town ought to have a fever hospital, capable of enlargement during an epidemic by the erection of temporary buildings of iron or wood. All general hospitals should have one or more separate fever wards.

A patient suffering from typhus should be placed upon a spring bed, or upon a hair mattress, with not too many bed clothes. The temperature of the ward or room should not exceed 60°. After the first few days he should not be allowed to get up, even to pass his excreta. It is important that his nurse should be strong enough to lift him up in bed when necessary. From time to time he should be turned over to one side or the other, and kept from rolling back by pillows.

Food.—A nourishing diet is of the highest importance. Dr. Graves, of Dublin, desired, as his own future epitaph, the testimony "He fed fevers;" and it is to typhus rather than to any other fever that this is applicable. So long as the patient can be tempted with food, it is important to vary it as much as possible. Besides milk, eggs, and beef tea, he may have broth made of mutton, veal, or chicken, meat juices and extracts, jellies, custards, blancmange of isinglass or ground rice, white wine whey, etc.; and vermicelli may be given in the beef tea, small pieces of bread or toast in the broth, and arrowroot in the milk.

For beverages he may be allowed to choose among barley water, toast and water, lemonade, tamarind water, currant water, effervescing drinks, and cold weak tea. Murchison recommends that he should drink often, in order

to keep up free excretion by the kidneys, but he must only take a small quantity at a time. Before many days have passed, however, he acquires a distaste for everything but cold water. It is then essential that food should be pressed upon him and administered at regular intervals of from one to three hours; to give it more often than this is, according to Murchison, injurious rather than useful. If he be drowsy he should be roused for the purpose, and this can generally be done without seriously disturbing him; but if he should be sleeping quietly after having been very restless, the interval may sometimes be a little lengthened. It is, however, of great consequence that his strength should not be allowed to run down at night or during the early morning, when the risk of exhaustion is always greatest. When, as is often the case, he clenches his teeth and obstinately refuses food, it must either be introduced into the stomach by means of a tube passed through the nostril, or else injected by the rectum. When there is obstinate vomiting, the latter is the only plan that can be had recourse to.

Stimulants.—In many cases the administration of alcohol is a very important part of the treatment. Whether it should be regarded as a food or as a drug is still a question. The former view, in which I for my own part concur, is held by Buchanan; the latter was held by Murchison. But as to the practical rules for its use there is at present very little difference of opinion. All observers are agreed that alcohol seldom need be given to children, that it is almost always required for patients over forty, and that persons whose habits have been intemperate must have it earlier and in larger quantities than others. The state of the heart affords the best indication; the necessity for stimulants is greater in proportion as the cardiac impulse is feeble, and as the radial pulse is rapid, compressible, or, above all, irregular. The propriety of continuing to give alcohol may often be determined by its effect on the heart's action. If the pulse becomes quicker than when the patient was taking none, it is likely to do harm; if slower, it may be expected to do good. It is most needed during the second week; very seldom before the appearance of the mulberry rash. Low muttering delirium is often controlled by stimulants, a dry, brown tongue becomes moist, and other "typhoid" symptoms become less marked. On the other hand, severe throbbing headache and violent maniacal excitement are often aggravated. A burning, dry skin is so far a reason for withholding alcohol; profuse perspiration, especially if the limbs are cold, calls for an increased supply. As a rule it should not be given when the urine, although scanty, is of low specific gravity, or when there is suppression of the renal function.

Probably there is no direct advantage in employing one alcoholic liquor rather than another. Some observers prefer brandy or whisky, others port wine; Buchanan speaks highly of beer. The amount must vary with the urgency of the symptoms. Murchison thought that it was very rarely necessary to give more than eight ounces of brandy in the twenty-four hours, but some physicians order as much as twenty ounces. Of port wine as much as a bottle may be taken with advantage by patients who are desperately ill. But only small doses should be allowed at a time, repeated very frequently, at intervals of an hour or two; and when the daily quantity is very considerable, the fact that milk mixes with brandy much better than with wine is an important reason for preferring it. When there is great prostration, with cold sweats, hot brandy or whisky punch, or hot wine whey, is recommended by Murchison. At Guy's Hospital it has of late been the fashion with house physicians to inject stimulants under the skin in such cases. The practice was, I believe, first suggested by Zülzer. Thirty or forty drops of brandy, or of the spirit of sulphuric ether, may be administered in this manner. Ether, carbonate of ammonia, camphor, or musk may also be given by the mouth.

Treatment of Symptoms.—The *pyrexia* itself seldom requires direct treatment in typhus. So far as I know, the routine use of cold bath has not yet been sufficiently tested in this disease to prove its value. Murchison satisfied himself that quinine in large doses of ten or twenty grains, although it lowers the temperature by three or four degrees for twelve or eighteen hours does no real good. Only in exceptional cases, when at the crisis the thermometer was rising instead of falling, did it seem to him to be sometimes instrumental in saving life. The medicine usually given at the London Fever Hospital appears to be dilute hydrochloric acid, in doses of twenty minims, with a little syrup and tincture of orange. Murchison says that he has often "observed the tongue become moist, and a marked improvement follow the commencement of the acid treatment, at whatever stage it was tried." When there is insatiable thirst he recommends a very weak infusion of cascarilla or quassia.

The early *headache*, which is often a very distressing symptom, may sometimes be checked by an emetic. When the patient is young and robust, there is no objection to applying three or four leeches to the temples, and they frequently give complete and permanent relief. Or the hair may be cut, and the head be covered with a bag of ice, or a tubular cap through which cold water is running. In other cases a blister to the forehead is of service ; or hot fomentations, as recommended by Graves, may be employed, especially in old or debilitated subjects.

But in many instances it is advisable to prescribe opium for the relief of headache, particularly when it is accompanied by an inability to sleep. Murchison says that *sleeplessness* at any stage of typhus, if it continues for two or three nights, is of itself sufficient to kill ; and that whenever it lasts for thirty-six hours it should always be combated by medicine. During the first week one may almost always give fifteen minims of Battley's liquor opii or a quarter of a grain of morphia at night, following this, if necessary, by another half dose two hours later. But at more advanced periods of the disease there is often danger in prescribing these drugs. When the patient is in a state of maniacal delirium, Murchison recommends chloral hydrate in a dose of twenty grains, which may be once repeated should the first dose not succeed. If this treatment fails, recourse must be had to opium, with which antimony may often be combined, as suggested by Graves. The chloral hydrate may also be given in some cases in which the delirium is low and muttering, or like that of alcoholic origin ; but when the heart's action is feeble or irregular it sometimes does harm. Murchison then used to prefer opium, prescribing it in conjunction with tincture of digitalis and sulphuric ether. Other drugs which are more or less useful are bromide of potassium, the extract of Indian hemp, and the tincture of hyoscyamus or of belladonna. They must each be given in rather large doses to do any good.

When profound *stupor* appears to threaten the patient's life Murchison advised that a cupful of strong coffee should be given every three or four hours. He also thought it important to use dry cupping to the loins, and to apply mustard poultices, or flannels wrung out of hot water and covered with mackintosh, especially when the urine was scanty and albuminous. And he believed that he had saved some cases by blistering the scalp with a piece of lint soaked in strong liquor ammoniæ, and applied for five or six minutes under oiled silk. Should high fever accompany coma, cold affusion to the head may sometimes be employed with great advantage.

For the *pulmonary complications* of typhus, ammonia is the chief remedy. But in some cases turpentine is said to be even more decidedly effectual ; it is given in doses of fifteen minims every three hours. Mustard poultices should also be applied to the back and sides of the chest.

For *inflammatory swellings*, usually in the parotid region, both Murchison and Buchanan recommend the application of a blister at an early stage, as sometimes appearing to prevent the occurrence of suppuration. When an incision is required it should be made early.

After the subsidence of the pyrexia the patient should be kept upon a restricted diet for the first two days; but upon the third day, if the tongue be clean, he may have a little boiled fish or chicken, or the lean part of a chop. If wine or brandy has been given, beer should be substituted while the case is still under treatment, so that there may be no excuse for carrying on the habit of tippling when health is restored.

RELAPSING FEVER.

HISTORY OF ITS DISTINCTION FROM TYPHUS—ITS OCCURRENCE IN EPIDEMICS—COURSE OF RELAPSING FEVER—OCCASIONAL MORTALITY, DIRECT OR INDIRECT—THE SPIRILLUM—MODE OF CONTAGION—FAMINE AS A PREDISPOSING CAUSE—DIAGNOSIS—TREATMENT.

We shall hereafter find that the differentiation of enteric fever from typhus was brought about by a long series of researches which occupied all the first half of the present century. During the same period, a third fever also was discovered, which is generally known as Relapsing Fever, from its regularly consisting of two or more attacks, separated by intervals of apyrexia.

According to Murchison, the distinction between this affection and typhus was first drawn in Ireland, in 1826, when they both prevailed at the same time; but there is evidence that it also formed part of the epidemic of 1817–19, and even of several earlier Irish epidemics, as far back as one recorded by Rutty as having occurred in 1739. Being comparatively seldom fatal, and being of much shorter duration than typhus, it was in 1826 regarded as a mild variety of that disease. But in the year 1842, when it next appeared, Dr. Henderson, of Edinburgh, brought forward good grounds for believing that it was a distinct fever, and due to a different poison. The same view was strongly upheld by Jenner, in 1849–51. The arguments used by these writers were chiefly three. 1. That the symptoms and course of the two diseases were different. 2. That even when they prevailed together in the same town, one could never be traced to infection from the other. 3. That neither of them afforded protection against a subsequent attack of the other. To these arguments may now be added the fact that, in relapsing fever, the blood always contains a microscopic organism, a spirillum or spirochæta, which is absent in typhus. But although the doctrine in question is at the present time universally accepted as true, the evidence adduced under the second and the third heads appears to have been somewhat weakened by the investigations which have been made since the publication of Henderson's and Jenner's papers. For, whereas they maintained that cases of relapsing fever were not proved to come from the same houses or families as those of typhus, Murchison has shown from the later records of the London Fever Hospital that exceptions to this rule sometimes occur, and moreover, that in certain narrow streets or courts it is no uncommon thing for typhus fever gradually to replace relapsing fever in the course of some months. Again, since it has been ascertained that the same individual often has relapsing fever more than once, the fact that this disease does not protect against typhus has lost its significance. But in the opposite direction it has still been thought that there is an immunity. Dr. Steele, of Glasgow, in 1847, remarked that persons who had previously suffered from typhus were not attacked by relapsing fever; and the same thing is said to have been noticed recently in epidemics at St. Petersburg and at Breslau. Dr. Henderson, however, related six cases in which typhus

occurred first and relapsing fever afterward; and Murchison says, that of thirty-one persons who contracted relapsing fever in the London Fever Hospital, in 1868–9, no fewer than thirteen were known to have had typhus. Lastly, Lebert draws attention to the fact that in his cases of typhus, among those who had already passed through relapsing fever, the mortality was only half as great as in the population generally. Do these facts indicate that after all there is between the two diseases some connection, the nature of which is at present not understood?

At the beginning of the great epidemic of 1847–8, and also in 1851, it was particularly noticed, both in London and in other large towns of Great Britain, that the chief patients were destitute Irish, many of whom had recently left their own country; afterwards the English or the Scotch were attacked. Whether, in 1842, the disease was derived from Ireland is not clear. The latest epidemic, that of 1868–71, seems to have had its origin in the east of Europe. There is reason to believe that in 1848 relapsing fever had prevailed in Upper Silesia. With this exception it is not known to have ever existed on the Continent of Europe until, in 1863, it was observed in Odessa, and in 1864 at St. Petersburg. Next, in 1867, it broke out for the second time in Silesia, and in the following year it prevailed in Berlin, and in other German towns, as well as in Breslau. In 1868 it reappeared in England. The first case, that of a woman of Irish birth who had been for several years in London, was brought into the Fever Hospital on July 4th. Four days later, another case occurred in the person of a Polish Jewess, who could speak no English; she came from a house about a quarter of a mile distant from that in which the former patient resided. From the same house, on the same day, a Polish family was admitted consisting of father, mother, and child; they, however, were merely in a state of extreme prostration, having no fever then upon them. Three weeks later there arrived from the house next adjacent, a girl who had lived all her life in the metropolis. The only other persons who are known to have been attacked in London in that year were eight German Jews; they all were admitted into the Dalston German Hospital. But in October, a severe outbreak occurred at Tredegar, in South Wales. In the autumn of 1869 relapsing fever became epidemic in several of the large cities of England; in London the disease reached its height in December of that year, and then gradually declined until June, 1871, when it finally disappeared. In Breslau it was again epidemic in 1872–3. I think there can hardly be any doubt that the Poles admitted into the Fever Hospital in 1868 had either directly introduced the contagion from their own country, or perhaps had received it in a letter or in a parcel, or had derived it from other Poles or Germans who had themselves so received it or brought it with them from the Continent. In a city like London, one can but seldom expect to trace such events to their sources, and it is instructive to observe that the disease did not spread then until a year later, although it had in the meantime broken out in a remote part of Wales. In September, 1869, it appeared at Philadelphia, and in November, at New York. The patients were chiefly poor Irish or Germans, and it seems to be almost certain that the contagion was imported from Europe, although its origin could not be discovered. On two or three former occasions it had been introduced into the very same cities by Irish emigrants, but it never showed any tendency to spread among the American population.

With regard to the occurrence of relapsing fever in Africa and in Asia, our information is still very imperfect. Griesinger observed it in Egypt in 1851. There is reason to believe that it has repeatedly prevailed in India. The latest epidemic there, that of 1877, at Bombay, was carefully studied by Dr. Vandyke Carter ("*Med.-Chir. Trans.*," vol. lxi).

Course.—The period of incubation for this disease appears to be very variable: Murchison says that it sometimes breaks out immediately after exposure to its exciting cause, sometimes not until from two to fourteen days have elapsed. During the interval, no symptoms whatever are present; only in some very exceptional cases is there malaise, with a little loss of appetite for a few hours, or a day or two, before the beginning of the attack. This generally takes place with remarkable suddenness. The patient, while engaged as usual during the day, or perhaps on first waking in the morning, is seized with chilliness or with rigors; his head aches, he feels giddy, he has pains in the back and limbs. In a short time his skin becomes burning hot. All his complaints grow rapidly worse; he at once takes to bed, and he often seeks admission into an hospital on the first or the second day. One of the distinctions from typhus is that it is giddiness rather than prostration which prevents his keeping about. He is sometimes able to walk to the hospital two or three days after the seizure.

The thermometer indicates a very rapid rise of temperature. This begins even before the rigor, and within twelve or twenty-four hours it reaches 104° or 106°. The pulse, too, becomes quickened much earlier than in typhus; it is scarcely ever found below 110, and not infrequently it reaches 140 on the second day. The tongue is moist and covered with a white or yellowish fur. There is great thirst. Appetite is generally wanting, but sometimes it remains good, or even voracious, the patient being able to take solid food without any harm resulting. In some cases, however, there is much nausea, and even vomiting of a green, bile-stained fluid. Pain, with tenderness on pressure, is often present in the epigastric and the hypochondraic regions of the abdomen. By percussion one may make out some increased dullness of the liver; the spleen usually becomes very decidedly enlarged, so that its edge reaches far below the costal cartilages. Jaundice often occurs on or after the third or the fourth day; in some epidemics it has been observed in one of every four or five cases, but generally not oftener than in one of eleven or twelve. It is now and then very bright, and the urine may be loaded with bile pigment; but the fæces appear always to remain of a dark color.

The face is flushed and the eyes are injected in relapsing fever, but the countenance is not usually dusky, nor is the expression stupid and confused, as in typhus. As a rule, there is no eruption on the skin; but several observers have noticed a roseola in certain exceptional cases. Murchison, who met with this in eight out of about six hundred cases, says that the rash consisted of small spots, or of a reddish mottling, sometimes resembling measles, but more often undistinguishable from typhus at an early stage; yet always disappearing under pressure, and fading after a few hours, or within three or four days at the least. It came out sometimes during the first attack, sometimes in the relapse; and either as early as the third day, or immediately before the crisis. Petechiæ are not uncommonly present; no doubt flea bites have frequently been taken for them, but Murchison says that this is certainly not always the case, for large numbers of them may come out in a single night after the patient's admission into the hospital. The surface of the body often remains very dry, but sometimes on the second or third day there is profuse sweating, which may last for hours without in any way relieving the other symptoms of the disease. Sudamina may be developed in large numbers; I suppose that they are the cause of the marked desquamation of cuticle which frequently occurs at a later period.

When relapsing fever occurs in a pregnant woman, gestation is almost invariably brought to an end, whatever may be its period; but it is curious that the abortion or miscarriage is not seldom delayed until the relapse.

The child, if not stillborn, survives only a few hours. There is, in these points, a marked contrast with what occurs in typhus.

In the regular course of the disease, the patient's condition undergoes but little alteration after the first day or two, for about a week. The temperature and pulse remain high, oscillating upward and downward a little at one part of the day or another, the pulse-rate sometimes reaching 150, 160, or even 180. The patient continues to suffer from severe headache, often of a throbbing character, and from intense pains in the muscles and joints, much increased by movement. Murchison remarks that relapsing fever is altogether attended with far more suffering than typhus; persons who have passed through both diseases invariably look back upon the former as the worse to bear. Reasons for this are that sleeplessness is almost always a marked symptom, and that the mind generally remains clear, although there may be a little delirium toward the end of the attack.

It is generally on the fifth or the seventh day that a sudden subsidence of the fever occurs, constituting the most remarkable instance of a *crisis* with which we are acquainted. Sometimes, however, this takes place as early as the third day, sometimes not until the tenth. Just before it, the fever often reaches its *acme*, touching a point higher than at any previous time; there may even be a rapid rise through as many as 4°. In some exceptional instances a maniacal delirium suddenly comes on at this time, the patient screaming and struggling violently, and passing his evacuations under him, but within a quarter of an hour becoming again calm and having no recollection of what has occurred. In other cases the crisis is said to be ushered in by epistaxis, diarrhœa, or the catamenia. But, as a rule, what characterizes it is the occurrence of profuse sweating. The change is wonderfully complete. In the course of a few hours the temperature becomes normal, or even lower than normal, having fallen probably 8° or 10° without a break; Murchison cites one case in which there was a range of 13° in six hours, and another in which the difference amounted to 14.4° in twelve hours. At the same time, the pulse drops from 120 or more to about 70, the tongue becomes clean, and the patient loses all his pains. Lebert remarks that the period from evening to morning is that at which the crisis is most apt to occur. Litten has pointed out that it is sometimes preceded by a *pseudo-crisis*, the temperature falling below normal, but within twenty-four hours rising to as high a point as before, again to fall on the following day.

During the interval which follows, the patient feels perfectly well. At first he is rather languid and exhausted; but his appetite is good, he quickly regains his strength, he gets up and walks about, he may insist upon leaving the hospital, and may even return to work. His temperature for two or three days is almost always lower than in health; but afterward it becomes normal. On the other hand, the pulse is at first rather above the natural standard; but at a later period it is often remarkably infrequent, perhaps not more than 44 or 50. Murchison remarks that in such cases it may rise to over 100 when the patient assumes the erect position.

In exceptional cases this apparent convalescence is in reality the end of the disease. But, as a rule, it is abruptly terminated, when it has lasted seven days, by a sudden reappearance of all the symptoms which had belonged to the first attack. Sir Robert Christison related a striking anecdote in reference to his colleague, Dr. Hughes Bennett, who took relapsing fever at the commencement of the epidemic of 1843, when its characters were known only by the older members of the profession. "When he had detailed to me his case," being then supposed to be convalescent, "I told him he had sustained an attack of my old acquaintance . . . whose face I had not seen for a good many years; that he was not yet done with

it, and that he would have another attack, commencing with rigor, on the fourteenth day," (of the disease). "Dr. Bennett, surprised—I will not say incredulous—replied that the relapse had no time to lose, as there were only three or four hours of the fourteenth day to run. It did, indeed, lose no time, for I must have scarcely reached home from his house before the rigor set in with violence." Sometimes, however, instead of occurring on the seventh day of the interval, the relapse begins on the second, or the third, or the fifth day; sometimes it is postponed for several days, and even as late as the twenty-fifth day. Lebert says that it usually sets in at night.

This second attack is generally shorter than the first one, lasting three days, or not more than one or two, but sometimes it is prolonged to five days, or even to as many as seven or eight. Both in the details of their symptoms and in their severity the two attacks may resemble one another exactly, but in some patients the relapse is the worse, especially when the original attack was very mild; in others it is by far the less severe of the two, and it may be so slight as almost to escape notice, being indicated only by a trifling malaise with a little elevation of pulse and of temperature. When it is well marked, it ends in a sudden crisis, just like the former one.

Recovery.—The patient now generally passes on to a permanent recovery, but sometimes there is a third attack, which begins between the twenty-first and twenty-fourth days of the disease; it is almost always very mild, and seldom lasts more than forty-eight hours. There may even be a fourth or a fifth attack. In any case convalescence after relapsing fever is slow, as compared, for example, with that after typhus. The patient is a long time in regaining his strength. Notwithstanding that the duration of the disease up to the second crisis is only about eighteen days, he is seldom able to return to work within six weeks.

I have already remarked that relapsing fever seems to confer little or no immunity from subsequent attacks. All writers record instances in which persons have taken the disease two, or even three, times in the same epidemic.

Fatal Event.—It is only in exceptional cases that relapsing fever proves fatal. Murchison states that in this country the mortality has been 4.03 per cent. In Bombay, however, Dr. Vandyke Carter estimated it at 10 per cent. In persons advanced in years the disease is decidedly more dangerous than in younger persons. One way in which death may occur is by sudden collapse. At and after the crisis the pulse is often very feeble, small, or irregular; there may even be a temporary impairment of the first sound of the heart and of its impulse. These symptoms usually soon disappear, but the liability to their occurrence probably affords some explanation of the fact that a patient who may have had a mild attack, and who may have appeared to be doing perfectly well, is now and then found to become pulseless, cold, livid, and unconscious, and dies in a few hours. This sometimes takes place at about the period of the first crisis, but sometimes during the interval. In three such cases observed by Murchison the heart was found fatty and dilated. Another mode of death is by suppression of urine leading to convulsions and other cerebral symptoms. In some instances of this kind, recorded by Dr. Henderson as far back as 1843, Dr. Maclagan discovered urea not only in the blood, but also in fluid from the cerebral ventricles. It is probable that on post-mortem examination the kidneys would be found to be in a state of chronic disease, and that albuminuria would have been present at an earlier stage of the attack, but the urine not seldom contains albumen, and even blood, in relapsing fever, without any serious consequences resulting. Again, there is a variety of the disease attended with typhoid symptoms, and invariably accompanied by jaundice, which

often terminates fatally; this Griesinger described under the name of "bilious typhoid" as a distinct form of fever. In it the interval is imperfectly marked, the pyrexia running on more or less continuously from the first attack to the second. As, however, Heydenreich, in St. Petersburgh, and Carter, at Bombay, have each shown that the spirillum was present in cases of this kind, we may now safely follow Murchison in rejecting Griesinger's view with regard to them. Among his symptoms are hemorrhages into the skin and from various mucous surfaces, including that of the stomach, so that the vomited matters may be black, like those of yellow fever.

There is little to be said about the state of the organs in the cases of relapsing fever which prove *directly* fatal. The spleen, if death occurs during the attack, is greatly enlarged, more so than in either typhus or enteric fever. Küttner is said to have found this organ four and a half pounds in weight. It is often, but not always, soft. Infarctions are sometimes seen in it. In the one case which came to an autopsy at Guy's Hospital during the epidemic of 1869-71, the spleen, which weighed fifteen ounces, had a remarkable appearance. It contained a number of small, yellow, softened patches or abscesses, the largest being of the size of a horse-bean, which seemed to follow the branchings of minute veins. Dr. Moxon could discover no thrombi in any of the vessels, even with the aid of the microscope. Very similar appearances have been described by Litten as having been observed at Breslau in the epidemic of 1872-3. As a rule, the kidneys are gorged and swollen, and the epithelium in the renal tubules is in a state of cloudy swelling.

In other cases, however, relapsing fever proves fatal *indirectly*, through some secondary lesion arising as a complication. Thus pneumonia has in certain epidemics been of rather frequent occurrence, as, for example, at Breslau in 1872-73. Lebert says that it was generally double, that it occurred chiefly in those who had been intemperate, and that it sometimes set in during the attack, sometimes in the relapse. It has been known to lead to pulmonary gangrene. Bronchitis is commonly present, but it is seldom severe. In St. Petersburgh hemorrhagic pachymeningitis is said to have been observed. The spleen has sometimes ruptured, discharging a quantity of blood into the peritoneal cavity, and causing death by rapid collapse. In other instances an infarction in this organ has broken down and set up a fatal peritonitis or pleurisy. Another cause of peritonitis may be dysentery, which has been a frequent and a very dangerous complication in some epidemics. The occurrence of abortion is sometimes fatal by profuse hemorrhage, or by rapid sinking. Inflammatory swellings or "buboes" in the parotid regions or in the groins are said to have been often the cause of death at St. Petersburgh, but in England they appear to have been more frequent in cases which recovered.

Sequelæ.—The disease sometimes leaves troubles behind. Severe articular pains often continue during convalescence, and effusion into the knee or ankle joint has now and then been seen.

Another affection, which sometimes does not set in for several days, weeks, or even months after the subsidence of the fever is a form of ophthalmia. This is described as beginning with an amaurotic stage, in which the retina, or perhaps the choroid, is alone attacked; afterwards, in what is termed the second or inflammatory stage, when the more superficial structures are involved, there is intense pain in and around the eye. It fortunately seldom happens that both eyes are affected, for recovery is always tedious, and there is sometimes permanent loss of vision.

Origin and Spread.—In reference to the ætiology of relapsing fever the first question is as to the significance of the minute organisms which are present in the blood, and which were originally described

by the late Dr. Obermeier, of Berlin, in 1873, although he had observed them as far back as 1868. They are commonly spoken of as *spirilla*, but the technical term for them is strictly *spirochætæ*, for Ehrenberg, in 1833, distinguished two genera of *schizomycetæ* under these names, the difference between them being that the spirochæta possesses a flexibility which is wanting to the spirillum proper. It seems to be doubtful whether the form found in relapsing fever is identical with that which had previously been observed in water containing decaying vegetable matters and in the saliva of healthy persons. Dr. Vandyke Carter, at any rate, says that the dimensions of an organism of this kind which he met with in the water of a tank at Bombay were much larger than those taken from his fever patients ("*Med.-Chir. Trans.*," vol. lxi). Provisionally we may leave to the former Ehrenberg's name of *S. plicatilis*, distinguishing the latter as *S. Obermeieri*.

Its ordinary appearance is that of a delicate, homogeneous, spirally-twisted filament; its length is from $\frac{1}{2000}$ to $\frac{1}{500}$ of an inch, or equal to from one and a half to six times the diameter of a red blood disc. It is never still, and its motion is compounded of a rotation on the long axis, a progression forward or backward, and a lashing movement, which lengthens it out and causes the twist for the time almost to disappear.

All observers admit that the presence of the spirochætæ is coincident with the attacks of relapsing fever, and that during the interval or intervals it cannot be detected. Birch-Hirschfeld, indeed, once found it for two days after the second crisis, and several writers, including Litten, have stated that it is seldom to be seen during the first two or three days of the disease. But Heydenreich, of St. Petersburgh, who has since made what seems to have been a series of careful investigations, declares that while it appears before the thermometer begins to rise, it ceases to be discoverable before the commencement of the crisis. He has specially inquired into the influence of heat upon this organism outside the body, and he finds that it remains alive, as evidenced by the persistence of its active movements, much longer when it is kept at a temperature of 60° or 70° F. than at blood heat, whereas at fever temperatures it dies more quickly still. His hypothesis accordingly is that the pyrexia, which is itself caused by the existence of this organism in the blood, proves directly fatal to it. In all probability the filament breaks up into a number of minute granules, and some of them may perhaps constitute the germs from which fresh crops of spirochætæ are afterwards developed. Heydenreich has occasionally seen some of the filaments beset with granules so as to resemble a necklace, and Dr. T. R. Lewis, of Calcutta, speaks of having once noticed a beaded appearance. If the germs remain alive in the blood throughout the whole of the interval, they must possess the attributes of *Dauersporen*, for the spirochæta itself dies at the temperature of the blood in about twenty hours.

Even during a single attack, Heydenreich finds that there are extraordinary fluctuations in the number of the spirochætæ from day to day. He therefore supposes that successive generations are more or less constantly being produced throughout the fever. Sometimes, after the filaments have been present for two or three days, they suddenly cease to be discoverable; but a few hours later they reappear in greater or less abundance. Their recognition in the blood, at the end of an interval, has repeatedly enabled him to foretell the approach of a relapse, which, however, he admits may be so slight as to be scarcely noticeable. On the other hand, Dr. Carter says that at Bombay he sometimes detected the spirochætæ at periodic dates, when there was absolutely no rise of temperature; while in other cases a single one-day febrile attack would occur at the proper time for a relapse, without the blood containing the organism. This observer

remarks that the numbers of them most often amount to hundreds of millions, dozens being seen in the field of the microscope at the same time. It even seems likely that they may become aggregated together into dense masses with blood corpuscles, and seriously interfere with the circulation, so as to produce lividity of the face, etc. In fatal cases they cease to be discoverable after death. None of the solid organs have hitherto been found to contain them. Heydenreich could neither detect them in the urine, nor in the conjunctival secretion, in the fluid from the pleura, the intestine, or the bronchial tubes.

As to the origin of the *Spirochæta Obermeieri* we at present know nothing whatever. All attempts to cultivate it experimentally have hitherto failed. Injections of blood infected with it into the circulation of dogs, rabbits, or guinea pigs were made by Obermeier himself without result. Nor did he find that the disease was propagated by the penetration of minute quantities of blood from patients with relapsing fever into scratches upon the hands of healthy persons, although Dr. Carter has since recorded the fact that a few days before he himself was attacked he had scratched his own finger in making an autopsy upon a fatal case.

It is, however, certain that contagion from the sick plays a very important part in the spread of the disease. Thus in hospitals it is exceedingly apt to pass from patients to nurses and clinical clerks. In 1843 the post of house physician in a fever hospital in Edinburgh had to be filled six times in five months, five gentlemen having been attacked in turn. In the London Fever Hospital, during the years 1869 and 1870, twenty-seven of the nurses and officers, and five patients admitted for other diseases, contracted relapsing fever. In 1870 a nurse from the Fever Hospital was transferred to St. Mark's Hospital for fistula; and about the same time a ward maid from St. Mark's paid a visit to the Fever Hospital. These two women both fell ill at St. Mark's, and gave the fever to five other persons there. Two striking instances were recorded by Mr. Reid, of Glasgow, in 1843, to show that the spread of the disease was not due merely to a widely diffused atmospheric influence. (1) At Dalmarnock Colliery there was a large building, consisting of three stories, which was entered by separate stairs and contained forty families; some Irish people brought relapsing fever with them into a single apartment on the uppermost flat, whereupon it spread from room to room, and ultimately descending a stair, attacked twenty-two individuals in the space of two months. (2) Into a house of two apartments, in which eleven human beings were lodged, a person from a neighboring village introduced the fever; every person there fell ill, but all of the occupants of the next house, separated only by a brick partition, escaped, although they were almost as closely crowded together. That the disease may be transported to a distance by infected clothes seems to have been proved by two cases recorded by Dr. Parry, of Philadelphia, in 1870; and in 1843 it was noticed in Edinburgh that a large number of laundry women contracted relapsing fever, although they had no communication with the sick, beyond having to do with their clothes and bedding. Litten has related an instance from the epidemic at Breslau in 1872–3: a mason, who himself remained well, but who slept in a street in which the disease was prevailing, carried the contagion to his mother, who lived where no case had before appeared.

Special interest, indeed, attaches to this particular epidemic, because it is the only one which has as yet been traced out in detail with the object of referring each case, if possible, to infection from some previous one. It is impossible for me here to cite the facts in reference to it, which are to be found in the "*Deutsches Archiv*" for 1874, but I may say that they strongly support the view that this is the mode in which the disease really spreads, even where, as must often be the case, this cannot be directly demonstrated.

On the assumption that the contagious principle consists in the spirochæta, or in its germs, we must suppose that they are given off either with the breath or from the skin. In all probability they are received into the bodies of other individuals with the inhaled air. Litten has clearly shown that at Breslau they were not conveyed in drinking water. And in this connection it is, perhaps, worthy of mention that Dr. Carter has ascertained that the spirochæta does not exist in the contents of the thoracic duct.

The cases observed by Litten, in Silesia, seem to have belonged to the very poor classes, with the exception of the medical men who took the fever in the hospital; and English writers have generally been disposed to state as the cardinal fact in the ætiology of this disease its occurrence in those who have been in a state of starvation. It is the *famine fever* of Ireland. Murchison cites instance after instance to prove that those who had suffered from it in England and in Scotland have, with certain exceptions, been in a state of extreme destitution, often reduced to live upon raw turnips, rotten apples, etc. When it occurred in Silesia, in 1847, the inhabitants, in consequence of a succession of bad harvests, had been reduced to subsist on clover, grass, mushrooms, and the roots of trees. Carter states that it was brought to Bombay, in 1877, by the peasantry flocking into the city from famine-stricken districts of Western India. Murchison himself was of opinion that the specific poison of the disease might be generated *de novo* in the human body as the result of inanition. He cited the observations of Holland and of Donovan to prove that persons in a state of starvation suffer from delirium, stupor, and coma; that they may have a quick pulse, a dry tongue, flushing of the face, intolerance of light, and neuralgic pains over the body, and that they exhale putrid odors from the skin. He believed it to be a point in favor of such a view that tramps have often been found to bring the disease with them into London after sleeping by the roadside, under hedges. Dr. Carter also says that one of the earliest cases he saw in Bombay was that of a destitute lad who, while tramping up from the famine district of Poonah, was seized with fever in the upland country, and was barely able to crawl into the hospital on his arrival at Bombay. But it is obvious that in none of these instances could the reception of the disease by infection be positively disproved. A further argument adduced by Murchison is the fact that the relief of extreme destitution in districts where relapsing fever was prevailing has repeatedly been followed by the subsidence of the epidemic. But this may only show that starvation renders individuals more susceptible to the contagion, not that it is capable of generating it; and the same remark applies to the statement that the disease is never seen in private practice among persons belonging to the higher classes, who do not come into direct contact with the poor.

Relapsing fever prevails at various seasons of the year. It may attack persons of all ages, from five months old to seventy-five years, but the majority of cases are between the ages of fifteen and twenty-five. More males than females suffer from it, the reason probably being that a larger number of beggars, hawkers, and vagrants belong to one sex than to the other.

It is only at the commencement of relapsing fever that its *diagnosis* in ordinary cases presents any difficulty. Murchison remarks, that during the first two days, one may be scarcely able to distinguish it from smallpox; but in consequence of the frequency with which it prevails epidemically at the same time with typhus, the early differentiation of these two diseases,

so unlike one another in the danger which they entail, is of special importance in practice. I have already insisted on the greater rapidity with which the temperature and the pulse rise in the less severe form of fever. How useful for diagnostic purposes the detection of the spirochætæ may be, has been shown by Dr. Carter, at Bombay, where there was often difficulty in distinguishing the effects of malaria from the more continued varieties of relapsing fever, corresponding with the "bilious typhoid" of German writers. This observer states that so multiform were the phases and degrees of the spirillum fever that about 25 per cent. of his cases could properly be termed irregular.

On account of the contagiousness of relapsing fever, it is important that patients should be taken to a hospital as early as possible. They should always be placed in wards set apart for the purpose, since Litten seems to have shown that the acccumulation of a large number of cases together neither augments the mortality of the disease, nor increases to an appreciable extent the danger of its spreading to medical attendants and nurses.

The *treatment* is not so successful as might be hoped, for we have as yet no means of preventing the relapse. With this object, quinine and arsenic have been freely employed, both in England and in Germany, but in vain. At Breslau, carbolic acid, and in America, the sulphites, the hyposulphites, and the preparations of chlorine have been used with no better result. It remains to be seen whether the salicylate of soda, or the sulphocarbolates, or any of the drugs which have recently been introduced into the practice of "antiseptic surgery," may be more effectual. According to Murchison patients often find great comfort from frequent sponging of the surface with cold or tepid water, and from cold affusion to the head. An emetic at the first onset of the disease affords much relief to the severe pains in the hypochondriac regions. Throughout the febrile attacks, castor oil or some other mild aperient, should be given when necessary. Murchison also recommended the systematic administration of diuretics, by which he believed that it was often possible to prevent one of the chief dangers of the disease, the supervention of uræmic poisoning. He directed that from one to two drachms of nitrate of potass, one drachm of dilute nitric acid, and half a drachm of tincture of digitalis should be taken in divided doses in the twenty-four hours. He allowed rather a liberal supply of food, and to persons beyond middle age he administered stimulants about the period of the crisis, especially when he suspected that the heart was weak. He often found it necessary to give opium for the relief of headache, or of the muscular and arthritic pains which cause so much distress, but states that the hydrate of chloral proved a useful substitute for it. Lebert recommends frictions with a liniment of chloroform and oil for temporary alleviation of the pains in the limbs.

ENTERIC FEVER.

A CONTAGIOUS DISEASE—ALWAYS PRODUCED FROM A PREVIOUS CASE—METHODS OF CONTAGION ; BY AIR, WATER, ETC. ILLUSTRATIVE CASES—PREDISPOSING CAUSES : SOIL-WATER, CLIMATE, AGE—MORBID ANATOMY ; HISTORY OF THE DISTINCTION OF TYPHOID FROM TYPHUS FEVER—INCUBATION, SYMPTOMS AND COURSE—CONVALESCENCE—COMPLICATIONS—SEQUELÆ—PROGNOSIS—TREATMENT.

Within the last few years our knowledge with regard to the ætiology of Enteric Fever has made great advances. We are not, indeed, yet acquainted with its actual exciting cause ; but many of the chief details as to the way in which it is propagated have been positively determined, certain theories concerning its nature have been no less decisively negatived, and thus we can now limit somewhat narrowly the scope of future researches.

Origin of the Disease.—It has sometimes been said to be an *endemic* malady, but although it may prevail in certain districts rather than others, and may even remain limited to them, there could be no greater mistake than to suppose that its diffusion is, like that of ague, independent of the movements of human beings and of their intercourse. I shall presently adduce numerous instances in which an outbreak has followed the entrance into a place of a patient suffering with this fever, although for months or years there had not been a single case there.

Among the most famous examples of such an occurrence are those recorded by Dr. William Budd, of Bristol, in his well-known paper on the epidemic in and near North Tawton, Devonshire, during the autumn of 1839. Three persons left that village after having taken the fever. Two of them went to Morchard and gave it, one to his two children, the other to a friend, and he again to his two children and to his brother. The third went to Chaffcombe, seven miles off, where ten others were attacked in turn, and two of these carried the disease to fresh places, with the result that several more cases occurred at each of them.

Dr. Budd tells this story as if direct infection from the sick to the healthy had been at work, at least in some instances. Thus the friend of the second patient at Morchard is described as having been called upon to assist in raising the sick man in bed, as having been overpowered by the smell from his body, and as having felt very unwell from that time. In 1875 Sir William Jenner, in his Presidential Address to the Clinical Society, stated that he had twice known enteric fever contracted by students who diligently took temperatures, before the registering thermometer was in use, so that they many times a day put their heads almost into the beds of patients suffering under the disease. In 1879 Dr. Collie, of the Homerton Fever Hospital, expressed his belief that certain cases which were among the attendants at that institution were caused by direct infection, emanating either from the freshly-passed evacuations of patients or from their lungs or skins.

But striking as such occurrences naturally appear to the individual observer who watches their progress, there are the strongest possible grounds for rejecting this interpretation of them. Murchison tells us that during

nine years, from 1861 to 1870, cases of enteric fever were treated in the same wards of the London Fever Hospital with various non-specific febrile complaints, to the number of 3555 of the former class, and 5144 of the latter. The same night chairs were used by both sets of patients, and the employment of disinfectants was unexceptional. Yet enteric fever was not contracted by one of those who were under treatment for other diseases. In the *British Medical Journal* for 1879, Dr. Shirley Murphy has brought down to 1878 the experience of this hospital in regard to the occurrence of enteric fever among the nurses and other attendants. During twenty-four years only nineteen persons engaged in the institution were attacked by it. "Of these, ten were in no way connected with the enteric-fever patients or the enteric-fever wards. Of the other nine, one was a laundry maid, whose duties would bring her into contact with the soiled linen of the patients, but who otherwise was not in contact with them. Of the remaining eight there were special circumstances connected with drainage which would probably account for fever, leaving four for whose attacks there is no explanation given." In this time 5569 patients with enteric fever passed through the wards. Is it conceivable that if the disease were directly infectious there would not have been more numerous instances of its spreading? In other buildings one often cannot trace the origin of enteric fever to sewage emanations or to its other known causes, and the same thing must necessarily sometimes occur in hospitals. Some writers have brought forward instances in which two or more cases of enteric fever have arisen successively in the same house, as indicating that it is directly infectious; but much caution is required in drawing such a conclusion on account of the long duration of this disease, which allows abundant time for the production in other ways of new cases, while the original one is still running on. Murchison cites, but without attaching very much importance to them, a few instances in which nurses contracted the disease immediately after having been attending upon patients suffering from it, but most of them lose much of their force when it is remembered that the patients probably lay ill under the nurses' care for three or four weeks. It is obvious that the fact of a nurse taking enteric fever who is engaged with a patient laboring under this disease is not even presumptive evidence of direct infection, unless the patient is away from the place in which he himself became attacked. So, again, the circumstance that several inmates of a house are affected in tolerably rapid succession proves nothing, unless the first case was introduced from elsewhere.

Dr. William Budd, although he believed all the emanations from cases of enteric fever to be contagious, made it his chief object to show that the intestinal discharges were incomparably more virulent than anything else. He seems to have thought that an important argument in favor of such a view could be based upon an analogy which he drew between the specific cutaneous eruption of a contagious fever and the lesions in this disease which affect Peyer's patches and the solitary follicles. To express this very relation, the word *enanthem* had been already coined by German writers as a correlative to the term *exanthem;* and the doctrine propounded by Dr. Budd appears to have been previously taught at Munich by von Gietl. It is now widely adopted. But in order to account for the fact already stated, that nurses do not take the disease, notwithstanding that they come frequently into contact with the stools of patients suffering from it, an additional theory has been promulgated: that fresh typhoid stools do not contain the poison, which is afterward developed in them. We shall hereafter see that exactly the same thing is known to be true in the case of cholera; in that disease the "rice-water" discharges have been proved to be innocuous when first voided, and to become virulent subsequently. I may remark, in passing, that if the poison of enteric

fever is specific, the theory of its being evolved outside the human body after an interval, necessarily implies that it must be a living organism, and not a mere chemical substance. Dr. Cayley, in his "Croonian Lectures" for 1880, expresses his belief that this change may occur within twelve hours; for in the Middlesex Hospital patients have apparently caught enteric fever from using closets in which pans were placed containing stools from other cases set apart for the inspection of the physicians. Probably the development of the poison may occur even in fecal matters smeared upon linen or sheets, for washerwomen have often been observed to take the disease after washing the clothes and bedding of patients, having perhaps inhaled particles of the dried fæces, which had become detached and suspended in the air. Biermer, in one of the "Clinical Lectures," published by Volkmann, says that he has met with several instances of this. Dr. Cayley mentions two cases which occurred in patients already in the wards of the Middlesex Hospital, and which were traced to emanations from dried discharges upon the bedding of a typhoid patient in a neighboring bed. Dr. Murchison relates the case of a woman who brought to her house in Warbstowe, on the Cornish moors, the bedding of a sister who had died of enteric fever at Cardiff, in Wales. She remained free, but her sister, who was employed in hanging out the clothes, took the disease; and it spread from her as a centre.

Murchison, although he teaches that the stools of patients suffering under the disease are at first incapable of propagating it, and become so only when they have undergone a change, takes a very different view from that of Dr. Budd. According to him, the change in question is not the development of a specific poison, but a decomposition, to which typhoid are more prone than healthy fæces, on account of their being alkaline and containing ammonia and triple phosphates in abundance.

It is almost a necessary part of his theory, that it should be possible for the intestinal discharges of persons who are not affected with enteric fever to be decomposed in a precisely similar manner, and so to give rise to the disease *de novo*. He was, indeed, so strongly convinced that this was of frequent occurrence, that he at one time proposed the name of *pythogenic fever* (*πύθομαι* = *putresco*) in place of the objectionable term "typhoid fever," which was then generally in vogue. Sir William Jenner also, in 1875, expressed his opinion that the weight of evidence was in favor of the development of the disease, in many instances independently of any specific poison derived from previous cases. The main argument adduced by both these observers is that it is so generally impossible, at the commencement of an outbreak, to trace any probable, or even possible, source from which the specific poison could have come. Jenner cited the case of a young lady who, being an invalid, had been confined to her own room in a detached villa, where she saw very few people, for some months before she was attacked with enteric fever. A sewer-gas odor was detected, and when the flooring was taken up a crack was found in the soil pipe of a water closet on the floor on which she slept. This was no doubt the cause of the disease, but for about two years, during which she had lived in the house, no one who was at all likely to have been the subject of enteric fever had used the water closet; and from the town drains it was cut off by new and efficient traps.

Murchison laid especial stress upon certain outbreaks of the disease in which it was traced to emanations from cesspools, or from choked-up sewers, having no communication with the drains. But the only one of his cases in which the attempt was made to show that the cesspool or sewer could not have recently had the specific poison introduced, in the fæces passed by some one suffering under mild enteric fever, was one which occurred at a school at Colchester. And even of this, all that is said is that

"there were no other cases of fever, before or after, in the rest of the Union."

Before, however, we can determine the real value of such observations, we want to know how long the contagion of the disease is capable of remaining undestroyed in sewege. If it be a living organism, which may germinate and multiply outside the human body, there is apparently no reason why it should not survive, under favorable conditions, for an indefinite period. A case in point is related by von Gietl. A man, who had acquired enteric fever elsewhere, brought it to a village. His evacuations were buried in a dung heap. Some weeks later, five persons engaged in removing some of the dung were attacked by the disease; their discharges were sunk deep in the heap. At the end of nine months it was completely cleared out by two workmen, one of whom fell ill and died. In such a case as that related by Jenner, one can imagine that the typhoid poison might have been lurking in some stagnant corner of the water closet or soil pipe from the time when, perhaps many years previously, some one connected with a former proprietor of the house suffered from the disease. That no one should have been attacked in the interval is sufficiently explained by the fact that this young lady was the only person who always remained on the same floor of the house, breathing the infected air both by day and night; possibly, too, her being an invalid and being confined to her own room may have rendered her more susceptible. Or, again, what is more likely than that a living organism, if it constitutes the exciting cause of enteric fever, should sometimes remain for years in a dormant state, multiplying itself just sufficiently to escape extinction; and then that, under the accidental supervention of more favorable conditions, it should suddenly undergo an immense development? Such an interpretation seems to be the only one applicable to a fact which Murchison himself adduces; namely, that he has seen single cases of enteric fever arising in the same house again and again, at intervals of a year or longer. Thus between 1849 and 1857 six cases were admitted from a certain house into the London Fever Hospital; one in June, 1849, one in October, 1851, one in February, 1854, one in November, 1855, one in November, 1856, one in July, 1857. It would surely be a remarkable coincidence that the disease should have been six times generated *de novo* in a single building during these eight years, or that its specific poison should have been six times introduced from without. But if the poison was there all along, perhaps undergoing an excessive development from time to time when the conditions were especially favorable to it, we can never, I think, exclude the possibility of its presence in any cesspool, or sewer, or water closet.

Again, there is the clearest proof that neither the inhalation of ordinary sewer gas, nor the drinking of water impregnated with ordinary fecal matters, sets up enteric fever. In an immense number of villages throughout England the water supply is exceedingly impure; and both in villages and in towns the drainage is often as bad as it can possibly be. Yet for years the inhabitants of such places escape the disease, until a case is introduced from elsewhere, and then an outbreak at once occurs. It is probably only under such conditions as these, when the channels for the entrance of the poison of enteric fever into the human body are already prepared, that there are seen such distinct indications of its being virulently contagious, as were recorded by Dr. Budd in the case of the outbreak at North Tawton. And, commonly, if the conditions are absent, no evil consequences follow the entrance of a case of enteric fever into a place. How frequent this is is shown by a statement of Murchison's, that in private practice more than forty instances had come under his notice, in which persons came to a house ill with enteric fever; but that in only two out of the entire number

was there any evidence that the disease spread further, and that one of these two was perhaps not really a case in point.

The poison of enteric fever may enter the human body in various ways:—

First, *it may be transported by currents of air*. Many instances have now been recorded in which the disease has been shown to have been caused by exhalations from drains, or sewers, or water closets. I may briefly cite a few of them mentioned by Murchison.

1. In 1857 six policemen were admitted into the London Fever Hospital from the Peckham police station. The drainage of the building was said to be in perfect order, but the men declared that they had often complained of dreadful odors in a room in which they sat. On investigation, one water closet was found to have no connection with the sewer, and to empty itself into an old well, situated immediately underneath a passage adjoining the room, and covered in only by flag stones. In this cesspool night soil had accumulated to a depth of more than ten feet. The fever ceased when its cause was removed.

2. In 1862, at Chatham, nine persons out of twelve who had occupied a newly-built house were attacked with enteric fever. The first person to suffer was the master, and he had for weeks complained of a bad smell in the cupboard of his dressing room. It turned out that between the syphon pipe of the water closet and the soil pipe below, there was a gap of several inches, which was plastered round with cement, the pipes being embedded in the parting wall of the house. The cement had cracked, and there had been an extensive leakage of fecal matters. The residents in the adjoining house also had noticed a foul odor whenever this water closet was used, and three of them were presently taken ill with the fever. After the defect was made good no fresh cases occurred.

3. In 1848 a formidable outbreak, of what appears certainly to have been enteric fever (although Sir Thomas Watson held that this was not the case), occurred in the School and Abbey Clositers at Westminster. Its distribution followed the line of a foul and neglected sewer, in which fecal matter had been accumulating for years without any exit, and into which the contents of several smaller cesspools had been pumped immediately before the outbreak began. It communicated by direct openings with the drains of every house in which the disease appeared except one; and the boys from that house were in the habit of playing every day in a yard in which there were gully holes leading from the sewer.

4. In August, 1879, twenty out of twenty-two boys at a school at Clapham were attacked with a disease which was believed to be typhoid fever; the only point adverse to this view was the rapidly fatal course which it took in two cases, one patient dying in twenty-three and another in twenty-five hours. Two days previously the boys had been watching the workmen engaged in opening and cleaning out a drain at the back of the house, which had been choked up for many years; it gave off a most offensive effluvium, and its contents were spread over a garden adjoining the playground.

Secondly, *it may be conveyed by drinking water*. The propagation of the disease in this way is of immense importance, on account of the very large number of persons who may be affected by it, whereas the action of sewer gas, when carried through the air, is necessarily limited to a comparatively small area. In their details the outbreaks of enteric fever that have been traced to impure water vary greatly; no two, indeed, are exactly alike. I must briefly refer to a few of them, and it will be well to begin with those in which the circumstances are least complicated. Such are, of course, the small epidemics that occur so frequently among the inhabitants of a village or a hamlet, who derive their water supply from one or

more surface wells, into which sewage finds its way through a porous soil.

1. At Wicken Bonant, in Essex, the disease prevailed in 1869, and Dr. Buchanan investigated its origin for the Privy Council Office. He found that there was a broad division among the people in regard to the sources from which they obtained their water. One hundred and eighteen persons used private wells, and among them was only one positive case of fever; eighty-eight drank the water of one well called the "parish well," and no fewer than forty of them were attacked. Now, this well was situated four or five paces distant from a brook channel which ran through the place. At the upper end of the village the brook always contained water; but lower down the channel was dry during the greater part of the year, the water being carried beneath the surface in a stratum of gravel, to reappear as a stream at the bottom of the village. That there was a direct communication between it and the parish well was evident from the fact that in times of flood, when the channel was full, the water in the well ran to a corresponding height and became discolored. On June 24th the first case of fever arose, in the person of a boy, who lived in a cottage situated about thirty-five yards above the well. He had much diarrhœa, and his stools were thrown, without being disinfected, into a privy which stood almost on the edge of the channel. At this very time the soil water in the village was falling, after abundant rains which had taken place a month before, and pools of water were to be seen here and there in the channel. A month after the boy's illness the persons who made use of the well began to fall ill with the fever. Can it be doubted that the well water became impregnated with the specific poison from his intestinal discharges? Previously, on May 30th, two cases of fever had been imported from London into a house, of which the sewer opened into the brook two hundred and fifty yards above the well. I think it is almost certain that the boy derived the disease from that source, although it is not at all clear why he should have been the only person to suffer, until he, in his turn, gave it to the other inhabitants.

2. At Page Green, in the parish of Tottenham, a great many cases of enteric fever occurred in 1864 and 1865. Dr. Seaton investigated the matter and found that whereas there was, to some houses, a supply of water from the works of the Local Board of Health, the occupants of many other houses drank water from shallow surface wells. In three instances, in consequence of the families having removed from the place, he could not learn from which source the drinking water had been taken, but in all other cases, with the single exception of one child, it was ascertained that those who were attacked had used well water. Some of them had had the water of the Local Board distributed to their houses, but had been in the habit of borrowing water from their neighbors' wells, because it was bright and pleasant, whereas the other was hard, turbid, and red, from rust in the pipes, in consequence of its supply being intermittent and irregular. When, however, the well waters were analyzed by Prof. Miller, he declared them to be quite unfit for dietectic purposes.

3. At Terling, in Essex, between the beginning of December, 1867, and the end of February, 1868, there occurred an epidemic of enteric fever, upon which Dr. Thorne reported. It was of extraordinary severity; amongst a population of nine hundred persons, at least two hundred and sixty were attacked during the first two months; there were in all forty-one deaths, and so panic-stricken was the village, that it was necessary to discontinue the tolling of the church bell at death or funeral. Whether the disease was introduced from elsewhere could not be ascertained, for there had been isolated cases during previous years. But the extension of it was clearly

traced to contamination of the drinking water by sewage. The cottages were supplied, singly or in groups, by shallow surface wells, sunk in a loose and porous gravel. Round about them, but at a higher level, there were numbers of manure heaps, cesspools, and privies, the ordure from which was often spread out for yards over adjacent fields. During the autumn the water in the wells had been unusually low; doubtless, therefore, the filth had accumulated in the soil; so that it was washed into the wells in very large quantities, when, towards the end of November, a sudden rise of water took place. Among seventy-one persons, living on the outskirts of Terling, who procured their water from ponds, only six cases of fever occurred; and in all but two of these cases in individuals who had not been in the habit of frequenting the village.

It is often impossible to trace the source from which the drinking water derived the poison, but we have seen that there are slight forms of enteric fever, the real nature of which is never suspected. Thus a person who seems to be suffering under a trivial ailment, or who even appears to be quite well, may sometimes introduce the disease. Dr. Cayley cites three cases of which the origin was very clear.

4. At Over Darwen the water pipes were leaky and the soil through which they passed was soaked at one spot by the sewage from one particular house. No harm resulted until a young lady with the fever was brought to this house from a distant place; within three weeks of her arrival the disease broke out and fifteen hundred persons were attacked.

5. At Calne a laundress occupied the middle one of three houses supplied by one well, into which the slops of her house leaked. She received the linen soiled by the discharges of a case of enteric fever, and after fourteen days cases occurred in all those houses.

6. At Nunney a number of houses got their water supply from a foul brook contaminated by the leakage of the cesspool of one of the houses, but no fever showed itself until a man with the disease came into that house from a distance. Then in about a fortnight an outbreak took place in all the houses.

Still more interesting are certain outbreaks of enteric fever which have been traced to contamination with fecal matter of water supplied by pipes; and their importance is the greater, because similar occurrences are likely to be increasingly frequent, as water companies become multiplied.

7. In Caterham, during the fortnight which ended February 2d, 1879, there occurred forty-seven cases of the disease; and, at the same time, no fewer than one hundred and thirty-two cases were observed at Red Hill, eight miles distant. In each town the persons attacked were using the water furnished by the Caterham Waterworks Company, but considerable numbers of persons who derived their supply from other sources escaped altogether. On the other hand, cases occurred at the Earlswood Asylum and in other places which also used the Company's water. Now, this water is drawn from chalk wells more than 500 feet deep, and it had a deservedly high reputation for wholesomeness. Every point in regard to its sources, storage and distribution was carefully inquired into, but for a long time in vain. At last, however, the attention of Dr. Thorne was drawn to the fact that, in January, 1879, the Company had been constructing an adit, at a depth of 455 feet, from one of their old wells to a new bore which was then being sunk. A number of men had been employed upon this duty, and one of them it was found had been ill and had left work, in the course of the month. He was sought out, and on inquiring, it appeared clear that he had been suffering from a mild attack of enteric fever, which began on January 5th, and which he had probably acquired at Croydon, where he had spent December 25th and 26th. He had much diarrhœa, the bowels acting at least two or three times

during each shift of eight or twelve hours, and in accordance with the usual practice under such circumstances he made use of the buckets by which the excavated chalk was being raised to the surface. He denied that he had ever relieved himself in the adit, without waiting for a bucket, but that this had been the case was very probable. It seems to be almost certain that in some way his fæces passed into the water of the well in which he was working and gave rise to the epidemic. The poison must have been diluted to an extraordinary degree.

8. But in this almost infinitesimal subdivision of the contagion of enteric fever, no epidemic seems to approach that which occurred at Lausen, in the Valley of Ergolz, in the Jura, at least if its origin was correctly interpreted. Early in August, 1872, 130 out of a population of about 800 persons were attacked, all of whom used the water of a public fountain. This fountain was fed from two sources, one being a spring into which it was known that water would penetrate by percolation from certain meadows in another valley, separated from the Ergolz Valley by a mountain, the Stockhalder, through the base of which the water must therefore have passed. Now, in that other valley there were, in July, 1872, two cases of enteric fever, the discharges from which were thrown into the stream that traversed it. In the middle of the month the water of this stream was used to irrigate the meadows, and three weeks later the epidemic at Lausen began. But I must confess that it seems inconceivable that the effects of a poison should be traced after it had been thrown into a running stream, and after the water taken from the stream had been poured out over the ground, to make its way by subterranean channels into a distant spring!

9. At Guildford, in September, 1867, a severe epidemic of enteric fever occurred; within ten days, 150 cases came under treatment, and the number reached 264 by the end of the month. A singular circumstance with regard to this outbreak was now observed, namely, that it was almost restricted to the higher parts of the town, to which water from a new well had been distributed by engine power, after having been first stored in a reservoir. Certain exceptions, in school children and others who resided where water derived from other sources was used, was easily explained by the fact that those persons spent their days in houses supplied by the high-service water. Now, it was known that this new well was fed not merely by percolation, but by a fissure in the chalk; and that fecal matter might easily find its way into it was probable, from there being within ten feet of the well a sewer, into which water closets were drained, and cesspools and privies discharged their overflow. These facts were so striking, that Dr. Buchanan, who investigated the matter on the spot, was at first perplexed when he was told that from the beginning of August, in consequence of the engine having broken down, the high-service water had not been taken from this source at all, but from another well, the old well, which also supplied the lower parts of the town, where there was no fever. But, on further inquiry, it turned out that on one particular day, the 17th of August, the water wheel which was used to charge the high-service mains being under repair, they were filled with some water which had been raised from the new well on or before the 1st of August and had in the meantime remained in the high-service reservoir. Subsequently the sewer above mentioned was ascertained to have been leaking in various places, so that the surrounding soil was a quagmire of dark-colored, fetid slush, which made the men vomit who had to dig it out.

10. At Sherborne, in Dorsetshire, enteric fever became epidemic at the beginning of the year 1873, and prevailed severely until the middle of April. Dr. Bloxall, who went down to inquire into the matter, found reason to conclude that the drinking water, which was delivered from a reservoir through pipes, and which was believed to be originally pure, became contaminated in

a way which would not at first sight have been obvious. In December, 1872, and in January and February, 1873, the water was frequently shut off from the town, at a point near the reservoir. Now, it was known that when the water was thus shut off a rush of air would take place *into* certain delivering pipes as soon as their taps were turned on; but many of the mouths of the pipes were situated in the pans of water closets, consequently if a tap was broken, or if a person forgot to turn it off when he found that it gave exit to no water, the corresponding pipe might continuously suck up sewer gas, or even liquid excrement, supposing the water-closet pan to be full. Then, when water was again delivered, this would wash away whatever particles might have been deposited in the pipes and convey them to be drunk by the people of the town.

11. At Caius College, Cambridge, a local outbreak of the disease occurred in November, 1873, which was traced by Dr. Buchanan, with very strong probability, to a precisely similar origin. Twelve out of fifteen cases in students at the college were among the sixty-three residents in Tree Court, a part of the building which had been erected only four years previously, with every care as to sewers, drains, and water pipes. Now, Tree Court had an independent water supply direct from a high-pressure main. This supply was intended to be constant, but there had, in fact, been a complete intermission of it on two occasions shortly before the outbreak. After such intermissions the water had been noticed to come in with a rush, "like soda water," evidently in consequence of its having become mixed with air, which had been sucked up into the pipes. Within the Tree Court buildings there were two water closets, one in the basement of the porter's lodge, the other on the first floor of one of the staircases. The tap of the lower one, or that over an adjoining sink, if left open during the intermission of water supply from the main, would have allowed water to drain from the whole pipe system of the court; that of the upper one would, under such circumstances, have permitted of the free entrance of air. This air, however, would have been mixed with sewer gas from an unventilated sewer in Trinity Street, which, at the very time under consideration, was receiving the excreta of patients ill with fever in other parts of the town. The effect of recharging the pipes with water must necessarily have been to distribute sewer gas in solution to every part of the building. It was, indeed, positively ascertained that not merely air, but water impregnated with fecal matter, had been sucked up into the supply pipe of the upper water closet, for that pipe was lined with a brownish deposit, containing phosphoric acid and a large portion of intermixed organic matter. The obvious method of preventing the occurrence of such outbreaks of fever as those at Caius College and at Sherborne is for the sanitary authorities, whenever the water supply is intermittent, to insist upon there being a proper service cistern for each water closet.

Thirdly, *milk may be contaminated with the poison of enteric fever.* This is clearly shown by the following remarkable instances:—

1. The first epidemic that was traced to such an origin occurred in Islington, in 1870. It was investigated by Dr. Ballard. Between July 3d and September 10th the occupants of sixty-seven houses were attacked, one hundred and sixty-seven individuals, of whom twenty-five died. It was a most remarkable circumstance that the district affected was included in a semicircle, with a radius of a quarter of a mile, drawn immediately on the north side of the line of the North London Railway from a centre almost upon this line. There was no fever in the area contained in the corresponding semicircle south of the railway, which here passes through a cutting. This, of course, at once suggested that human intercourse was in some way concerned in spreading the disease. The right clue was first hit

upon by a lady, whose family was attacked; and a little inquiry convinced Dr. Ballard that, far-fetched as such an idea had appeared, there was much probability in it. The milk vendor whose milk was suspected had himself fallen a victim to the epidemic, but his father, greatly to his credit, readily consented, when applied to, to give a list of the customers. It was then found that the dairy supplied one hundred and forty-two families, a very small portion of those who lived within the semicircle. In no less than seventy among the hundred and forty-two families there had been cases of enteric fever. The way in which the disease picked out the customers of the dairy in particular streets and rows was most striking. In one long road and a street running from it the milkman supplied three families; two of them were affected. In a crescent with twenty-five houses he supplied four families; they were all attacked. In a new neighborhood, where there were about seventy houses, he supplied four families; three had the disease. In a square with fifty-nine houses he supplied four families; all had it. On the other hand, there were scarcely any cases among those families who had invariably bought their milk from other sources. As might have been expected, women and children were attacked in much larger numbers than men, who drink comparatively little milk. The source of infection was traced, with much probability, to the water of an underground tank in the cow-yard. When this was cleaned out, the woodwork in one corner of it was found to have been broken away, leaving a gap, from which a rat burrow passed into two old drains. Sewer gas had no doubt entered the tank along this channel, and it is quite possible that liquid sewage had taken the same course a few months before the outbreak, when the ground had been disturbed to lay a drain pipe for some neighboring houses. It could not be ascertained that water from the tank had ever been used to dilute milk, but the pails were washed out with it, and some of it might have been left in one of them, by accident. One of the customers had several times complained that the milk was poor, and that when kept it not merely became sour, but stank.

2. In the summer of 1872 an outbreak of enteric fever took place at Armley, near Leeds. This also was investigated by Dr. Ballard, who found that with a single exception all the early cases occurred in families supplied by a particular dairyman. After the first three weeks this rule was less strictly observed, the reason, doubtless, being that the disease then began to spread in other ways, since the privies, cesspools, and drains in the place were in the most neglected and offensive condition. That water from a pump on the premises of the dairyman had been the means of infecting the milk, was rendered most certain, from the fact that, the handle of this pump having been chained up on July 10th, the fever abruptly ceased to appear in fresh families among the customers about a fortnight later. The way in which the poison had entered the well beneath the pump was also satisfactorily made out. During the month of May the dairyman himself had been ill with enteric fever; toward the end of the month there was a good deal of rain, and this, no doubt, washed into the well fecal matters which had escaped previously into the soil from the privy or from defective drains. On inspection of the well black matter was found to be oozing into it, and at the bottom there was a deposit of filth and mud which gave off bubbles of gas when disturbed. A point on which Dr. Ballard relies as corroborating his conclusion that the early part of the outbreak was due to infection by milk, is that it was only during that period that multiple cases occurred in the same families.

3. In the summer of 1873 an outbreak of enteric fever occurred in St. Marylebone, and in certain parts of St. George's (Hanover Square) and of Paddington. It affected chiefly the households of well-to do people,

and among others the family of Dr. Murchison, who quickly became convinced that the only probable mode of introduction of the disease into his house was by the milk supply. This was confirmed by a minute investigation made by Mr. Netten Radcliffe and Mr. Power. It was shown that nine-tenths of the two hundred and forty-four cases to which the inquiry extended were in households which consumed milk from a particular service of a particular dairy. Certain ramifications of the same milk supply extended to the east end of Regent's Park, to Belsize Park (Hampstead), and St. Anne's (Soho), and in these districts also enteric fever occurred among the consumers. There was a special incidence of the disease upon women and children, and many striking instances are noted in which those members of a family were attacked who were in the habit of drinking milk, while others escaped who did not do so. The milk which appeared to convey the poison was a special kind, sold as "nursery milk," and taken from three or four cows, set apart for the purpose, at Chilton Grove Farm, in Buckinghamshire. Now, on the 8th of June the occupier of this farm had died in the fourth week of an attack of enteric fever. His evacuations, instead of being thrown into the common privy, were buried in an ash heap outside the farm buildings. Subsequently, however, it turned out that this was the very worst thing that could have been done with them. For there was a well close by, the water of which was used for dairy purposes, although not for drinking or cooking, as it had been noticed to have a disagreeable taste. Excavations made for the purpose showed that there had been a line of soakage into the well, along the foundations of a wall, of the filth from a pig sty which formed a pool in immediate proximity to the ash heap above mentioned. If due intervals are allowed for the gradual penetration of the matters containing the poison of enteric fever through the soil, and for the incubation of the disease, the date at which the outbreak in London began—during the last days of June and the first days of July—corresponds exactly with this theory of its origin.

Fourthly, *there is some reason to believe that meat may under certain circumstances convey the poison of enteric fever.* At Kloten, near Zürich, six hundred and sixty-eight persons were attacked in July, 1878, all of whom had partaken of some veal provided for the festival of the choral societies, and derived from various sources, but partly from two diseased calves. Huguenin thinks that one of these calves was actually suffering from an epizoötic complaint equivalent to enteric fever, but that its flesh might have been eaten with impunity, if it had not been in a state of putrefaction, the contagion being, as he supposed, developed after death. The liver and the brain of this animal fell to the share of persons who did not go to Kloton, and they also fell ill with the fever. A few other outbreaks, also attributed to diseased meat, are referred to by Dr. Cayley.

I may take this opportunity of remarking that it has hitherto been considered an open question whether the lower animals are liable to enteric fever. The so-called typhoid fever of pigs is now known to be an entirely different affection.

It must not be supposed that it is always, or even generally, possible to trace to their source isolated cases of enteric fever, especially when they occur in large towns like London. Probably there are many chances of infection, from sewer gas in air, and from impurities in water, which no inquirer, however acute, could trace out. The number of cases admitted into the London Fever Hospital varies from year to year much less than might have been expected. Dr. Cayley thinks that they are not dependent upon the presence of the contagion of the disease in water taken from the Thames. Although there can be no doubt that the poison frequently passes into the upper part of the river from the towns and villages

on its banks, he thinks it is always destroyed by exposure to the air and by other agencies, among which vegetable life is probably one of the most important.

Soil Water.—Before leaving the question of the immediate causes of enteric fever, I must briefly mention a view held by certain German professors, according to which the fundamental point in the ætiology of the disease is its relation to periodic fluctuations in the level of the soil water. In speaking of cholera I shall have to discuss a similar theory, based upon observations made by von Pettenkofer at Munich, from the year 1856 onward. It was, however, Buhl who applied these observations to enteric fever; he showed that when the soil water in that city (as measured by the depth of water in the surface wells) is falling, the number of cases of enteric fever increases; when it is rising, the number of cases diminishes. That the facts really are so is generally admitted, but the interpretation which was at first put upon them is, I believe, rejected by every one qualified to offer an opinion. It was that the falling of the soil water enables air to penetrate more deeply into the ground than before, and so brings about changes in the organic matters there which result in the giving off of a poison that sets up the disease in persons exposed to it. But this is obviously inconsistent with everything that is known as to the way in which enteric fever spreads; and I cannot doubt that Liebermeister and Buchanan are right in supposing that the soil-water observations simply illustrate its communication by means of drinking water. Not only is the water contained in surface wells generally more impure when the level of the soil water is persistently low, but there is far less movement of it in a horizontal direction towards its natural outlets in brooks and streams, so that any noxious matters in it accumulate and acquire an increased virulence. It must also be added that in no other place except Munich has a fixed relation been found to obtain between the soil water and the spread of enteric fever. In the case of particular epidemics, as notably at Terling, in 1868, the disease had broken out with great severity precisely when the wells have been high.

It is certain that *climatic influences* greatly affect the prevalence of enteric fever. In the London Fever Hospital there have been far more admissions during dry and hot summers (*e. g.* 1865, 1866, 1868, 1870) than in damp and cold summers (*e. g.* 1860, 1872); but it is natural to take this in connection with the fact that each year there is an increase of the disease during the four months from August to November, while its frequency falls from March to May. Similar observations have been made in Berlin and in Basle, and there can be no doubt that the cause is the heat of summer on the one hand, and the cold of winter on the other hand, the effect being, however, not immediate, but retarded by two or three months.* Such conditions play but a secondary and intermediate part in the ætiology of the disease: their effect is merely to favor, or to hinder, the operation of its real causes.

Age.—Certain circumstances remain to be stated, which affect the disposition of individuals to take enteric fever at particular times. Chief among these is age. The disease is far more frequent in persons between fifteen and thirty than in those who are either younger or older. In babies it is very uncommon, but in 1864 Murchison showed at the Pathological Society the intestine of an infant six months old who had been attacked at the same time with her mother. During childhood the liability to the disease increases from year to year, but I think it may be a question whether this does not depend upon an augmented exposure to its exciting cause. After the age of twenty the

* At Munich the influence of season seems to be reversed, the maximum prevalence of enteric fever being in February; but Liebermeister suggests that this, after all, may be but the result of a still greater retardation of the same action which obtains elsewhere.

liability begins to decline, after thirty more rapidly, and beyond forty very few cases occur. However, I remember seeing enteric ulcers in the body of an old woman of seventy, examined by Dr. Wilks; and some foreign observers have recorded instances in persons aged seventy-two, eighty-six, or even ninety. Liebermeister gives a tabular statement of the proportion of cases at varying ages at Basle, corrected according to the numbers of persons at the corresponding ages in the population generally. This, of course, diminishes the differences very considerably, and it seems to be possible that what remains of them may be due to the protection acquired by having already passed through the disease.

There does not appear to be any constant predominance of one sex over the other among patients suffering from enteric fever. Liebermeister maintains that it is more apt to attack strong and healthy persons than those who are sickly and delicate; and there seems to be a certain degree of immunity against it among women in pregnancy, after labor, and during lactation. Several French writers have declared that students, servants, and foreigners are especially liable to contract this disease when they first come to live in Paris; and Murchison has shown that more than 6 per cent. of the patients admitted into the London Fever Hospital have arrived in London within three months. This cannot merely indicate, as Trousseau suggests, that such persons are devoid of protection from their not having already passed through enteric fever. But, on the other hand, it may not necessarily prove that an acclimatization occurs as the effect of longer residence; it may merely show that certain individuals are so very susceptible to the poison as necessarily to succumb as soon as they are exposed to it.

History of the Recognition of Enteric Fever.—Almost at the beginning of the present century, the study of morbid anatomy in Paris was rewarded by the discovery that in the bodies of those who died of fever in that city the intestines were inflamed and ulcerated. The true bearings of such observations were not, however, at once fully appreciated. Broussais and others maintained that a "gastro-entérite" was the essential cause of "fever." Presently the suggestion was made by Serres and Petit that the intestinal lesions were specific, and resulted from the introduction of a poison into the system, and that they were of an eruptive nature, like the pustules of smallpox. It was reserved for Bretonneau, of Tours, to point out, in 1820–29, that they were specially localized in the solitary and the agminated follicles of the ileum. He endeavored to introduce the name "dothiénentérie" (*δοθιήν*, a pustule, and *ἔντερον*, intestine) for the fever in which they occurred. This clumsy term was used by his pupil, Trousseau, but it never met with general acceptance. Unfortunately, much greater success attended a proposal made by Louis, in 1829, to designate it "fièvre typhoïde." This proposal was, at least in England, universally adopted; and it greatly impeded a right understanding of the matter. Up to that time, all the French physicians who had written upon the intestinal affection had assumed that the same lesion would be found in every form of the disease known by the various names of typhus, putrid fever, jail fever, etc. But when the morbid anatomy of patients who had died of these varieties of fever came to be investigated in Scotland and in England, the bowels were often found to be healthy. For a time the difference was supposed to be accidental, or of little consequence. But gradually it became apparent that two distinct diseases were being confounded with one another. The credit of indicating the clinical distinctions between them is divided by Murchison among a number of observers; Perry, of Glasgow (1836), H. C. Lombard, of Geneva (1836), Gerhard and Pennock, of Philadelphia (1836), Shattuck, of Boston (1839),

H. C. Barlow, of London (1840), A. P. Stewart, of London (1840). Louis, in the second edition of his work, published in 1841, expressly announced that his "fièvre typhoïde" and the typhus of English writers were very different. There were, however, many who maintained the opposite view, that the two forms were identical. The ultimate issue of the controversy still appeared uncertain when, in 1849–51, Jenner published a series of researches, based upon observations made at the London Fever Hospital. The peculiar value of his papers lay in the proofs which he brought forward of the fact that the different forms of fever owe their origin to distinct specific causes. During two or three years he investigated with great care every instance in which more than one patient was brought from the same house; and he found that the later cases invariably corresponded in character with the first one, even where both kinds of fever were prevalent in London at the time. All subsequent writers whose opinions carry weight have adopted the doctrine for which Jenner contended, and there no longer remains any doubt about the matter. Within the last few years, however, Louis' name of "typhoid fever" has in this country been generally discarded. It could in no case have been appropriate, since the object in giving a title to a disease really distinct from typhus, but liable to be mistaken for it, is to mark the difference between them, rather than the resemblance. But, further, the very same word has long been fitly used to designate a group of symptoms, consisting of stupor, with low delirium, a dry, brown tongue, sordes, etc., which are really *typhoid*, since they are like those which are seen in typhus. The two meanings necessarily led to much confusion. The term *enteric fever*, or *enterica*, as Dr. Wilks proposes, has therefore been substituted, and it appears to me to be very suitable, as indicating the fact that it is characteristic of the disease to be attended with intestinal lesions, though not, indeed, always with intestinal symptoms. Murchison proposed as an alternative name "pythogenic fever;" but this has met with very little support, and it involves an erroneous ætiological theory.

It has naturally been a point of great interest to inquire whether the presence of enteric fever can be recognized in the descriptions given by medical writers in former centuries; and the answer is decidedly in the affirmative. In John Hunter's Museum there still are two preparations showing the characteristic intestinal lesions; and a well marked case was dissected by Morgagni. It appears, too, that the disease is identical with the "slow, nervous fever" which Gilchrist, of Dumfries, described in 1734, and which Huxham, in 1738, distinguished from "putrid malignant" fever. In works of a still earlier date, it must probably be looked for rather among the "remittent fevers," attributed to the same cause as that of ague. The mistake of confounding enteric fever with malarial fevers was commonly committed in the marshy districts of England up to a very recent period; and in India they are even now separated with difficulty. These facts would in themselves suggest that in former times, when the marsh miasm was much more widely spread than at present, the distinction would fail to be recognized; and an instance in point is probably to be found in the writings of Baglivi (1696) and Lancisi (1718), who recorded cases of fever at Rome in which the intestines were ulcerated, but which they identified with the "hemitritæus," *i. e.*, semi-tertian, a form of ague mentioned by Galen.

Characteristic Lesions.—No other acute specific disease is accompanied by a definite series of internal lesions, which, being peculiar to it, constitute the principal test of its presence in all doubtful cases that end fatally. It is on account of the importance of these lesions, and also because I wish to mark the distinction which is created by them between enteric fever

and the various diseases with which it is most closely allied, that I describe them, before entering upon the clinical symptoms and course of the fever.

The most important lesions are situated in *Peyer's patches and in the solitary follicles* in the small and large intestines. These first become injected and swollen, so that they project further above the level of the surrounding mucous membrane than is naturally the case, even in children. As a rule, they rise to the height of one or two lines only; but in some exceptional cases, according to Hoffmann, the patches become as much as three-eighths of an inch thick. In these structures, Louis and other French writers have described two separate forms of affection under the names of *plaques molles* and *plaques dures;* the difference between them is, however, merely one of degree, the hard ones being those in which the change is most intense and most rapidly developed. After a few days the redness passes off, and the diseased follicles become gray, and even white. So marked is their whiteness, sometimes, that they have been compared to a thin layer of a soft medullary or encephaloid new growth spread out beneath the mucous membrane. The microscope shows that there is a very abundant formation of new cells, which are considerably larger than leucocytes, have large nuclei, and often exhibit signs of multiplying by fission. These cells, besides distending the lymph follicles, infiltrate the intervening fibrous septa, and may even spread into the mucous membrane above, and into the interstices of the muscular coat and subserous tissue beneath. I have never seen them forming gray granules on the peritoneal surface, as is described by Hoffmann. But it is not uncommon for adjacent Peyer's patches to become fused together by an extension of the morbid growth, which thus may affect the whole circumference of the lower part of the ileum for some inches above the valve.

The next occurrence is generally ulceration. This seems sometimes to begin as an abrasion of the surface of the diseased solitary follicles or patches, and gradually to extend through their substance. But much more often the whole of the infiltrated tissue, or at least a large part of it, dies *en masse.* It then forms a soft, shreddy, flocculent slough, which is of a bright ochrey-yellow color, apparently from imbibition of bile pigment from fecal matter. Presently the slough is detached, either entire or in fragments. There is then exposed the floor of an ulcer, in which the transverse muscular fibres of the intestine are often plainly visible, and which is extremely thin and translucent when held up to the light. The muscular fibres themselves may undergo destruction to a greater or less extent, so as to lay bare the subserous tissue, to the imminent risk of perforation into the peritoneal cavity. The edges of the ulcer are thin, soft, and rounded; and when examined under water they are seen to be slightly undermined. Its form at first corresponds with that of the lymphatic organ in which it took its origin; if in a solitary follicle, it is small and rounded; if in a Peyer's patch, it is usually elongated in the direction of the axis of the intestine. In the latter case, too, its position is always remote from the line of attachment of the mesentery. In these respects, as well as in the characters of their floors and edges, the ulcers of enteric fever differ from tubercular ulcers. They never seem to show any signs of spreading by gradual destruction of their edges. And yet, at advanced stages of the disease, it not seldom happens that they are found to be elongated transversely to the axis of the intestine, their form being, in fact, the reverse of that which generally characterizes them. At length, their floors become covered with a thin, gray layer of granulation tissue, and they heal by the gradual growth of mucous membrane from their edges toward their centres. I find, in our records of autopsies at Guy's Hospital, five cases in which death has occurred from some other disease within a few

months after recovery from enteric fever; in two of them the patches were of a blackish color, with slaty margins; but in the others the cicatrices seem to have been white; and in one of them it was only on very close examination that a deviation from the normal appearance, consisting in a slight unevenness of surface, could be recognized. There is never any thickening or puckering of the affected tissues after this disease, so that it cannot lead to narrowing of the bowel.

Enteric fever by no means affects all the lymph glands of the intestines equally. In a large number of cases the solitary follicles entirely escape; in many others those of the ileum are alone attacked; and when those of the large bowel suffer, it is often only in the cæcum, or in the ascending colon; in a very few instances the morbid change extends even to the rectum. It seems, in fact, to spread from the ileo-cæcal valve as from a starting point. Precisely the same thing is observed with Peyer's patches; the highest of them are very seldom, if ever, affected. As a rule the lesion is limited to patches within two or three feet of the valve, and sometimes it does not reach beyond a few inches. In very exceptional instances the agminated follicles remain altogether free, and the solitary follicles bear the whole brunt of the disease. This apparent caprice of distribution in different cases cannot but suggest the inquiry whether there may not be some in which no glands suffer at all, so that, if the nature of the disease were otherwise doubtful, the criterion afforded by morbid anatomy would fail. The nearest approach to this, with which I am acquainted, is afforded by a specimen which I showed at one of the meetings of the Pathological Society in 1875. The only lesions in the intestine were the following: "One ill-defined purplish-red patch, of about the size of a shilling, situated a foot above the valve; and a little higher up another patch, presenting similar characters, except that in its centre there was a darker spot the size of a pea, with a breach of surface, visible only when it was examined under water." I think it is by no means unlikely that in mild cases of enteric fever, such as could never prove fatal except by some accident, the intestinal lesions are often very slight, and may, possibly, in rare cases be altogether absent.

Again, it seldom or never happens that at an autopsy all the diseased glands in a case of enteric fever are found in the same state. Those close to the valve are almost always the furthest advanced, and very often every stage is present, from clean ulcers below, through ulcers containing scattered shreds of slough, to those in which sloughs have just been formed; and from these, again, to patches which are but slightly excoriated on the surface or only swollen. Sometimes, indeed, the progression is not perfectly regular, a patch less affected being seen at a lower point than one which is more affected. Most pathologists think that these differences depend upon the fact that the follicles are attacked, not simultaneously, but in succession. Dr. Moxon, however, is of opinion that the morbid change is of less severity, rather than of later date, in the higher patches. He thinks that the greater tendency to destruction of those which lie at the extreme lower end of the ileum is due to the irritating contact of the intestinal contents, held back by the action of the valve upon them. Such a view, however, seems to me to be contradicted by the details of the differences which are observed, as I have stated them above, and I shall presently point out that in some of the more protracted cases of enteric fever there can be little doubt that the glands are affected successively and at considerable intervals of time.

But the swollen glands, instead of sloughing or ulcerating, sometimes entirely subside, as was long ago pointed out by Chomel and Louis. The original idea was that the morbid material in the follicles underwent softening

and that they ruptured so as to allow it to escape into the channel of the intestine. Hoffmann still says that this often occurs, and that the follicles become stained in consequence of there being at the same time a little hemorrhage, so that the Peyer's patches acquire a dotted appearance or exhibit, as the French term it, the *état pointillé*. I thought that this condition, which has also been compared to a shaven beard, had been proved to be at any rate not peculiar to enteric fever, seeing that it is not uncommon in those who have died from other causes and at all ages. But Hoffmann is still disposed to maintain that its origin is always in a former attack of the disease, which may have occurred so long ago as to have been forgotten. Its frequency in the bodies of those who have lived in Basle, where enteric fever is very prevalent, has led him to speak of it in joke as the *légitimation* for residence in that town. However this may be, there seems to be no doubt that subsidence of the swollen glands often takes place in a very different manner, namely, by disintegration of the cell growth in the follicles, and by its undergoing absorption like an inflammatory exudation. Among cases which end in recovery it is, perhaps, not uncommon for all the patches and all the solitary glands which are affected to take this course, but when death occurs at an advanced stage it very rarely happens that some are not found ulcerated. I have only met with one instance of the kind; the patient died on the twenty-first day, and the patches were found swollen, raised and reddened, but marked by irregular depressed lines and spaces so that they had a reticulated appearance. They were not "medullary" in character, as would probably have been the case if the affection had been early; there was no trace of sloughing or ulceration. I preserved the specimen in spirit for the Museum, but after a few weeks it showed nothing. Chomel described *plaques à surface reticulée* as a special modification of the enteric lesion, but since, as Hoffmann points out, reticulation indicates that the septa between the follicles are more swollen than the follicles themselves, and since the morbid change begins in the follicles and affects the septa secondarily, such a condition can hardly occur except as the result of their subsidence. As a rule, absorption is found to have taken place in certain patches only, or even in parts of them, while elsewhere there are sloughs or ulcers. Thus in 1862 Dr. Wilks examined a case in which, while the solitary follicles had each a depression in its centre, some patches appeared to be in a state of retrogression, parts of them only being slightly raised and nodular.

It is a matter of some practical importance to determine as accurately as possible the dates at which, reckoning from the commencement of the disease, one may expect to meet with the several stages of the intestinal lesions. According to Liebermeister, the first week is occupied by swelling and infiltration of the lymph glands of the bowel; during the second week they either slough or begin to subside; in the third week any sloughs that may have formed become detached, so that by the end of it all the ulcers have clean floors; during the fourth week they begin to heal, but the process is often not completed until a considerably later period. This statement is easy to remember, and for many cases it is probably accurate, but it certainly is not universally applicable in such a way that a pathologist should be able, from the appearances observed after death in a given case, to state positively the duration of the patient's illness. According to Trousseau, the swelling of the intestinal glands does not begin to appear until the fourth or fifth day; according to Chomel and Louis, not until the seventh or eighth day; but Bristowe and Hoffmann found them enlarged in cases fatal on the fifth and fourth days respectively; and Murchison relates one instance, that of a girl who died forty-seven hours after being suddenly attacked with vomiting and fever, in which the solitary follicles were of the size of hemp seeds or split peas, Peyer's patches being also similarly affected. But it seems to me very doubtful whether

the disease did not, in that case, begin earlier than its symptoms. The same remark applies to certain cases quoted by Murchison, in which sloughs with the usual characters have been found in patients who have been but a short time ill; and in 1871, a man died in Guy's Hospital, who had been definitely attacked twelve days before his death with shivering and aching pains in his limbs, so that he was obliged to go home, but his intestine contained extensive ulcers, all of which had clean floors except one, this having a few fragments of slough still adherent to it. Surely the lesion must have been in progress before his illness began. On the other hand, it is not uncommon, in cases fatal at the end of six or seven weeks of continuous fever, to find sloughs still adhering to several of the patches, while others are merely swollen and medullary looking. But probably the lesions are there of different dates, and have been developed in successive outbreaks at intervals of a fortnight or longer. In the case of a boy who had been ill for five weeks, and who died in Guy's Hospital, on October 26th, 1879, Dr. Goodhart describes the glands as fleshy looking, and as just beginning to ulcerate, so that he would have supposed the disease to be at about the eighth or the ninth day. Doubtless some other glands had been affected from the first but had afterwards subsided.

Another lesion in enteric fever has its seat in the *mesenteric lymph glands*. This does not appear to be absolutely constant; at least, I find, in the records of autopsies at Guy's, two cases, one of them fatal on the seventeenth day, in which the glands are said not to have been enlarged. It may be present at a very early period of the disease; thus, in Hoffmann's case, fatal on the fourth day, the glands were already of twice the normal size. That it results from the absorption by the lacteals of matters from the affected parts of the intestines is probable from the fact that, in many instances the lower glands, corresponding with these parts, are alone attacked; but sometimes the glands in the upper portion of the mesentery become also involved, and even (as in a case which I inspected in 1878) glands in the portal fissure, close to the liver. As a rule, the enlargement is moderate, the diseased glands not being larger than hazel nuts, but they may be as big as horse chestnuts, projecting conspicuously from the mesentery. Their substance is white, or gray, or pinkish, with spots of hemorrhage. Under the microscope they are found to contain large cells, some of which have two or more nuclei, like those in the intestinal follicles. Suppuration sometimes occurs, or partial sloughing, and under such circumstances a cheesy mass may result if the disease subsides. But I confess to feeling very doubtful whether Hoffmann is right in saying that caseation, or the deposition of calcareous salts, may form part of the ordinary retrograde process.

Wagner and other German pathologists describe the frequent occurrence of *minute gray nodules*—sometimes visible to the naked eye, sometimes microscopic—in the substance of the liver and of the kidneys. Hoffmann noted their presence in thirty-eight among two hundred and fifty cases. The only instance that I know of in which a similar observation has been made at Guy's Hospital was by Dr. Goodhart, in 1879, in the case of a girl, aged 17. If it stood alone, one might, of course, suppose that an acute tuberculosis was accidentally developing itself at the same time, just as occurred in a child who died a few weeks later of scarlatinal nephritis; but against such a view is the fact that the lungs in the fever case were free.

Incubation.—This period appears to vary within somewhat wide limits. The date at which a patient receives the poison can seldom be directly fixed. At Guildford, in 1867, an epidemic was traced to the fact that contaminated water was supplied on a single day, the 17th of August; a

large number of cases came under medical observation on the 3d and the 4th of September, so that, allowance being made for the gradual development of the symptoms, the incubation probably lasted about eleven days. On the other hand, that it may be as short as two, or four, or eight days seems to have been shown by instances in which the disease has broken out within such periods after a definite exposure of the patient to sewer gas, or after his having drank contaminated milk, or after his having first arrived in an infected locality. With regard to cases in which the incubation has been supposed to be protracted there is great theoretical difficulty. Persons sometimes have not fallen ill until three weeks, or longer, after leaving a place in which there is reason to believe that they must have taken the fever; but the question is at what date the intestinal lesions began to develop themselves in such cases; and this I shall discuss almost directly. Some observers think that the incubation varies according to the mode in which the poison enters the body, being shorter when it is inhaled with the breath, longer when it is swallowed in drinking water.

Course.—The commencement of enteric fever is generally slow and insidious. The patient feels depressed and weary, more and more so every day. He has headache, and giddiness, and pains in his back and limbs. He loses appetite; he may vomit or have diarrhœa; or, if he takes an aperient under the idea that it will set him right again, his bowels may remain relaxed. After about five or six days he becomes so ill that he has to give up work and take to his bed. In such a case the duration of his illness is reckoned from the day at which he first felt ailing. There are, however, some cases in which marked symptoms set in with chilliness, or even a rigor, so that medical assistance is at once sent for. If then, on inquiry, it appears that the patient has been unwell for a few days previously, the disease is said to have had a *prodromal stage.* It is clear that we ought to count the whole duration of such cases. For I have already remarked that the lesions are often found after death to be further advanced than had been expected from the clinical history. It may even happen that malaise and other slight symptoms run on for two or three weeks, or longer still, and that afterward a definite illness begins, the subsequent course of which is not shorter than usual. These instances create a difficulty from which Dr. Irvine fairly enough proposed to escape by assuming that the apparent primary attack is in reality a relapse, which, according to circumstances, may either be intercurrent or independent. But it must not be forgotten that such a theory involves the impossibility of proving that the incubation of the disease is ever prolonged, since one could not deny that in certain cases the real primary attack may be altogether latent. A case referred to at p. 200 shows that even the evidence afforded by an autopsy would not be conclusive. After all, however, the only practical point is that in every instance of this kind, whether the patient may or may not have been ailing before he fell ill, one must be prepared in case of death to find some of Peyer's patches presenting appearances corresponding with an advanced stage of the disease. In some very exceptional cases, enteric fever sets in with sudden maniacal delirium, so that the patient's removal to a lunatic asylum may appear to be necessary until the real nature of his case is manifested.

According to Wunderlich, the *temperature* during the first three or four days rises in a zigzag fashion; from morning to evening there is an ascent of about 3° F., from evening to morning a fall of about 1°. The result therefore is that each evening the thermometer stands at about 2° higher than on the evening before, and he declared this course to be in itself tolerably decisive for diagnostic purposes, so that enteric fever might generally be excluded if the temperature rose during the first two days to 104°; if (at

least in a patient under middle age) it did not rise between the fourth and the sixth day to 103.1° ; if it stood at the same level on two successive mornings or on two successive evenings ; or if it was ever lower than at the same hour on the previous day. He did, indeed, point out that this stage of the disease comparatively seldom comes under medical observation, but he seems to have overlooked the fact that the exceptional cases in which its onset is marked by definite symptoms are almost the only ones in which one is likely to have to use the thermometer during the first few days, so that they afford no proof that similar results would be obtained in cases beginning in the usual insidious manner. Jenner remarked to the Clinical Society, in 1875, that in private practice there were found to be numerous exceptions to one of Wunderlich's rules, and this certainly accords with my more limited experience in the matter. In 1879 a patient in Guy's Hospital, convalescent from pleuritic effusion, was attacked with enteric fever. He first felt unwell, on April 2d, and his temperature immediately rose from 98.6° to 102.6° ; next day he had rigors ; on April 4th there were characteristic stools ; on April 8th rose spots appeared. In 1878 a girl was admitted who had been carefully watched from the third day of her illness by Dr. Ingoldby ; the morning and evening temperatures were on that day 103.4° and 105.2° respectively, on the fourth day they were 103.4° and 104.2°, on the fifth day 103.4° and 102.3°, on the sixth 103.5° and 104.2°.

As the disease advances the patient's state slowly becomes more and more serious. He generally grows weaker from day to day, his mental faculties are more obscured, he manifests less concern about his own condition. Yet the maximum temperature to be attained in the whole course of the fever is often registered by the fourth, the sixth, or the eighth evening. From that time the thermometer indicates only trifling variations during the next ten or fourteen days ; in the morning it generally stands one or two degrees lower than in the evening. The skin may either be dry or moist. Profuse perspirations are by no means infrequent, especially at night ; they may be accompanied by an abundant eruption of sudamina, especially toward the end of the second and in the third week, and then again lead at a still later period to desquamation of the cuticle of the chest and abdomen, the importance of which lies only in the fact that its cause may be misunderstood.

The *aspect* of a patient with enteric fever is in general that of languor and weariness ; the face is not dusky nor the aspect stupid, as in typhus. There is usually pallor ; with a circumscribed pink flush on one or both cheeks, especially toward the latter part of the day or when food or stimulant has been recently given.

The *pulse* may not be very rapid ; its beats are generally from 100 to 110 per minute, but sometimes they remain at the normal rate, or are even less frequent. Murchison lays stress on the liability to variations in the pulse-rate at different periods of the day, in correspondence with the fluctuations of the temperature or independently of them. Changes of posture also affect it much more than in health, but the most important peculiarity of the pulse is its soft, compressible character, with a dicrotism which may be so marked that an unskilled observer may actually count it at twice the frequency of the heart's beats. These features are well shown by the sphygmograph. The *breathing* is more or less quickened. In many cases bronchitis is present from an early period.

The *spleen* becomes enlarged in the course of the first week, and by the end of the second week it is often two or three times its normal size. Sometimes its edges can be felt below the ribs, but in most cases its position can be mapped out only by percussion. Indeed, I, for my part, think that even this method of detecting enlargement of the spleen often fails, and that as a symptom it is of far less clinical value than most writers state.

With resonant lungs and a full tumid abdomen, I believe that the organ may be many ounces heavier than natural, without there being any appreciable percussion dullness over it. Nor is it a fact that the spleen is always found after death to be swollen, even in young subjects who have succumbed when the disease is at its highest. I have notes of instances in which it weighed only four, five, or six ounces. Its substance may be much softened, but it is not unfrequently firm.

Epistaxis is of rather frequent occurrence, especially soon after the commencement of the disease. It may be so profuse as to destroy life.

At about the end of the first week there appears the *rose rash*, which is the one symptom of enteric fever that is almost, if not quite, pathognomonic. This is not always the earliest cutaneous affection observed in the disease, for it is sometimes preceded by a diffused scarlatinoid eruption which comes out two or three days sooner, and which may be attended with a slight sore throat, so that it has led to a mistaken diagnosis in several instances. The rose rash itself is, of all rashes, generally the least conspicuous; to an untrained observer the idea seems absurd of attaching great significance to the presence of small red spots hardly larger than pins' heads, the whole number of which may not exceed ten or twenty. Yet, although a case which I shall briefly relate further on seems to show that similar spots may very rarely be seen in at least one other disease, miliary tuberculosis of the lungs, one is, I think, justified in regarding them, when well marked, as practically conclusive of the presence of enteric fever. But I quite agree with Murchison that it is often impossible to say of individual spots whether they are really rose spots, or what, for want of a better name, he terms "ordinary pimples." The regions in which they are most often looked for are the lower part of the chest and the front and sides of the abdomen; but the same author speaks of having often found them on the back when there were none anywhere else. In exceptional cases they are scattered thickly over the whole of the trunk, and even upon the face and the limbs. The date at which they first appear is usually between the seventh and the twelfth day, sometimes as early as the fifth or sixth day from the commencement of symptoms, sometimes not before the fourteenth or even the twentieth day. Liebermeister speaks with hesitation as to their ever being absent throughout the whole course of the disease; but it is certain that, in children, especially, one fails to observe them in many cases in which careful search is made every day. They can not only be seen, but felt, as rounded, smooth, solid papules; their color, which is pink or rose-red, disappears beneath the pressure of the finger; they never become petechial; after death they are invisible. In some exceptional cases it is said that their summits are vesicular. One of their most important characters is that they appear in successive crops, so that although the duration of the rash as a whole may be from one to three weeks, each individual spot never remains for more than three, four, or five days, sometimes not longer, even, than twenty-four hours. It has sometimes been noticed that they have come out in large numbers after a warm bath, or that they have first shown themselves on a part that had been reddened by mustard.

Next in importance, as helping to distinguish enteric fever from other acute specific diseases with which it may be confounded, are certain symptoms referable to the digestive organs. The *tongue*, even at first, while it is covered with a white fur, has commonly a bright-red tip and bright-red edges. In the course of the second week it often becomes clean, and its whole surface may then be of the same vivid color, and smooth as though it had been glazed. It sometimes remains moist, sometimes becomes dry; and deep painful transverse fissures often form in it. I need hardly mention anorexia and thirst, which are common to this and to almost every other febrile

complaint; nor is much stress to be laid upon nausea and vomiting, although they are very often complained of at the commencement. But the occurrence of *diarrhœa* is of considerable diagnostic significance. Sometimes, indeed, it is not present at any period of the disease. The proportion of such cases probably varies in different places and in different years, but Murchison, as the result of his experience, from 1862 to 1873, places it at one-fifth. And in many other instances the bowels cease to be relaxed when the patient has been ill for a few days, or they first begin to do so during the third or the fourth week. Louis stated, and Murchison has confirmed it, that, as a rule, the severity of enteric fever is proportionate to the urgency and persistence of the diarrhœa. But it is to be observed that there is no necessary relation between the symptom in question and the extent of the intestinal lesions in this disease. It was thought by Addison that diarrhœa was more constantly present when the colon was ulcerated than when the morbid change was confined to the glands of the ileum;* but if such a rule exists, it is certainly not without exceptions. The number of the evacuations is generally about four to six in the twenty-four hours; but sometimes it is much more. The stools have an appearance which has been compared with that of pea soup, and which, although not uncommon in other intestinal affections, is probably seen in no general disease except enteric fever, the color is yellowish, almost like that of yellow ochre. Addison used to lay stress on their being almost identical with the normal contents of the small intestine, and he supposed that they were hurried on through the large bowel and discharged without having undergone the usual changes there. Albumen is present in considerable quantity. The reaction is alkaline, and there are very numerous crystals of the "triple phosphate." Blood may often be recognized microscopically or in the form of small clots, even when the patient has been ill less than a week, so that ulceration is not likely to have begun. Of abundant intestinal hemorrhage, as a serious complication, I shall speak further on. I am under the impression that Dr. John Harley first pointed out the importance, in all doubtful cases, of searching during the third week for fragments of the sloughs from Peyer's patches. He advises that the stools should be strained through a little net, and that the matters caught upon it should be washed and then examined by a lens when flocculent shreds may be obtained, in which the remains of intestinal follicles can be plainly recognized. In 1873 a man died in Guy's Hospital, who two days before his death passed a slough an inch and a half long, in which muscular fibres were discerned; at the autopsy the corresponding ulcer was found to be two inches in length and to have the peritoneum exposed in its floor.

Another sign of intestinal disturbance is, in some cases, the production on pressure (which must be cautiously exerted) of a gurgling sound in the right iliac fossa. There may also be more or less tenderness in this region, and the patient may complain of pain there or in other parts of the abdomen. During the second week of the disease the bowels generally become distended with gas, so that the abdomen assumes a rounded form. Sometimes the enlargement is extreme, constituting what is termed "meteorism." This is a grave symptom. Murchison says that the colon is the chief seat of the accumulation, and that the bulging is therefore at the sides rather than in front.

The *urine* is at first scanty and high colored, and may remain so throughout the whole course of the disease, but after a time it may become copious, pale, and of a low specific gravity. Toward the end of the third week it often contains albumen in small, and sometimes in large, quantity; there may be even hematuria.

[* The same was observed by Mr. Busk at the Dreadnought Hospital.—Ed.]

In some mild cases of enteric fever, *cerebral symptoms* are almost absent. In the course of the second week the headache subsides, and the patient may seem to have but little the matter with him. There is no doubt that before the thermometer came into use persons were often wrongly allowed, and even persuaded, to leave their beds with the disease still upon them. There is not always very much muscular prostration. Murchison says that forty-four out of one hundred patients under his care were all always able to sit up, and to get out to the night chair. But in the third week, or even earlier, many do become altogether helpless and lie upon the back, unable even to turn over to either side. In such cases the hands and the tongue are tremulous; there is sometimes, but rarely, inability to protrude the tongue. Jenner believes that a disproportionate intensity of tremor, as compared with other nervous symptoms, is of diagnostic and prognostic significance, pointing to the presence of deep ulceration of the intestine, such as is likely to lead to perforation or to dangerous hemorrhage; and a similar statement was made by Murchison. In some rare cases the limbs or the trunk become rigid, or the back of the neck is retracted, or there is strabismus, or spasm of the pharynx, or trismus. Murchison's large experience yielded him only six instances of the occurrence of epileptiform convulsions. There may be all degrees of delirium in this disease, from the slightest rambling or moaning during sleep to the most violent maniacal excitement, with the patient screaming or shouting for hours together. Liebermeister remarks that one may sometimes fail to appreciate the extent to which the mental powers are enfeebled; a man may answer questions as to his actual condition clearly, but when asked how long he has been ill, what is the day of the week, what the month, what the year, he may be altogether unable to reply. Active, noisy delirium (which is more common in enteric fever than in typhus) is always a dangerous symptom. A condition resembling delirium tremens is comparatively infrequent, even in persons who have been intemperate. Sometimes the patient unexpectedly jumps out of bed, and may throw himself out of the window. Or, as Gairdner observes, he may lie for several days perfectly still and quiet, apparently understanding everything that is said or done, but unable to articulate intelligible replies. The *pupils* in enteric fever are almost always dilated, as Jenner first remarked; but Murchison observes that where there is complete unconsciousness they may be as narrowly contracted as in any case of typhus.

Resolution.—When recovery is to take place the fever sometimes, though very rarely, subsides by a crisis. A medical friend of mine, in whom the temperature from morning to evening had been ranging from 101° to 103° with almost absolute regularity, was found, on what was calculated to be about the twentieth evening, to have a temperature of only 101.1°; next morning it was 100°, in the evening 99.3°; on the twenty-second morning it was 98.3°, and for several days afterward it remained slightly below normal. But the rule is that at some period of the disease, generally toward the end of the third week, the morning temperatures begin to fall, while the evening temperatures rise to the same level as before. In other words, on a daily chart, a series of exceedingly acute zigzags are formed. After a few days, these become still more exaggerated, there being perhaps a difference of 4° or 6° between the temperatures of a single day; but the absolute height of the evening rise now begins to decline in its turn, so that each day it is about 1.5° or 2° lower than on the previous day, and thus, towards the end of the fourth week, a temperature is reached which is normal or slightly below normal. This *intermittent* type of pyrexia at the termination of the disease is so characteristic that I have known a correct diagnosis based upon it alone, in the case of a child about whose earlier symptoms no information was

attainable; but, on the other hand, there are not a few instances in which the fever runs on for four, five, or even six weeks without any definite change. In a woman who was under treatment at Guy's Hospital in 1874 the temperature at the expiration of six weeks was still 103.8°, then it fell suddenly, and within two days became normal. It is in such cases that, if death occurs after several weeks' illness, one finds all stages of the lesions in Peyer's patches and solitary glands down to early swelling. I therefore do not concur in the statement which has been made that fever protracted for more than four weeks is generally due either to some complication or to non-cicatrization of intestinal ulcers formed in the early period of the disease. Murchison himself says that he met with several instances in which rose spots appeared as late as the thirty-fifth day, and one in which fresh spots were noted almost every day up to the sixtieth day. Some observers think that the right view to take of such cases is to regard them as examples of relapse occurring "intercurrently;" that is, that before the primary attack comes to an end, a second one begins and carries on the fever without apparent break.

During the stage of subsidence of the fever the pulse sometimes remains rather disproportionately high. This is often a period of much suffering to the patient who, as his mind becomes clear, complains more and more of weakness and of prostration and of the impossibility of finding a comfortable posture for his wasted body and limbs.

The loss of weight during an attack of enteric fever is often very considerable, amounting to twenty pounds or even more. Moreover, the microscope reveals changes in many of the tissues. Thus Zenker, in 1864, pointed out two kinds of degeneration in the fibres of voluntary muscles; some become granular, others undergo conversion into a glassy-looking substance, in which no striæ can be recognized, and which splits up transversely into discs. As he himself remarked, however, the same lesion occurs likewise in other febrile diseases, if sufficiently severe and protracted. It is especially marked in the adductors of the thighs and in the recti of the abdomen. Muscles so affected are said to be sometimes obviously altered in appearance, being of a yellowish-gray color, but I have never been able to convince myself that such is the fact. They may during life be so much softened that they become extensively lacerated; thus, in 1870, a patient died of enteric fever in Guy's Hospital both of whose recti were torn across, and much blood was extravasated into them and into their sheath; and in another case, in 1866, the inner part of one rectus was found to be not only ruptured, but in a state of suppuration. Hoffmann has shown that similar forms of degeneration occur in the tongue, accounting, perhaps, for the tremor of its movements. The liver, again, has been described by many pathologists as undergoing a diffused change; it is soft, and on section it has a pale or "clayey" look. Hoffmann maintains that its cells become granular and break down. There are doubts as to whether this can account for jaundice, even in any of those very exceptional cases in which that symptom is present. I may remark that the occurrence in the gall-bladder of a colorless mucus, or of a muco-purulent fluid, after an acute illness, is not to be taken as showing that there has been a deficient secretion of bile of the liver. Probably it only indicates that for several days before death there was no storage of bile, in consequence of the very short intervals at which food was taken. The kidneys are often of a grayish-yellow color and have their epithelium granular. Perhaps this causes the albuminuria which we have seen to be sometimes present. Another organ in which Hoffmann believed that he detected changes was the brain. He describes the nerve cells in the great basal ganglia as loaded with brown and black pigment granules. Lastly, he found an enormous overgrowth of cells in the acini of the salivary glands

and of the pancreas. He says that those structures feel unusually hard and dense. In cases which recover all these various lesions are doubtless repaired. The process of regeneration of muscular fibres has already been traced, and according to Hoffmann the liver cells are reproduced by proliferation, of which there is evidence in the great excess of nuclei which these cells contain when death happens to occur at advanced stages of the disease.

The *convalescence* from enteric fever is slow in comparison with that from typhus, even when its course is uninterrupted by any accident such as will be presently described. Many weeks elapse after the subsidence of the pyrexia before the patient is fit to resume the active duties of life.

It will, I fear, have been by no means easy for the reader to take full possession of all the facts stated in the previous section with regard to cases of enteric fever which, although severe, yet terminate favorably. But the subject is by no means exhausted. I have still to describe (1) those forms of the disease which run an abortive course or are of exceptionally slight intensity, (2) the mode in which death is directly brought about, (3) a number of complications, some of which may themselves be fatal, (4) sequelæ, and (5) relapses.

1. *Slight or Abortive Cases.*—The symptoms are sometimes, from first to last, so slight that there is the greatest difficulty in persuading the patient that he is really ill, and in preventing him from going on with his usual work. Such cases are often seen in hospital out-patient practice in the second and third week, and the clumsy but expressive name of *ambulatory typhoid* has of late been adopted for them. Their duration may be as long as that of typical cases, or it may be slightly shorter. In many of them the affected intestinal glands probably subside without breach of surface, but that this is not always the case is shown by perforation sometimes occurring. When I come to speak of acute peritonitis I shall have to mention latent enteric fever among the very few causes that may give rise to it without warning.

Again, not a few cases of enteric fever attended with well-marked symptoms subside before the end of the third week. About some of them, which run on for sixteen or eighteen days, very little need be said. In all probability the intestinal lesions are comparatively slight, and the swelling of Peyer's patches or solitary glands very generally disappears by absorption and without ulceration. But since it is only by the rarest accident that their condition can be verified by an autopsy, I think it would be extremely unsafe to hazard an absolute statement about it, especially as in other forms of the disease there are so many exceptions to the rule of there being a correspondence between clinical symptoms and morbid changes.

Cases which terminate before the sixteenth day, however, require special study, because, their real nature being overlooked, they are very apt to be set down as examples of a simple febricula, or of a non-specific gastric or intestinal catarrh. Yet it is unquestionable that such cases may depend upon infection with the poison of enteric fever, for they often form part of "house-epidemics." Jürgensen, for example, mentions an outbreak near Kiel, in which fourteen out of twenty persons had an abortive form of the disease. Moreover, in addition to malaise, headache, and anorexia, many cases present rose spots, diarrhœa with characteristic stools, and enlargement of the spleen. The spots, indeed, are said to appear at an exceptionally early date, sometimes on the second, generally by the fourth or the fifth day. It is also to be noted that the onset of this variety of enteric fever appears to be particularly definite. In seventy-four of eighty-seven cases collected by Jürgensen it was sudden, and in forty-two it was attended with chilliness, or even with a well-marked rigor.

The temperature generally rises quickly, and it may be 104° by the second

or the third day. The maximum may be high; Liebermeister says that he has seen cases in which the thermometer rose to 106° in the axilla, and which yet subsided between the fifth and twelfth days, but in many instances the temperature at no time exceeds 101° or even 100°. Its subsidence is generally rapid, presenting scarcely any indications of the short zigzags seen in the commoner and more severe form of the disease, and is completed in from one to three days.

Lastly, enteric fever is sometimes not only of short duration, but also of very slight severity. Liebermeister even maintains that it may be altogether unattended with pyrexia, appearing as a slight non-febrile catarrh of the intestinal canal.

2. *Fatal Cases.*—The proportion of deaths to recoveries in enteric fever varies in different cities and at different times to some extent, but, perhaps, less than might have been expected. Murchison speaks of having seen the disease much more fatal in one village than in another a few miles off, and he seems to have been struck by the fact that several members of the same family have sometimes died of it at long intervals and at distant places. But, so far as I know, one never sees malignant outbreaks of it, such as occur in the case of the exanthemata. At the London Fever Hospital the average death rate from 1848 to 1870, was 17.26 per cent. of all cases admitted, the extreme figures in particular years being 28.42 and 12.82. For purposes of comparison, in estimating the effects of special modes of treatment, it would, however, be necessary to exclude all cases which ended fatally within forty-eight hours of admission; and this would reduce the average death rate to 15.82 per cent. Statistics from various sources given by Murchison, show a range of mortality from 11.37 to 32 per cent. At Basle, the average death rate from 1843 to 1864 was 27.3 per cent.

The cause of death is sometimes the direct severity of the disease; and this may bring about a fatal issue in two different ways, namely, either through the brain by coma, or through the heart by asthenia. Cases which are cut short by coma sometimes end within the first, and generally in the course of the second or by the beginning of the third week. It then scarcely ever happens that any special morbid appearances are discoverable in the brain or in its membranes; but one must be prepared for the discovery, in certain very exceptional cases, of meningitis, which I shall presently mention as a complication. Cases in which the mode of death is by asthenia commonly run on until the third or the fourth week, or even longer still. The heart is then found to be soft and pale, and sometimes its right ventricle is dilated, or even its left ventricle. Its muscular fibres are commonly more or less granular, and may even have lost their transverse striation. In one instance, Hoffmann detected in them a marked degree of the glassy change which I have already described as occurring in the fibres of voluntary muscles. Among one hundred and fifty-nine cases in which he examined the substance of the heart it was more or less profoundly altered in one hundred and three, but it is not stated whether in these cases there was a special preponderance of deaths by asthenia over those by coma.

3. *Complications.*—In a very considerable number of cases, death is brought about indirectly, by affections which can only be regarded as complications. Such affections are not all of them necessarily fatal, and they require to be somewhat fully described; they are more numerous and varied than in any other acute specific disease.

Foremost among these complications come certain *abdominal* affections arising out of the intestinal lesions. Indeed, it is here that the clinical importance of the lesions in question is chiefly shown; for I have already commented upon the small part which they play in the production of the regular symptoms of the disease.

One complication is severe *hemorrhage* from the bowels. Its frequency is difficult to estimate, because more or less blood may generally be found in the evacuations, if it is looked for from day to day. But Murchison says that in 3.77 per cent. of his cases it occurred to the extent of more than six ounces. The color of the blood may be either bright red or dark, and it may be fluid or partially clotted; if it has been retained for a time in the intestinal canal it appears a dark brown, like chocolate, or even almost black. When it is passed during the second week, it must be due to a general oozing from the swollen Peyer's patches, unless, indeed, the real date of the commencement of the disease should have been earlier than was supposed. But at later periods it often comes from an artery exposed during the separation of the sloughs. I have more than once seen one particular ulcer deeply blood stained or with a clot attached to its floor; and Jenner, in a case of his, injected water into the superior mesenteric artery, and found that it escaped from one of the ulcers. In other instances, however, after four or six weeks of illness, I have found a number of ulcers all equally reddened, or the whole mucous membrane of the last two feet of the ilium intensely congested, soft and swollen. Probably, too, intestinal hemorrhage is sometimes an indication of a scorbutic condition; I have known it accompanied by epistaxis, and by the appearance of purpuric spots upon the legs.

When the amount of blood which is lost is large the patient exhibits the usual symptoms of such an occurrence, and becomes blanched and cold. The temperature of even the internal parts is lower, by perhaps two degrees, a point of some importance, as it may afford the earliest indication of what has occurred, if none of the blood should, for a time, be discharged per anum.

Liebermeister says that in such cases the part of the bowel which contains the blood may be dull on percussion. According to him, hemorrhage also diminishes the rapidity of the pulse, and is often attended with a marked alleviation of the cerebral symptoms; but all these effects are transitory, by the end of twenty-four hours the fever is as high as before, or even higher.

There has been a remarkable difference of opinion with regard to the influence of intestinal hemorrhage upon the course of the disease. Graves, and after him Trousseau, declared that it was not unfavorable, but probably the real basis of their opinion was the fact that a good many patients recover from it, or, in other words, that it is not so often directly fatal as might have been anticipated. For the statistics of Murchison and Liebermeister show conclusively that the death-rate among cases in which this complication occurs is far higher than the average death-rate of the disease. The rejoinder may, however, be made, that the cases in question are altogether of greater severity than average cases, independently of their being attended with hemorrhage. As Murchison remarks, the deep ulceration which opens an artery is very likely to extend still further and to perforate the peritoneum; and thus it is well established that hemorrhage, when it subsides, is often followed, after a few days, by a fatal peritonitis. In connection with this point, however, it is worthy of notice that hemorrhage appears never to afford the earliest clinical indications of an enteric fever hitherto latent, as is often the case with perforation.

But even if we admit the force of the rejoinder, it by no means tends to support the dictum of Graves and of Trousseau, and one can hardly doubt that a considerable loss of blood in enteric fever must impair the patient's power of resisting the disease and favor the occurrence of cardiac failure. Thus Murchison says that he has repeatedly seen patients who had been doing well die unexpectedly, of syncope, a few hours after a copious bleeding. A case in point occurred at Guy's hospital, in 1879; the patient, a child aged six, went on favorably for a week after the hemorrhage and then suddenly expired. Dr. Goodhart found considerable dilatation and some

degree of fatty degeneration of the left ventricle of the heart. But the change, after all, was perhaps only such as we have seen to be of very frequent occurrence in uncomplicated cases; I doubt whether one can say more about it, than that the hemorrhage probably intensified it, and gave to it a special clinical significance.

The other chief abdominal complication of enteric fever is *peritonitis*, which is generally dependent upon perforation of the bowel. Its frequency is very great, especially in England. Thus, whereas Liebermeister gives it as the cause of death in 8 per cent. of fatal cases of the disease, Murchison found it present in nearly 20 per cent. of fatal cases, and in no fewer than 3 per cent. of all his cases, including those which ended in recovery. All writers are agreed that it more often occurs in males than in females, and in seventy-three instances of it, observed by Murchison, the proportion was as fifty-one to twenty-two. When there is an actual perforation, the aperture is sometimes exceedingly small and rounded, but in other cases, as Dr. Bristowe pointed out in vol. xi of the "*Pathological Transactions*," it is linear, showing that it was due to laceration; and stress must be laid upon the fact that its occurrence is sometimes directly traceable to disturbance of the bowel during defecation, by the administration of an enema, in the act of vomiting, or in sitting up to take food. Or it may perhaps be due to irritation from solid fecal matters, or to the presence of pent-up gas. Again, in certain cases, the gangrenous process affecting a Peyer's patch extends directly through the whole thickness of the bowel, and then a large slough may drop out, leaving a hole of considerable size. One may even find a number of such holes at different spots. Or the mere spreading of a putrid inflammation of the serous surface may light up a peritonitis without any actual perforation taking place. The distinction is said by Liebermeister to be sometimes manifest during life, from the fact that when gas escapes freely it allows the liver to fall backward from being in contact with the ribs, so that the percussion note in the right hypochondriac region becomes tympanitic. Sometimes, too, a large quantity of fecal matter is extravasated, and round worms may make their way outward into the peritoneal cavity. The seat of perforation is almost always in the lower end of the ileum, within a foot or two of the valve. It is said to be sometimes in the cæcal appendix or in the colon, but I believe that no instance of either kind has occurred at Guy's Hospital within the last twenty-six years. The date at which peritonitis sets in is usually the third, fourth or fifth week. Cases have been recorded in which it has been as early as the eighth, ninth, eleventh or twelfth day of the patient's illness, but in all probability the disease has then really been of longer duration. Perforation is often the cause of death in some cases in which there has been profuse diarrhœa, and in which all the symptoms have been well marked; but, as I shall hereafter have again to point out, it is sometimes the first sign that anything is amiss with the patient, the course of the disease having hitherto been altogether latent. So, also, it may occur at a very advanced stage, when two, three, or even four months have elapsed, and this may be the case not only when the fever has been unusually protracted, or when there has been a definite relapse, but even when convalescence has apparently been established, and when perfectly formed and healthy fæces have been passed. In two instances that have occurred at Guy's Hospital, each at the end of the sixth week, perforation has been distinctly traced to the ingestion of improper food. One patient was seized with pain very soon after eating two raw apples, another while in the act of eating watercresses. The symptoms of perforation are identical with those of a sudden attack of peritonitis, from whatever cause, but it is important to note that enteric fever yields even more than a due proportion of cases in which peritonitis sets in insidiously, and may even remain

altogether unnoticed. In a case observed in 1864 at Guy's Hospital the chief sign of an unfavorable turn in the patient's illness was his sudden refusal to take food, after which he died, in a few hours. Perforation and consequent peritonitis are often found at a post-mortem examination when they had not been suspected, the fatal issue having been attributed directly to the intensity of the primary disease. It has been much discussed whether recovery from this complication is possible. The doubt lies in the difficulty of determining whether there is an actual perforation in any given case in which peritonitis has set in, but Liebermeister says he has had four cases of recovery in which the abdominal cavity contained pus, and several instances have been recorded in which, death having occurred from some other cause, a perforation has been found to have been sealed up by adhesions, or at least closed off from the general peritoneal cavity. In other cases a circumscribed abscess has formed, which has discharged itself either externally or into the bowel.

But acute peritonitis does not always start from an intestinal ulcer. It may be set up by sloughing of a swollen mesenteric gland, as in a case recorded by Jenner, in which recovery took place, so that the real nature of the complication would not have been known if the patient had not afterward died of erysipelas. It has also sometimes been due to extension from an inflamed and ulcerated gall-bladder, or from a suppurating infarctus in the spleen.

Infarctions of the spleen are, in fact, not infrequently found after death from enteric fever. They are doubtless the result of plugging of branches of the splenic artery with portions of clot that had formed in and been washed away from the dilated and softened left cavities of the heart. In the same way *embolism* of one of the cerebral arteries, causing hemiplegia, with or without aphasia, may arise either during the course of the disease, or later on, when convalescence has already occurred.

In two cases at Guy's Hospital suppurative *nephritis* has occurred as a complication of enteric fever, and has apparently been the cause of death. The inflammation probably ascended from the bladder as the result of retention of urine.

Other complications of enteric fever have their seat in the respiratory organs. We have seen that a certain degree of *bronchitis* is almost always present, but sometimes it becomes so severe as to constitute a very important part of the disease. I do not, however, remember to have ever seen a case diagnosed as simple bronchitis which, on an autopsy, has proved to have been one of enteric fever. Not infrequently there is broncho-pneumonia, and lobar pneumonia is far more common than in typhus, and sometimes passes on to gangrene. Pleurisy, too, is often seen, and may lead to empyema.

Ulceration of the *larynx* is present in a considerable number of cases. Hoffmann observed it in twenty-eight cases out of two hundred and fifty. It has generally been set down among the primary lesions of enteric fever, but the absence of a specific cell-growth constitutes a distinction which seems important. Probably the explanation of its occurrence lies in some subtle association in morbid tendencies between the larynx, the lungs, and the intestine, of which we have other examples in acute pneumonia and in phthisis. As a rule, there is a sharply-defined ulcer over the base of one or both of the arytenoid cartilages. I believe that such an affection very rarely gives rise to any symptoms, but sometimes there is hoarseness, or even aphonia. In some instances, however, the morbid action involves deeper structures, and it may even be attended with necrosis and exfoliation of the cartilage, as in a case which I inspected in 1879. Hoffmann once saw the cricoid cartilage almost completely denuded. The entrance of air may, under such circumstances, be greatly obstructed. When I come to describe laryngeal perichondritis in

general, I shall have to discuss an opinion of Dittrich's, which would assign to this affection, when occurring in any other cases as well as in those of fever, a mode of origin analogous to that which produces bed sores. And as, according to Ziemssen, modern antipyretic treatment has much diminished the frequency of laryngeal complications in enteric fever, it seems fair to conclude that the pyrexia is in some way concerned in producing them. In another of Hoffmann's cases tracheotomy was rendered necessary by the formation of a polypoid blood clot, which hung down into the interior of the larynx. Or a laryngeal ulcer may endanger life by eating its way deeply into the connective tissue. In a case that occurred many years ago to Dr. Wilks, subcutaneous emphysema arose from this cause; the patient was a boy aged twelve. A similar instance, in a girl four years old, has since been recorded by Ziemssen. In his report of this case it is specially stated that the seat of the ulcer, which led down to the necrosed cricoid and arytenoid cartilages, was below the cords, and as the child had had much bronchitis there can be no doubt that the cough had forced air through the ulcer into the connective tissue. In two cases I have seen sloughs in the larynx, of a bright yellow color, exactly like that which, in the intestine, is attributed to the imbibition of bile. In one of them there were two linear ulcers, situated one on each side of the epiglottis, a position which is described by Hoffmann as frequent.

Sometimes a thin pellicle of lymph has been found lining both the interior of the larynx and the epiglottis. This brings one to the consideration of cases in which a membranous layer resembling that which characterizes diphtheria has been observed as a complication of enteric fever. Whether the specific poison of diphtheria has then been in operation it is difficult to say, but Murchison relates an instance in which fluids escaped from the nostrils when the patient attempted to swallow them, exactly as in that disease.

Another complication of enteric fever is parotitis. This, however, is much less frequent than in typhus, although Hoffmann regards it as simply an exaggeration of the morbid change which he finds so often present in the former disease. It sometimes affects one side, sometimes both. It may either subside after a week or two, or it may lead to suppuration, with extensive brawny induration and purulent infiltration of the tissues of the side of the neck as far as the sterno-mastoid muscle.

I have already remarked that in some very rare cases meningitis is believed to occur as a complication of enteric fever.

A point of some interest is that, as Liebermeister observes, the occurrence of enteric fever often leads to the reopening of fistulous openings which have healed up, with consequent necrosis of extensive portions of bone. In patients who have been suffering with soft chancres he has seen wide-spreading gangrene, under the influence of an attack of fever. In two cases at Guy's Hospital an ordinary gonorrhœa has led to sloughing of the penis or of the scrotum under similar circumstances.

4. In certain cases, some of the affections which are described as complications of enteric fever do not begin until the patient is already convalescent. It is, therefore, impossible to draw a fixed line between them and the *sequelæ* of the disease, which themselves may set in before it has come to an end. What really justifies the distinction and renders it useful in practice, is that the latter often run on for many weeks, and obviously constitute the sole obstacle to the restoration of health. For example, this is the case with *bed sores*, which are apt to form upon the buttocks and hips, unless great care is taken to prevent them, and which sometimes destroy life by exhaustion, or indirectly by setting up pyæmia. I shall discuss their causes and their treatment hereafter, in the chapter on paraplegia.

Another sequela, which will also be described elsewhere, is *thrombosis* of the femoral vein, with the attendant liability to pulmonary embolism.

Again, in some cases enteric fever is followed by *marasmus*. Murchison says that there is not always a repugnance to food; the patient may eat well, but what he takes is not assimilated, and slight errors in diet cause flatulence or diarrhœa. Yet the temperature is normal or below normal, and if death occurs, perhaps after many months, nothing can be discovered, except an unusually smooth appearance of the mucous membrane of the ileum, and a shriveled condition of the mesenteric glands. In other instances, however, where diarrhœa has persisted after the subsidence of the fever, it has been supposed that the ulcers have remained unhealed, or (to employ the usual phrase) have passed into an "atonic" condition. But although Dr. Murchison supports this statement with the weight of his authority, I am not sure whether it rests on strict pathological evidence; Dr. Wilks long ago taught me to doubt its accuracy, and I have never myself met with any example of it. I remember one case in which a patient was admitted into Guy's Hospital, whose sole complaint was pain in the right iliac fossa, which appeared to have been left behind by an attack of enteric fever; after a few weeks this pain subsided.

Writers generally speak of pulmonary *phthisis* as frequently occurring after enteric fever. Murchison seems to have thought that the correctness of this opinion might almost be taken for granted, on account of the long duration of the fever and the emaciation which it causes. But it is a very remarkable circumstance that, after searching the records of post-mortem examinations at Guy's Hospital, I have failed to find a single case in point. Can one suppose that the febrile disturbance which often accompanies the early stages of lung disease, where no physical signs may be discoverable, has been mistaken for enteric fever in the cases which have been so interpreted?

5. But the return of the temperature to normal is not necessarily followed by the restoration of the patient's health; enteric fever is apt to *relapse*. The frequency of such an occurrence seems to differ in different places; at Basle, Liebermeister met with it in 8.6 per cent. of 1743 cases; Murchison says, that in the London Fever Hospital, it was observed in 3 per cent. of 2591 cases; the figures given by other writers vary from 11 per cent. to 1.4 per cent. As a rule, there is an interval of some days between the termination of the first and the beginning of the second attack. Murchison states it as eleven days on an average; Liebermeister found that among 111 cases it was less than four days in twenty-seven, from five to seven days in seventeen, from eight to fourteen days in thirty-five, still longer in thirty-two. The late Dr. Irvine, however, in a valuable paper in the "*Medical Times and Gazette*" for 1879, maintained that the average interval is not longer than five days. He believed that a relapse sometimes begins before the original illness has come to an end. I have already remarked that this may explain those anomalous cases in which enteric fever seems to run on indefinitely for six or seven weeks or longer. It is not very uncommon for the termination of a relapse to be followed after a second interval by a second relapse, and this, again, may be succeeded by a third, and even by a fourth. There is scarcely a more important use of the thermometer in clinical practice than in the detection of such secondary attacks of fever. For it sometimes happens that the patient is unaware that anything is amiss with him, and yet to allow him to get about and to return to ordinary diet is to expose him to fearful risk. Thus at Guy's Hospital, in 1876, a man was apparently going on favorably through convalescence, when on the twenty-ninth day his temperature was found to be 104.2°; he looked ill, but he declared that he did not feel so. A few days later he died, of perfora-

tion of the intestine. In other instances the thermometer itself indicates but a very moderate degree of fever, ranging from 100° to 101° or 102°, but nevertheless taking a perfectly typical course. Dr. Irvine suggested that in those cases in which a relapse has been supposed to occur many weeks after convalescence, there have really been one or more intervening attacks which escaped notice. The duration of a relapse is by most writers said to be generally shorter than that of the original attack. Murchison found it to be, on an average, sixteen days. Dr. Irvine, however, maintains that it is typically twenty or twenty-one days. Cases in which it appears to be longer he explains by the hypothesis of an intercurrent second relapse, interrupting the middle of the first relapse, and subsequently running a regular course. According to this observer the temperature in a relapse generally rises pretty steadily, until on the fifth day it attains its maximum; it remains high until the ninth or the tenth day, when it undergoes a marked fall of from 2° to 6°; directly afterwards, however, it rises again, but it henceforth shows daily remissions, which at length bring it to an end.

It is now well ascertained that relapses of enteric fever are attended with a renewal of the intestinal lesions, although Trousseau maintained the contrary. We have had at least nine fatal cases at Guy's Hospital within the last twenty-two years, and in every one of them recent morbid changes have been found. As a rule, some Peyer's patches are in a state of early swelling, or have partly formed sloughs, while others show clean ulcers or cicatrices; but in one instance the floors of the patches are said to have been smooth and bare, while their edges showed the pink tumefaction of commencing disease. The clinical symptoms of a relapse may be identical with those of a primary attack. Rose spots are said by Murchison to appear somewhat earlier; in twenty-two out of thirty-eight cases they were visible on the third, the fourth, or the fifth day. In all of Dr. Irvine's cases the stools were characteristic in appearance. The patient is often delirious and insensible, so that, considering the debility resulting from his first illness, one is surprised that he does not more often succumb. As a fact, however, relapses are seldom fatal, except by some complication, such as perforation, from which recovery is practically impossible.

The cause of the liability of enteric fever to relapse is still imperfectly understood, and has naturally been the subject of many speculations. It cannot be due to a fresh infection with the specific poison from without, for the patient is often in a hospital, far removed from the original source of his disease, and little exposed to other chances of acquiring it. Very often, the time at which he falls ill for the second time is when he has just begun to take solid food, or when he has once or twice been out of bed. But other instances cannot possibly be thus accounted for; and, at the best, such an apparent exciting cause can only be regarded as setting up the symptoms of a morbid process which would otherwise have remained latent; since, if the patient happens to die a few days later, the intestinal lesions are found to be so far advanced that they clearly must have begun before the obvious relapse set in. For instance, in 1876, a case ended fatally on the sixth day of relapse, and several of the Peyer's patches had already ochrey yellow centres. Such cases, indeed, seem to me to afford the key to the whole question. In August, 1861, a man was admitted into Guy's Hospital with bronchitis and emphysema. A month later he was attacked with enteric fever, which ran a regular course, and from which he recovered, so as to be able to sit up. But early in October, the weather became very cold, whereupon his chest symptoms underwent rapid aggravation, and in a few days he died of them. At the autopsy, the lower Peyer's patches were found to be roughened and flocculent, as if sloughs had separated from them; but high up in the ileum one or two patches were still somewhat

raised, and several solitary glands showed an early stage of the affection. This case seems to prove that after an attack of enteric fever has to all appearances passed off, intestinal lesions may smoulder on, without giving rise to any symptoms. Dr. Maclagan some years ago suggested that sloughs thrown off from the patches first diseased might perhaps infect other patches in their turn. Such an hypothesis is difficult either to prove or to disprove. But with the analogy of spirillum fever to guide us, we can hardly be wrong if we suppose that the actual relapse of enteric fever is due to a re-infection of the blood with a virus (doubtless consisting of specific organisms) derived from patches thus recently diseased.

In connection with this question it may be observed that there is a marked immunity from a second attack of the disease among those who have once passed through it. Murchison alludes to observations made by Gendron and Piedvache, in which, after an interval of many years a particular house or locality became for the second time the seat of an outbreak, whereupon it spared those who had previously suffered, but attacked almost every one else. Murchison, however, had himself met with several exceptions to this rule, and he cites others.

Diagnosis.—The diagnosis of enteric fever may either be easy or difficult. In some cases a single examination of the patient may enable one to speak positively of the nature of his illness, especially in the second or third week; in some others, the most careful clinical observation, from day to day, may leave one to the last in a state of doubt.

At its commencement, the disease can never be determined with certainty, although very strong suspicions may be excited if there should have been other cases in the same house, or even in the same neighborhood. The commonest thing, in the absence of such circumstances, is to mistake it for a "bilious attack;" and immense harm is done by the aperient dose which inevitably follows. The fact that the onset of enteric fever is generally insidious is apt, when it begins suddenly, to suggest the idea that the case is rather one of typhus, of some exanthem, or of influenza. Liebermeister lays stress on the rarity of nasal catarrh and of sneezing at the beginning of enterica; and another point, which has long been known, is that it is very seldom attended with herpes about the lips. I have already mentioned cases of enteric fever that were at first set down to mania.

At about the end of the first week, that is, within two or three days of the time at which cases generally come under medical observation, a positive diagnosis can, as a rule, be given. It has already been remarked that the early roseola has sometimes been mistaken for scarlet fever, in spite of its not coming out until the fourth or fifth day. Murchison notes that he has known a copious eruption of rose spots attributed to smallpox. Even where no rose spots appear, one can be tolerably confident of the nature of this disease from the course of the pyrexia, the presence of an enlarged spleen, and the characteristic appearance of the stools. But in most cases, one's opinion is in reality mainly founded upon negative considerations, and, as Liebermeister remarks, he who is sensible of the danger of committing errors is the most likely to avoid them.

The diseases which are liable to be mistaken for enteric fever without eruption, or for which it may be mistaken, may be divided into two groups.

First come certain general diseases. Of these the most important in many countries, although not in England, is *ague*. I have already more than once remarked upon the extreme difficulty of distinguishing the remittent forms of marsh poisoning from enteric fever, whether in individual cases or even throughout a district, unless opportunities should arise for making autopsies. Another is *general miliary tuberculosis*, without marked symptoms

indicating localization in any particular organ. But although it is quite true that at an early period of a case it is often impossible to say whether a patient is entering upon one or the other of these diseases, I find from our *post-mortem* records at Guy's Hospital that of late years there have been very few, if any, fatal cases in which a correct diagnosis has not been made. The most remarkable instance of persistent obscurity with which I am acquainted is one recorded by Senator, in the "*Berlin. Klin. Wochenschrift*," for 1881. A man, aged forty-two, was admitted into the Augusta Hospital of Berlin, on October 25th, 1880. He had not been quite well since the beginning of September, but his illness had been more marked for about ten days before his admission. His pulse was 80 to 84. His temperature was 100.4°, and for some days it ranged from 101° in the morning to 103° in the evening very regularly. On October 29th, the spleen was found to be distinctly enlarged, and on October 31st distinct rose spots appeared, and fresh ones again on the following day. On November 1st and 2d there was repeated epistaxis. The pulse, still 80 to the minute, was now plainly dicrotic. Signs of bronchial catarrh appeared in the backs of the lungs, and increased greatly up to November 11th. After November 2d the range of the temperature became narrower, scarcely moving downward below 101° in the morning, or upward above 102° in the evening. The pulse remained between 80 and 100. On November 10th the patient was found to be slightly deaf. The bowels acted every day or every other day. On November 18th it is noted that there was slight lividity. On November 21st suppuration of the left parotid occurred, with discharge of pus through Steno's duct. The diagnosis, about which up to this time there had been slight doubts, was now finally given in favor of enteric fever. Yet after the patient's death, which occurred on November 22d, miliary tubercles were found in the lungs and in other organs, while the appearances characteristic of enteric fever were altogether wanting. The ophthalmoscope had been used with negative results, and I think it impossible to say that a correct diagnosis could have been given. Yet another disease that may be mistaken for enteric fever is *pyæmia*, when its starting point is some deep-seated affection, and when it affects, not joints, but internal organs. Murchison says that he saw several cases of pyæmia due to caries of the temporal bone, of which the course was very like that of enteric fever. At Guy's I know of two instances in which such a mistake was actually made. In each of them the source of the mischief was latent disease of the lumbar or the dorsal vertebræ, there being secondary abscesses in the lungs and the kidneys, and once in the heart. Murchison says that in his experience the variations of temperature are in pyæmia always much greater. *Trichiniasis* has generally been set down as enteric fever by those who are unacquainted with its peculiar symptoms; but with due care this error might perhaps be always avoided.

Secondly, a great many local diseases may be overlooked, and their effects attributed to enteric fever, if a positive diagnosis of it be rashly hazarded, in the absence of rose spots.

Foremost among these is a cerebral effection, *tubercular meningitis;* but I reserve what is to be said with regard to this affection until I shall have described it. One very important point, which has been insisted on by Jenner, is that in enteric fever headache ceases before delirium begins; the two symptoms are not present simultaneously, unless, indeed, there actually is meningitis, as a complication. In the days when the clinical thermometer was not in use, I have known enteric fever mistaken for hysteria; in a diabetic patient I have seen it set down for diabetic coma; in a man who was the subject of lead poisoning I have known uræmia, dependent upon granular disease of the kidneys, mistaken for enteric fever.

Among thoracic affections, *miliary tuberculosis of the lungs* must especially be borne in mind. I have already pointed out that enteric fever is often accompanied with bronchitis: and even when miliary tuberculosis produces marked thoracic symptoms it may be difficult to say whether they are not rather due to this complication of fever. The points of distinction I shall discuss when I have described the other disease. Formerly, *acute phthisis*, causing a rapidly spreading consolidation of one or of both lungs, was sometimes mistaken for typhoid fever; and a similar error was even made with regard to *simple pneumonia*, where there was no pain in the chest, nor cough, nor expectoration. And even now, although the routine use of the stethoscope keeps us from overlooking the presence of a pulmonary lesion, we may sometimes be in doubt whether it constitutes the disease from which the patient is suffering, or is merely a complication. *Ulcerative endocarditis* has often been set down as enteric fever; the discovery of a cardiac murmur should in most cases suffice to put one on the right scent with regard to it, but sometimes the heart-sounds remain normal.

Various abdominal affections are liable to be confounded at the bedside with enteric fever. Foremost among them is *tubercular peritonitis*. In the absence of the positive signs of this affection its diagnosis from enteric fever may be impossible. I have seen more than one case of which the nature has still remained doubtful at the end of several weeks of illness. Again, two or three instances have occurred at Guy's Hospital in which *acute diffused inflammation of the colon*, generally of diphtheritic character, has been mistaken for enteric fever, there having been no symptoms of dysentery, so far as could afterwards be ascertained. Other diseases which have failed to betray their presence during life have been *typhlitis*, *perinephritic abscess*, and *abscess of the liver* secondary to ulceration of the colon, or (in another case) to suppuration in the broad ligament of the uterus, with an opening in the intestine.

Finally, it is important to remember that some patients omit to complain of *affections of the genito-urinary organs* sufficiently severe to cause profound constitutional disturbunce, and that others conceal them, from mistaken modesty. Sir William Gull used in his lectures to speak of cases of extravasation of urine to which he had been called, under the idea that they were enteric fever, there being stupor, muttering delirium, a dry, brown tongue, etc. Some years ago I was asked to see a young lady at a school at Brompton who had been suffering for a day or two from febrile symptoms, with a very quick pulse. It was not until she had been sent home that she mentioned to her mother that she had severe pain in micturition, and that her private parts were swollen. There was then found to be an intense diphtheritic inflammation of the labia and nymphæ, and she very narrowly escaped with her life.

Prognosis.—In giving a prognosis in enteric fever one has to be guided not only by its symptoms, but also to some extent by the age and circumstances of the patient. The mortality does not, indeed, vary at different periods of life to the same extent as in typhus; Murchison's statistics show that until after the age of fifty-five it is almost uniform, at least among cases sufficiently grave to be admitted into the London Fever Hospital; and even in old people it is not much more than twice as high as in young adults. Enteric fever is especially dangerous in drunkards, in those who are very fat, in gouty persons, and in those who are affected with Bright's disease, heart disease, emphysema of the lungs, or phthisis. The death-rate is not augmented by a state of poverty. Murchison says that in private practice it is probably greater among the upper classes than among the very poor. In women the existence of pregnancy does not modify it to any

great extent—abortion or miscarriage almost always occurs; but, as a rule, such patients recover.

The indications afforded by symptoms are exceedingly liable, when they seem to be favorable, to be unexpectedly upset by the supervention of some almost inevitably fatal complication. It is this which has led to the prevalent idea that a prognosis ought hardly to be hazarded at all in enteric fever; and one cannot too strongly insist on the fact that no case, however mild, can ever be declared to be altogether free from danger; but, if complications be left out of consideration, Liebermeister maintains that there is scarcely any other acute disease of which the course can be foretold at an early period with so high a degree of probability. The basis on which one's opinion must mainly rest is afforded by the thermometer. The death-rate is found to be greater in proportion as the maximum temperature—usually an evening temperature—is higher, and by the end of the first week the fever has generally reached its highest point, or nearly so. Still more important are observations on morning temperatures. The more marked the morning remissions the more favorable the case. A considerable fall on even a single occasion is a good sign, unless it is due to hemorrhage or some other complication. Fielder found that the disease proved fatal to more than half of those patients in whom the temperature in the morning reached 105.4°, and to every one, with a single exception, in whom it reached 106.25°. Liebermeister seems to think that morning remissions are of value by interrupting the pernicious action of heat upon the tissues, and that they also show the fever to be less obstinate than if it were continuous. He goes on to say that it is a favorable omen if a cold bath lowers the temperature much and for a considerable time, and that a dose of quinine affords a sort of test as to the gravity of a case, this being less in proportion as the antipyretic action of the alkaloid is greater.

But individuals differ in the extent to which pyrexia is injurious to them, and the best measures of such differences are afforded by the state of the pulse and by the degree of disturbance of the cerebral functions. Liebermeister put together the cases that had occurred at the Basle Hospital, and shows that, the average mortality being 16 or 17 per cent., no fewer than 40 per cent. of those patients died when the pulse reached 120, and 80 per cent. of those in whom it exceeded 140. He also arranged his cases according to the intensity of the delirium and coma, and corresponding variations were found in the death-rate. Fatal complications occur far more frequently in severe cases than in those of which the course has been mild.

Treatment.—But the prognosis of enteric fever also depends upon the treatment to which the patient is subjected. It is a point of extreme importance that from the very commencement of the disease he should be kept at rest, and free both from bodily fatigue and mental strain. Not the least of the advantages which result from the habitual use of a clinical thermometer in daily practice is that the discovery of pyrexia in what might otherwise have been taken for a trifling disorder of the *primæ viæ* gives one grounds for insisting that no work shall be done until the temperature has fallen to the normal level. Men are apt to do themselves irreparable injury by struggling on day after day, even if they do not try to "walk off" their illness, or go away for change of air and a holiday, when they ought to be lying quietly in bed. The members of our own profession show no more judgment than others in this respect; Liebermeister has known medical men to go on seeing patients after having themselves noted their temperature at 104° on the previous evening. Sir William Jenner, speaking at Birmingham in 1879, declared ("*Lancet*," 1879, ii, p. 715) that some of the worst cases of enteric fever which he had ever seen appeared to owe their gravity to the circum-

stance that the patient had traveled, after having begun to feel ill, in order to reach his home. He added that he scarcely ever allowed a patient to be removed from the place where he was seized with the disease if his residence were at a distance.

The sick room should be large and airy, so that it can be well ventilated without draughts. I do not know whether Liebermeister is right in asserting that a person whose temperature is much above normal is incapable of catching cold, and that therefore exposure to draught for a short time can do him no harm. Jenner says that it is well to let him have a different room at night from that which he occupies during the day.

Another matter which is of great consequence is the diet. This throughout the whole course of the disease should consist of fluids only. The staple ought to be milk, of which two, three, or four pints may be supplied each day; it should be given in regular portions every two hours, or every hour, or even oftener; as the case goes on it soon becomes necessary to persevere with the feeding systematically throughout the night. If curdled milk is rejected by the stomach, or if it appears undigested in the evacuations, recourse should be had to Benger's *liquor pancreaticus*, after the method which will be fully described elsewhere; the conversion of the casein into a peptone prevents all further trouble with it. I think that this may very likely hereafter become a routine practice. Other articles of food which may be allowed are animal broths, beef tea, and calves' foot jelly. It is generally said that beef tea is apt to cause increased diarrhœa. Jenner advises that some strong essence of vegetables should be added to the broths. He permits a little strained fruit juice to be taken, but does not let his patient suck grapes, because of the difficulty of preventing the seeds from slipping down the throat; even particles of the pulp of fruits may, he says, cause irritation of the bowel. Pure water may be taken without stint.

Almost all observers are agreed that no method of treatment has yet been discovered by which the course of enteric fever can be shortened but I certainly cannot see that there is any absurdity in supposing this to be possible. We have good reason to believe that the disease sometimes spontaneously aborts at the end of the second week, and there seems to be no reason why the number of cases in which this occurs should not be increased by the administration of some medicine or other. Liebermeister, indeed, is disposed to believe that by three or four doses of from eight to ten grains of calomel, given in succession within twenty-four hours at an early period of the fever, he succeeded, in a certain proportion of cases, in cutting short the fever. He adopted this plan in two hundred and twenty-three cases. The mortality also was less than when no calomel was used.

Of late years, however, the attention of physicians, so far as the treatment of enteric fever is concerned, has been mainly directed to the question whether or not it is advantageous to the patient to have his temperature systematically kept down by cold baths or in other ways. And a mass of weighty evidence has been accumulated in support of this practice, which, although it had been advocated in this country by Currie nearly a century ago, had fallen altogether into neglect. The revival of it was due first, in 1861, to Brand, of Stettin, and secondly by Jürgensen, of Kiel, in 1866. Since that time it has been adopted by many physicians, both in Germany and in England; among ourselves no one has studied it more carefully than Dr. Cayley, of the London Fever hospital, who recorded his experience of it in the Croonian Lectures, for 1880. A point of great importance is that those who recommend an *antipyretic* treatment of enteric fever appeal, not merely to the vague impressions derived directly from the observation of cases, but to a numerical analysis of the results which they have attained.

It must be understood that the fundamental proposition upon which the antipyretic method of treating enteric fever is based is that pyrexia is harmful, not because the bodily tissues undergo rapid waste in the maintenance of febrile heat, but because they are affected injuriously by exposure to a high temperature, especially when it is kept up for a length of time and without intermissions. For, on the former view, which till recently was generally adopted, it would be worse than useless to employ cold, since the effect is undoubtedly to increase for the time very greatly the evolution of heat within the body, even if the temperature does not actually afterward rise again to its former level.

SCARLATINA.

HISTORY—ONSET OF THE DISEASE—THROAT—RASH—ABORTIVE AND MALIGNANT VARIETIES—COMPLICATIONS AND SEQUELÆ—PROGNOSIS AND TREATMENT.

The recognition of Scarlet Fever as a distinct disease began with Sydenham (1675), and was completed by Withering a century later (1778). It had, indeed, been described as far back as 1556 by Ingrassias, under the name of Rossalia; but Morton, who was a contemporary of Sydenham, maintained that it was only a variety of measles, and called it *morbilli confluentes*. The designation *febris scarlatina* (Ital. *scarlatto* = *scarlet*), was employed by Sydenham. It is very remarkable that he made no allusion to sore throat as one of its symptoms.

Course.—The onset of the disease is generally quite sudden. In children the first symptom is often vomiting or a convulsive seizure. In adults it is usually soreness of the throat. There may be chilliness or even a rigor. The patient complains of headache, malaise, prostration.

The face quickly becomes flushed, the pulse quick, the skin hot. For a child's pulse to be at 140 or 160 within a few hours is not uncommon, and is of no unfavorable augury; and it may remain high for some days. The temperature may rise to 104° or 105° in the course of the first day, or it may attain the same point more slowly, while the rash is coming out. It then generally rests stationary, or nearly so, until the rash begins to fade. The extreme dryness of the surface is apt to give one an impression that it is hotter than is really the case; from the time of Addison the pungent heat of the skin in scarlet fever has been spoken of at Guy's as comparable only with that which is to be felt in acute pneumonia. Dr. Gee describes the fever as frequently ending in a complete crisis; this occurred in two of his cases on the fourth day, in four on the fifth, and in three on the seventh. But most writers say that its fall is far more often gradual, taking from three to eight days for its completion. The tongue is at first coated with a thick, creamy layer, as in other febrile diseases. But before long enlarged fungiform papillæ are seen projecting as shining, scarlet points; and after two or three days the white fur clears away from before backward, leaving a smooth, bright red surface; this, with the little prominences that are thickly scattered over it, has an appearance that has been aptly compared with that of a strawberry. Sometimes, however, no such enlargement of the fungiform papillæ occurs. German writers speak of a miliary vesicular eruption as occasionally present, especially on the dorsal surface. The tongue does not regain its normal aspect until convalescence has set in.

On looking at the fauces one finds the arches of the palate, the uvula, and the tonsils more or less reddened or purple. Sometimes, but not always, they are swollen; and the redness may extend to the roof of the mouth and to the pharynx. Swelling of the mucous gland often gives a granular appearance to the affected parts; and their secretion may accumulate upon the surface so as to simulate the presence of ulcers. The tonsils may even project inward until, with the club-shaped uvula, they block up the passage; and after a few days one or both of them may suppurate. There is often swelling of the neighboring cervical lymph glands.

The rash generally makes its appearance in from twelve to thirty hours

after the commencement of the disease. Sometimes it is later; but, as Dr. Gee remarks, the proof of this is difficult, not only because the first signs of it are easily overlooked, but because it may recede for a time, and then come out again. In some of the more severe cases it is altogether ill developed, and writers say that it may then be delayed until the third or the fourth day. Trousseau had under his care a boy, six or seven years old, who suffered from headache, vomiting, strabismus, slowness of pulse and stupor, so that "cerebral fever" was diagnosed, until on the eighth day sore throat and a scarlatinal eruption appeared, whereupon the brain symptoms ceased; but such a case is altogether exceptional, and I think that there may fairly be a question as to its original nature. As a rule, the rash is first to be discovered upon the sides of the neck and the upper part of the chest; and it generally takes twelve or twenty-four hours, or even two or three days, to reach its full development. But sometimes it comes out almost at once over a very large surface. The face often remains free from it. Thomas insists that in most cases it is limited to the forehead and temples, the cheeks showing only the ordinary flush of fever, while the parts round the mouth, nearly to the chin, remain pale and afford a striking contrast. The upper arms are often covered with the eruption, and it is exceedingly well marked on the abdomen and on the inner sides of the thighs. The color is usually a bright scarlet, so that Watson aptly compared it with that of a boiled lobster; but sometimes it is of a lighter pink, and sometimes purplish. It may appear to be uniformly diffused, but on careful examination one can generally see that it is made up of very minute red points, which are at first isolated from one another, and which, even when they have coalesced, often leave a little islet of healthy skin here and there. It momentarily disappears on pressure, except where there has been some degree of hemorrhage or perhaps capillary stasis, in which case isolated red spots remain, with a more or less general yellow discoloration. As Watson long ago pointed out, on the forearms and the legs, as well as on the backs of the hands and of the feet, the rash of scarlet fever consists of larger and more prominent papules than elsewhere; the palms and the soles, however, show only a faint, diffused blush. The skin of the affected parts is slightly turgid or even swollen; the eyelids and the cheeks look a little puffy. Trousseau remarks that tumefaction of the fingers often prevents the patient from closing his hand. Histologically—Löschner discovered exudation cells in the rete Malpighii, and probably they were seen by Dr. Fenwick also, who further observed that the basement membrane of the sweat glands was thickened, and their channels were obstructed by an overgrowth of epithelium, or by extravasated blood. That the scarlet fever eruption bears no definite relation to these glands, nor to the hair follicles, seems to follow from a case (cited by Thomas on the authority of Landenberger) in which it did not fail to develop itself over an immense cicatrix, the result of a burn which was said to have destroyed the whole thickness of the skin.

Certain modifications of the eruption of scarlet fever are sometimes observed. Instead of being punctiform, it may in rare cases consist of large, irregular, slightly raised maculæ, more or less like those of measles, or it may be markedly papular. Or, again, it may be associated with immense numbers of miliary vesicles, especially upon the neck and chest, when there has been much perspiration.

At a variable period after the subsidence of the rash the superficial layers of the cuticle begin to peel off, or, in technical language, to "desquamate." This is sometimes observed within a few days, sometimes not until two or three weeks have elapsed. Its amount is not necessarily proportionate to the intensity of the cutaneous affection; it may be well marked where that was so slight as to have been overlooked, and so may be of service in clinching

a doubtful diagnosis. In some cases it assumes a furfuraceous form; a mealy powder, or a fine scurf, becoming detached from the surface, especially from the face. But more generally it is "membranaceous;" distinct flakes come away, and may be some inches long. The first step toward the throwing off of the epidermis at a particular spot is often the formation of a little opaque, raised vesicle, very like those which are characteristic of eczema, but dry; this breaks at the summit, leaving a free edge in the shape of a ring, which gradually becomes larger and larger. The cuticle of the hands is now and then shed *en masse*, like a glove; and the same may be the case with the feet; in some cases not even the nails are left behind. So complete a desquamation as this necessarily takes several weeks for its completion. Much more often, however, the fact that the exanthem interferes for the time with the nutrition of the nails, is shown, not by their exfoliation, but by the formation of a transverse groove upon them; to this Dr. Wilks has especially drawn attention; it is seen upon several of the nails at equal distances from their roots, and, of course, it ultimately becomes lost at their distal extremities. Such grooves on the nails are not, indeed, peculiar to scarlet fever, being sometimes seen after other acute diseases; but even with this qualification, their presence sometimes throws valuable light upon the origin of sequelæ the real nature of which might otherwise have remained undetermined. The hair comes off with the cuticle; but, so far as I know, not more than after any other fever of equal severity.

Aberrant Forms.—A striking feature of scarlet fever, and one upon which all writers lay stress, is the great variability of its symptoms and of its course; so that, indeed, there is much difficulty in describing all the different forms it may assume.

In some cases the disease is *rudimentary* or *abortive*. Thus, during epidemics, it often happens that adults become more or less feverish, complain of a slight sore throat with redness of the mucous membrane, and have a little pain and swelling of the cervical glands, but are at no time really ill. This is especially apt to be the case with those who have already had an attack in childhood; medical men have told me that it has again and again occurred to them, when attending patients suffering under scarlet fever. But I lately saw an instance in which a father so affected gave to his children a disease of such severity that one of them died. Again, the chief symptom may be feverishness, so that the complaint passes for a mere febricula, there being little or no affection of the throat, and no redness of the skin beyond what might be attributed to the pressure of clothes or to some other accidental circumstance. Lastly, the sole indication may be an eruption, and this, perhaps, limited to a small part of the surface. The real nature of this affection is often first shown by the occurrence of desquamation, or even by the unforeseen supervention of dropsy or of some other sequela.

In a very different class of cases, the symptoms are incomplete, because the end comes too quickly for their full development. In several recorded instances the patients have died in from eight to fifteen hours; the chief phenomena have then been delirium or coma, convulsions, or even tonic spasms with trismus, incessant vomiting, and diarrhœa, with extreme rapidity of pulse and of breathing. Sometimes there has also been hyperpyrexia, in which case one would be inclined to regard this as determining the form of the disease. A temperature of 115° F. was observed by the late Dr. Woodman in some patients. The only thing that could render a diagnosis possible would be the occurrence of other cases of scarlet fever in the same neighborhood.

The cases just referred to constitute the most extreme examples of what

in England is commonly called *malignant* scarlet fever. But there are other varieties of it which are less rapidly fatal. Sometimes, with early severe constitutional symptoms of a "typhoid" character, the rash comes out late and imperfectly, and its hue is not bright scarlet, but rather a livid violet. In some instances the disease assumes a *hemorrhagic* form; at least, almost all writers say so, except Dr. Gee, who is evidently inclined to think that the early hemorrhagic roseola of smallpox may have been mistaken for it.

Sometimes, again, the eruption develops itself in the regular way; but the febrile disturbance, which from the first is high, runs on for two or three weeks instead of subsiding after a few days. The patient then falls into a typhoid state, with muttering delirium, a dry, brown tongue, and sordes upon the lips. Many of these cases end fatally. In a large proportion of them the affection of the throat is unduly severe. Either the fauces are covered with a diphtheritic exudation, or they become gangrenous and slough away, leaving a horribly offensive cavity. The inflammation often spreads to the lining membrane of the nose, and an acrid fluid flows from the nostrils, which excoriates the upper lip. The nasal bones may even become necrotic. Still more frequent is extension to the ear along the Eustachian tube; the tympanum then fills with pus, which is discharged by perforation of the membrane. Here, again, there is often great destruction of the tissues: Sir Thomas Watson mentions a case in which, every time that the child swallowed, some of the fluid food ran out immediately at one of its ears; and it is no uncommon thing for fatal hemorrage to ensue from perforation of the carotid artery.

In other cases, the chief local mischief is outside the throat, in the cervical lymph glands and the adjacent structures. A large swelling may form on each side of the neck, near the angle of the jaw; or the whole space from the chin to the sternum may be occupied by a brawny, shining mass, which has been termed a "collar." It does not appear that the salivary glands, whether parotid or submaxillary, take any part in this affection. The induration of the connective tissue may spread backward between the pharynx and the spine, or downward into the mediastinum. If suppuration occurs, the pus may be discharged by a series of fistulous openings burrowing and undermining the skin in all directions; or a post-pharyngeal abscess may be formed, which, pressing on the larynx, may cause suffocation. Extensive sloughing often takes place, so that the muscles are laid bare, as in a clean dissection; and dangerous or fatal hemorrhage may ensue from perforation of one of the arteries or veins of the neck.

Complications.—Some of these affections ought, perhaps, to be described as complications of scarlet fever, rather than under any other head; but I do not see where the line is to be drawn. We may certainly give that name to one morbid condition which is comparatively seldom observed, and unrepresented in the normal course of the disease, but which is by no means confined to cases of great severity. I refer to a painful swelling of some or all of the joints, which generally sets in while the skin is peeling. This affects sometimes the smaller, sometimes the larger articulations; it generally quickly subsides, and Thomas says it is even more fugitive than that which occurs in acute rheumatism, and which is in all respects very similar to it. In some cases, however, it "settles" into one particular joint, leading to chronic effusion, or even suppuration.

Another, and a far more important complication of scarlet fever is an acute nephritis, accompanied with albuminuria. Whether any affection of the kidney is present in ordinary cases of scarlet fever, which end in recovery, appears to be still doubtful. Thomas remarks that the question could be settled only by the microscopical examination of the organs from a patient

killed by an accident during the course of the disease. It is certain that nephritis has been found after death in some exceptional cases of scarlatinal dropsy in which the urine, although scanty, contained neither albumen nor casts during life. This fact obviously suggests the possibility that renal changes may be present in cases which do well, without any evidence of it being discoverable. And it is also certain that the more assiduously one tests the secretions of the kidneys throughout the whole course of the disease, the more numerous are the cases in which a trace of albumen is detected at one time or another. Thus some German writers look upon a renal catarrh as bearing to scarlet fever the same relation which bronchial catarrh bears to measles. But, as Thomas points out, albumen and tube casts are occasionally found in the urine in several other febrile diseases, so that their presence is not necessarily to be taken as part of the action of a specific poison upon the kidneys.

Scarlatinal dropsy I shall leave to be discussed with the similar affection that results from other acute forms of renal inflammation. And as for the rest of the sequelæ of scarlet fever, all that is required in this place is that I should mention the fact that pleurisy, pneumonia, and pericarditis are of rather frequent occurrence, especially in connection with the rheumatoid disease of the joints. This may also be attended with endocarditis, and so may doubtless be the starting point of chronic changes in the valves of the heart; but whether such changes ever arise when the joints have remained free appears to me to be doubtful. In making post-mortem examinations of children who have recently had scarlet fever I have always found the valves healthy.

Chronic enlargement of the tonsils is very common in children who have passed through this exanthem. Far more important are various affections of the ears, which are apt to be left behind by it, so that, indeed, it is almost always responsible for the occurrence of deafness acquired in early life.

Prognosis.—The prognosis of scarlet fever cannot be altogether devoid of anxiety; the disease is never so mild but that some dangerous complication may arise. Sporadic cases are sometimes quite as severe as those which form part of an epidemic. Indeed, different epidemics differ very widely in their gravity. I have known one to prevail for many months in a country town without there being a death, and without dropsy ever supervening. Graves has recorded the fact that, whenever scarlet fever appeared in Dublin, between 1805 and 1833, it was always mild, so that on one occasion, eighty children were attacked in the same public institution, and all recovered; but during 1834 and 1835, the city was the seat of a very malignant and fatal epidemic. Thomas says that a mortality of from 13 to 18 per cent. is very common, but that it not infrequently rises to 25, and sometimes even to 30 or 40 per cent.

I have already indicated most of the inferences which are to be drawn from particular symptoms or combinations of symptoms. But it may be added, that, according to Trousseau, the occurrence of convulsions during the first or second day is always a sign of danger, whereas, in other exanthemata it is not of evil omen. Another point which is made by this writer is, that in scarlet fever, as in smallpox, the more intense the rash, the more severe is the disease. Here, however, he differs from most other observers; and I think that the statement can only be admitted to be correct in so far, that those cases in which a bright red eruption covers the whole of the body and lasts for an unusually long time, are generally grave ones.

Treatment.—Little need be said on this subject. We have no means of

checking the disease, and must be content to treat it on the same principles as those indicated in the chapters on Typhus and Enteric Fevers. In the more severe forms stimulants are particularly needful; and cold affusion or bathing is the best method of combating hyperpyrexia. Great care should be taken to avoid chill during desquamation, and inunction forms a protection to the patient against this danger, and to others against infection.

The extreme tenacity and activity of the contagion of scarlatina renders isolation imperative, and the precautions enumerated on pp. 47–49, are in no case more essential than during convalescence from this disease.

RÖTHELN.

(*RUBEOLA, EPIDEMIC ROSEOLA*).

RECOGNITION AND NOMENCLATURE—ITS CHARACTERISTICS—TWO INCOMPATIBLE DESCRIPTIONS OF ITS SYMPTOMS AND COURSE—POSSIBLY A VARIETY OF MEASLES—DIRECTION IN WHICH A SOLUTION OF THE QUESTION MAY BE EXPECTED.

Hardly had scarlet fever become universally recognized as distinct from measles, in the latter half of the last century, when physicians in Germany began to try to isolate a third member of the same group of diseases, and adopted for it the term "Rubeola," for which they had no other use, since they adhere to "morbilli" as the Latin name for measles. And ever since a controversy has been smouldering in that country as to the existence of this complaint, which is commonly known as Rötheln, the corresponding term for measles being Masern. But in France and in England the question has until lately attracted little notice, one reason being, in all probability, that the word rubeola was otherwise appropriated. Dr. Paterson, of Leith, however, described the supposed third exanthem in the "*Edinburgh Medical Journal*," for 1840, and among English writers who have since expressed their belief in its existence may be mentioned Copland, Aitken, Squire, Bristowe, Murchison, and Robert Liveing. A suitable name has yet, I think, to be discovered. Copland and Aitken, having reverted to the use of morbilli for measles, call it rubeola, but this is obviously very likely to lead to confusion. Nor does it seem to me that the term "epidemic roseola," proposed by Squire and by Bristowe, is much better, since it would naturally convey the impression that the disease bears some relation to the other eruptions which have been known as species of roseola since the time of Willan. On the whole I prefer the barbarous expedient of borrowing, for the present, the German name of Rötheln.

If one were asked what are supposed to be the most essential features of rötheln, one would reply that it has a rash very much like that of measles, but resembles scarlet fever in having a very short prodromal stage, and in being attended with marked sore throat. Thus it clearly is allied to measles rather than to scarlet fever, and it is not distinguished from measles by any positive characters comparable with those which separate measles and scarlet fever from one another. A favorite idea with some of the older writers was that it was a "hybrid" between these two diseases; they even spoke of the symptoms as alternating in different cases, so that sometimes the rash of measles would be associated with the sore throat of scarlet fever, whereas sometimes the reverse would be the case. But this was purely fanciful, and whatever may be said as to the possibility of the specific causes of two exanthemata combining so as to produce in a single patient a modified affection, one can scarcely conceive that such an affection should be epidemic, still less that its contagion should protect against the recurrence of itself while affording no immunity against either of the constituent diseases. It must be added that Trousseau declares rötheln to have no protecting influence, even against itself, but in this I believe he differs from all other writers.

There being, then, no positive signs distinguishing rötheln from measles, one would expect that those physicians who insist upon the separate existence of the former disease, and who have described it from their own observations, should at least agree among themselves as to all the details of its symptoms and course. But, unfortunately, the very opposite is the case. No two writers seem to me to give exactly the same account of it. And at the present time two incongruous descriptions of it are to be found in current medical literature.

One of them is fully set forth by Aitken. It is identical with that which Paterson gave, in 1840, and indirectly it owes its parentage to the German writers of the early part of the present century. According to this view of rötheln it is often rapidly fatal, destroying life by suffocation, or by convulsions and coma. Its earliest symptoms (in addition to rigors and nausea, or even vomiting) are redness and pain of the eyes, with lachrymation, sneezing and watery discharge from the nose, cough, sore throat, and hoarseness. The eruption, which does not break out till the third or the fourth day, appears in minute dots, but rapidly assumes the appearance of irregular-shaped patches, with obtuse angles, sometimes of an intensely dark color toward their margins, and distinctly raised—more so than in measles. It is always less abundant on the limbs than on the trunk, but appears upon both at the same time. Copland says that in severe cases the whole surface may, after twenty-four hours, have a deep and almost equal red color, so that the disease may now be readily mistaken for scarlet fever. Both he and Paterson agree that the eruption lasts four or five days, or even from six to ten days; it is followed by a branny desquamation. The constitutional symptoms are often severe; sore throat is always a prominent symptom, and there may be complete loss of voice, and inability to swallow even the smallest quantity of fluid, which generally regurgitates through the nose. Affections of the respiratory passages and lungs may occur as sequelæ; or there may be dropsy; or the cervical glands may suppurate.

Contrast with this the account of rötheln which is given in Ziemssen's "Handbuch," by Thomas, who speaks of having observed two epidemics of it at Leipsig, one in 1868, the other in 1872. The prodromal stage, he says, is very short, lasting from two to twelve hours, or even being altogether absent. When it is present, it is attended with a catarrh of the fauces and air passages, less marked than in measles, but so that sneezing and coughing are never altogether absent; the conjunctivæ are injected, the eyes are painful, there is intolerance of light. There is always a more or less spotted redness of the fauces, and the tonsils may be a little swollen; but pain in swallowing seems not to be a constant symptom. The tongue is covered with a white fur, through which a few red papillæ may be visible. The rash consists of round or oval spots, from the size of pins' heads to that of lentils, or even of beans; they generally remain isolated, but are sometimes connected with one another by projecting processes; they do not form the angular concentric outlines which are seen in measles. They are very slightly raised, and fade off a little at their margins; their color is sometimes bright when the rash is fully out, but is mostly a rather pale rose. They cover the face (including the lips), neck, body, upper arms, and thighs; they are not darker in hue on the face than elsewhere. On the forearms and hands, and also on the legs and feet, they are often less marked. Their duration is not more than from two to four days; indeed, they remain fully developed for a few hours only. When they disappear, they leave behind them very faint yellow stains. In the large majority of cases desquamation is altogether wanting; if present at all, it is very slight and partial. The disease may be entirely unattended with fever; there may not even be any malaise. Sometimes, however, the temperature rises to 100° or even to

102° for twenty-four hours, or occasionally for two or three days. With the subsidence of the rash, the recovery of the patient is complete. It is doubtful whether there are any complications or sequelæ; the cervical glands may be a little swollen, but pus does not form in them, and there is no liability to renal dropsy. A fatal termination is unknown.

It is, I think, clear that these two descriptions do not both belong to one disease, complete in itself, and distinct alike from measles and from scarlet fever. If one had simply to choose between them, one would have little hesitation in preferring the more recent one, and in agreeing with Thomas that the older physicians were often unfortunate in selecting the cases upon which they based their accounts of rötheln. But it is impossible not to feel that the grounds on which we are asked to admit its existence are materially weakened by the necessity of thus throwing over all the earlier observations. And what makes the matter worse is that even the more modern writers fail to agree in their statements concerning the supposed third exanthem. Thus, Trousseau insists on the absence of catarrh, —lachrymation, coryza, and cough—during the prodromal stage, and he assigns to it a usual duration of one or two days, rarely prolonged to three or four. He follows Vogel in making the rash give rise to intense itching, whereas Thomas declares that this is seldom the case. The patches, he says, are exceedingly fugitive, but they "disappear and reappear alternately for some days." They are most abundant on the trunk and limbs. Bristowe, on the other hand, speaks of the eruption of rötheln as generally most abundant on the face and on the forearms and the legs (especially about the wrists and the ankles); according to him its hue is dusky red or purplish. Liveing ("*Lancet*," 1874), while in all other respects he follows the later descriptions of the disease, says that the rash lasts longer, as a rule, than that of either measles or scarlet fever—from four to ten days.

Let us now consider what would be the alternative, if we should reject the doctrine of the existence of rötheln as an independent exanthem. Obviously it would be that the cases which have been supposed to be examples of it were really examples of an ill-developed or anomalous attack of measles. And, except from one point of view, I think that there is very little to be said against such a conclusion. The characters of the rash would assuredly not stand long in the way. I find Mr. Squire admitting almost as much as this ("*Brit. Med. Journ.*," 1870), although he believes that the two diseases are distinct. Nor could the presence of sore throat be relied on as a criterion, since recent writers speak of this as by no means severe, so slight, indeed, that the patient may make no complaint of any affection of the fauces, which is then first discovered by the physician, exactly as in measles. Nor would the short duration, or absence of prodromata be a proof that the disease is not measles, for one could hardly be surprised that in one of the mildest forms of that exanthem some of its less essential symptoms should be wanting. Nor, lastly, would the fact that a supposed rötheln may prevail epidemically, without passing into ordinary measles in any instance, be conclusive, since epidemics of scarlet fever sometimes deviate quite as much from the typical course.

The one real argument in favor of the separate existence of rötheln is that it explains the occasional supervention of what appears to be a second attack of measles in the same individual. But the importance of this argument may, I think, be easily overrated. For an apparent recurrence is now and then observed in the case of other contagious diseases which, as a rule, confer immunity. And, again, measles has been said to have attacked a single patient three times, so that, unless Trousseau is right in saying that rötheln possesses no protective power against itself, the recognition of that exanthem does not, after all, entirely dispose of the

difficulty. At first it seems a striking statement of Bristowe's, that when rötheln breaks out in a family, or in a school, where some children have had measles and others not, it attacks them indiscriminately and with equal mildness. But before one accepts this as final, one must be sure that the question has been looked at with unbiased eyes. Like some other problems, it will probably be ultimately settled by the experience of general practitioners practicing in the less crowded districts of the country.

Thomas describes rötheln as occurring chiefly in children, from infancy onward, but it has been seen in adults up to the age of forty, and (according to Seitz) in an old woman of seventy-three. Its contagion is said by most writers to be decidedly less active than that of measles. With regard to the length of its incubation there are some differences of opinion. Bristowe speaks of it as probably lasting about a week; Squire seems to say that its duration is generally ten days; Thomas prolongs it to two and a half or even three weeks, but he himself admits that the facts on which this estimate is based are not to be relied on, since what they amount to is merely that such an interval has often been observed between the dates at which different children have successively been taken ill.

MEASLES.

(*MORBILLI.*)

NOMENCLATURE—ONSET—ERUPTION—VARIETIES—COMPLICATIONS—SEQUELÆ —PROGNOSIS.

Two centuries only have passed since the writings of Sydenham (*circa* 1675) first led to the separation of measles and smallpox from one another, although the distinctions between them had been pointed out by Forestus in 1563. It follows that little is to be gained by attempts to trace the earlier history of measles; but I may mention that Rhazes and Avicenna are stated to have described it under the name of Hhasbah. That of Morbilli (Ital. = a little plague) seems to have been constantly applied to it until the middle of the eighteenth century, when Sauvages first called it Rubeola. This designation was unfortunately adopted in Great Britain by Cullen, and by Willan; and their authority led to its general acceptance. But within the last few years most English writers have reverted to the use of the term "morbilli."

Course.—The *prodomal* stage of measles presents more than one peculiarity. In the first place, although the patient is taken suddenly ill in the ordinary way, with anorexia, headache, and malaise; although vomiting may set in, or diarrhœa, or epistaxis, or (in children) a convulsive seizure; and although by the evening of the first day the temperature rises rapidly, so as to reach 102°, or more, yet the course of the fever during the next two or three days is altogether uncertain. On the second or the third day the thermometer may fall to normal; and, as the appetite may at the same time return, the idea of an impending illness may be almost abandoned. In other cases, however, the temperature remains at about 102°, with only trifling oscillations backward and forward. In the second place, there are catarrhal symptoms from the first; the patient is troubled with coryza, sneezing, intolerance of light; fluid secretion pours from his eyes and nose, his face and eyelids are swollen, and his conjunctivæ are injected; he may be hoarse, he may have a cough, sibilant sounds or rhonchi may be audible on auscultation over his chest; and the cough may be even harsh and "croupy." By the second, or, at latest, by the third day, one finds, on looking at the fauces, that, besides a general injection of the soft palate, there is an eruption of scattered points and spots over its mucous membrane. Ringer attaches some importance to the presence of thin, opaque, white patches on the gums, and the inside of the lips. In the pharynx one can hardly recognize anything more than a diffused redness, and the same has been observed within the larynx by the aid of the laryngoscope. Some writers have regarded all these appearances as indications of a "mucous exanthem," and have proposed to term it an "endanthem." But I agree with Thomas in thinking that such a view is certainly tenable only for the affection of the fauces; although it may be of clinical value in enabling measles to be recognized among the dark races of mankind, in whom no cutaneous exanthem is visible. The tongue in this disease is coated with a white fur, through which a few red papillæ may, perhaps, be seen projecting. The

faucial affection and the early catarrhal symptoms in general continue into the eruptive stage and then subside. According to Thomas, indications of the cutaneous rash may sometimes be seen on the face during the prodromal stage, in the form of minute puncta, around which the characteristic papules afterward develop.

The *eruptive* stage which succeeds begins generally on the fourth, but sometimes on the third day; in exceptional cases it is postponed until, according to Trousseau, six, seven, or eight days have elapsed; or even, according to Watson, until the tenth day. There is now a further rise of the temperature, which, in thirty-six hours (as a rule, on the sixth day) reaches its acme, this being generally 104° or 105°. Afterwards it may either at once begin to decline, or may remain at or near the same point for a day or two. The fall, when it does occur, is rapid, and the normal temperature is reached by the eighth or the ninth evening at the latest.

Prof. Thomas, of Leipzig, seems to have studied the thermometric changes in measles with more care than any previous observer; it will be noticed that his conclusions accord perfectly with those of the older physicians, who laid stress on the fact that "the fever does not, as in smallpox, cease, nor even abate, upon the emergence of the eruption, but sometimes increases in intensity" (Watson). The rash itself first appears on the face; Ringer says that the earliest traces of it are seen on the forehead, close to the scalp. It spreads over the face (not avoiding the parts about the mouth) and the trunk; on the limbs, especially the lower limbs, it generally comes out rather less freely, but it shows no decided predilection for the flexor rather than the extensor surfaces, and it may be well marked upon the palms and the soles. In several of these minor points it differs from the eruption of scarlet fever. The rash commonly takes three days for its complete development, but sometimes not more than a few hours; the later it is in beginning, the more quickly it diffuses itself over the body. Thomas says that before it has existed at any one spot for twenty-four hours it always begins to decline, so that when it comes out slowly it fades upon the face and neck, while it has still to make its appearance over the more distant parts; but according to Watson, it may remain three days, at least, on the face before its subsidence commences. The old doctrine that a rapid retrocession of the rash was apt to be associated with the onset of a dangerous complication is disputed by Thomas, in common with many other modern writers. On the other hand, he says that there is sometimes a short-lived recurrence of it when from any accidental cause there is an exacerbation of the fever.

The color of this rash is a more or less deep rose or crimson, inclining to purple rather than to scarlet. It consists of spots of irregular form, of all sizes up to that of a split pea. They are at first isolated, but afterward coalesce into patches, the margins of which are sharply defined, and here and there present rounded notches, or (to use the current expression) are "crescentic" in outline. They are slightly raised; indeed, even the earliest papules can be distinctly felt with the finger, corresponding, as Thomas believes, with the hyperæmic mouths of sebaceous follicles. However, he himself alludes to the investigations of Gustav Simon, who, having excised a portion of the skin from a patient affected with measles, examined it histologically with negative results, so that he could only attribute the swelling to hyperæmia of, and serous exudation into, the tissues. When there is much sweating a few vesicles of miliaria are sometimes to be seen. Before it subsides, the eruption acquires a yellowish tint, which is particularly well marked when the blood vessels are temporarily emptied by the pressure of one's finger. This is no doubt the result of the diffusion of altered blood pigment in small quantity into the substance of the cutis; for even in cases which are doing well, it is no uncommon thing for actual hemorrhage to

occur; so that, after the rash had faded, purple stains remain, which afterward become brown and yellow, and do not finally disappear for two or three weeks. Desquamation takes place to a slight extent, especially from the skin of the face. No large scales are ever detached, but only a fine mealy powder which (as Trousseau remarks) is often best seen when one brushes the skin of the patient with one's coat sleeve, or (according to Ringer) when the surface is scratched and viewed sideways. This writer says that it begins on the sixth or the eighth day, and continues for eight or ten days.

Varieties.—In many cases, however, measles runs an irregular course, and, like those of other exanthemata, its aberrant forms are of two kinds; some *benign*, being shorter and more favorable than usual, others *malignant*, attended with very grave symptoms, and ending more or less rapidly in death.

Each of these, again, has to be subdivided into two varieties at least. In one form of benign measles there is a rash, but no catarrh; in another it is the rash which is absent. Of *morbilli sine catarrho*, Thomas remarks that it is especially apt to occur in very young infants, and that it is almost unattended with fever. There is reason to suppose that many cases of rötheln were formerly assigned to it, notwithstanding the slight affection of mucous membranes which accompanies the exanthem in question. Perhaps this is one reason why Sir Thomas Watson says that the incomplete form of the disease confers no immunity against recurrence. In other cases, as he goes on to say, the failure to protect is shown by the fact that the patient is soon afterward seized with regular measles, a true relapse probably, such as may take place after other mild varieties of the complaint. Of *morbilli sine morbillis*, on the other hand, Thomas observes that this form is in all likelihood diagnosed more often than it really occurs; but surely the only case in which one would think of suspecting its presence would be if during an epidemic an unprotected person should suffer from catarrhal symptoms without any apparent cause, or should even go through the ordinary prodromal stage and then get well. Some writers have asserted that, as in the case of latent scarlet fever, all doubts may ultimately be removed by the occurrence of desquamation, but this being so slight, even when the affection of the skin is intense, one would hardly expect to see it where there has been no eruption at all.

Of the malignant varieties of measles, there is one which is attended with hemorrhages from the mucous surfaces, and with an intensely *purpuric* form of eruption. At the present day, however, this is very infrequent; it is observed chiefly in young and sickly children. Thus, when one finds the old writers laying great stress upon "black measles," one is almost inclined to suspect them of having mistaken cases of hemorrhagic smallpox for this disease; and it is interesting to notice that Sydenham speaks of an unusually bad kind of measles as prevailing in London, in 1670 and 1674, at a time when variola also was remarkably malignant and fatal. The hemorrhages are said sometimes to begin before the ordinary morbillous rash comes out, sometimes afterward, in which case it quickly fades or turns to a livid purple color. Petechiæ and vibices cover the skin, while blood oozes from the mucous membranes of the nose, kidneys, intestines, uterus, as well as into the substance of the deeper tissues and internal organs. Death generally takes place within a very few days.

In other instances measles becomes fatal by the severity and prolongation of symptoms which are not in themselves unusual. The fever is from the first intense and persistent; the rash, though it may come out early, is generally of a livid purple color, and often develops itself very imperfectly; instead of the temperature falling on the eighth or ninth day, it remains high throughout the second week; the pulse is very rapid and feeble, the patient becomes delirious and drowsy, and passes into a "typhoid" condition

with a dry, brown tongue, and sordes on the lips. Death is ushered in by prostration and collapse.

One of the most important clinical uses of the thermometer is in drawing attention to the fact that cases of this kind deviate from the ordinary course of measles, and, according to Thomas, it is no less unfavorable for the temperature to be low when the eruption is coming out, than for it to continue high at a time when it should be falling.

However, it is a rule to which there seems to be scarcely any exceptions, that in all but the most rapid among fatal cases of measles some *complication* or other is really the cause of death. Even when the symptoms are not sufficiently marked to lead to its recognition during life, a local lesion is sure to be discovered at the autopsy; and in cases that recover similar affections are very frequent, and often seriously modify the symptoms.

A broncho-pneumonia, with consolidation of scattered lobules throughout the lungs, is the most frequent complication, particularly in certain epidemics; indeed, it occurs so often that some writers have been disposed to include it in the regular course of the disease. But this cannot be admitted; and since the effects of catarrhal inflammation of the pulmonary tissues are the same as when a like affection arises from other causes, I may leave it to be described elsewhere. In some instances a lobar pneumonia is found, and sometimes, perhaps, there is simply a capillary bronchitis.

Another very dangerous complication is the formation of a layer of plastic exudation upon the fauces or within the larynx, a secondary "membranous croup" or "diphtheria," according to the view which is taken of such morbid changes; and in many cases diarrhœa becomes so severe as to bring about a fatal issue, especially when the evacuations assume a dysenteric character. I believe that little more than redness of the intestinal mucous membrane is found under such circumstances.

Among the less serious complications, so far as the life of the patient is concerned, must be mentioned certain affections of the eyes and ears. Ophthalmia is common, and often runs on for a great length of time after recovery from the exanthem. I have once seen a diphtheritic membrane form again and again on the conjunctivæ. Sometimes iritis occurs, and sometimes a destructive corneitis. In other cases the morbid action extends from the fauces along the Eustachian tube, causing deafness and pain in the ear. As a rule, it seems merely to lead to an accumulation of mucus in the tympanic cavity; but now and then suppuration takes place, and even necrosis of part of the temporal bone. Trousseau relates one instance of this, and another has come under my own observation.

Almost any of the complications of measles may be so prolonged as to have a fair claim to be regarded as *sequelæ*, but there are some other affections which have a still better right to that title, since they do not begin until after the patient has recovered from the primary disease. Among these is a form of gangrene, attacking the mouth or the female genitalia, and commonly known under the name of noma. According to Thomas, it does not arise spontaneously, but is preceded by some slighter lesion of the same parts, such as a decayed tooth, an inflamed gum, or an infantile leucorrhœa respectively. It is generally, but not always, fatal. In other cases measles is followed by necrosis of a portion of the upper or lower jaw.

Again, children often remain for many months after recovery in a state of ill health, for which no obvious cause can be found, and during this time they are very apt to be seized with bronchitis, or even by lobar pneumonia, especially in the cold seasons of the year. Tuberculous affections frequently develop themselves under such circumstances, and prove rapidly fatal. An intercurrent attack of measles is said greatly to accelerate the downward course of a pre-existing phthisis. On the other hand, certain

cutaneous diseases, such as eczema, seem often to be favorably modified by the supervention of the exanthem, at least for a time; and according to Rilliet and Barthez the same thing happens in some cases of epilepsy, chorea, or incontinence of urine.

Prognosis.—The mortality of measles varies widely in different epidemics. It has sometimes been so low as 2 to 3 per cent. of those who have been attacked, sometimes as high as 50 per cent. Trousseau relates that in 1845 and 1846 he lost with broncho-pneumonia twenty-two out of twenty-four children under his care in the Necker Hospital. According to Thomas the few cases which occur during the first six months of life are generally mild; but with these exceptions the disease is more severe in infants than in older children, and especially during dentition. Thomas says that he has several times found teeth just protruding from the gums when death had been preceded by uncontrollable pain, and by cerebral symptoms. Among adults measles is most dangerous to women who are pregnant or who have recently been confined, and to the very few old people who are susceptible of it. It is, however, almost always more severe in grown persons than in children.

The contagion is less persistent than that of scarlatina.

The general plan of treatment is the same, for in neither have we any specific method of dealing with the malady itself.

SMALLPOX.

(VARIOLA.)

INCUBATION—PRODROMIC SYMPTOMS—EARLY RASHES—SPECIFIC ERUPTION—VARIETIES OF, COURSE, AND SYMPTOMS—DISCRETE, CONFLUENT, AND MODIFIED SMALLPOX—COMPLICATIONS AND SEQUELÆ—PROGNOSIS AND TREATMENT.

This terrible disease—before the introduction of vaccination the most fatal of all epidemics—was first described by Rhazes about 900 A. D. It is, in some respects, the most typical of the class of exanthemata.

The *incubation* of smallpox is, as a general rule, twelve days. On the thirteenth day from that on which the contagion entered the patient's body, he is seized with more or less severe symptoms of malaise and fever. Most writers allow that the interval may sometimes be longer; Curschmann extends it in exceptional cases to thirteen, Bristowe, to sixteen days; while Marson affirms that he at least has never found the eruption fail to appear in fourteen days after infection. He admits, however, that the incubation is sometimes shortened by three or four days, and Curschmann alludes to one instance in which it did not last more than five days. Zülzer believes that in the hemorrhagic form of variola it is constantly from six to eight days. During this period patient generally feels well; but occasionally he complains, from the very first, of a vague malaise, with gastric disturbance, headache and giddiness, and toward the end there may be a little pharyngeal catarrh, with reddening of the uvula and tonsils.

The *initial stage*, which follows the incubation, sets in with a severe rigor, or with a succession of slight chills. The temperature at once begins to rise, and may reach 102° to 104° within twenty-four hours, and 105° or even 107° by the second day. In some cases the patient becomes rapidly prostrate, and totters if he attempts to walk or to stand. Curschmann remarks that whereas a working man attacked with enteric fever will often come on foot to the hospital at a time when his temperature is already high, one who has smallpox must always be carried, unless, indeed, the disease is mild. Such a patient, while he is out of bed, has cold limbs, a pale, sunken countenance, and a small pulse, so that he is hardly to be recognized a few hours later, when he has become warm, and when his face is red and turgid. The pulse is much accelerated, varying from 100 to 120 in men, while in women it may reach 130 or 140, and in children 160. The breathing is quickened and is short and labored. The skin is generally dry, but sometimes moist, or even sweating. Some medical men, among whom was the late Mr. Stoker, believe that it already emits a peculiar odor. There is no doubt that the breath is fetid, and the tongue is often very foul. Anorexia and thirst are present. The patient almost always complains of pain across the forehead or over the whole head; and this may be so intense as to make him cry out. Children are not infrequently attacked by epileptiform convulsions; or they may become delirious or comatose; or they may be constantly grinding their teeth. They often have diarrhœa; but in adults constipation is usually present. Curschmann says that in the more severe cases a marked enlargement of the spleen may be detected.

So far, there is little to distinguish the early period of variola from that

of some other diseases. But one symptom rather peculiar to it is vomiting, attending with violent retching and pain at the epigastrium, and sometimes so severe and persistent, that Curschmann mentions a case sent up to the hospital with the provisional diagnosis of volvulus. Another symptom, still more characteristic, is pain in the sacrum and loins. This, however, is not very constant; Curschmann says it occurred in rather more than one-half of his cases, and that in only one-third of them was it sufficiently intense to give rise to spontaneous complaints. It appears to be especially frequent in the more severe forms of smallpox. Women are apt to suppose that it merely indicates the approach of menstruation; or, if they are pregnant, that labor is about to set in. And, as a matter of fact, the catamenia do frequently appear during this stage; sometimes prematurely, but often in their regular course. In men the pain in the loins is likely to be mistaken for that caused by gravel or some other affection of the kidney, or for lumbago. Curschmann has often seen it accompanied by pains in the limbs, so that the question of acute rheumatism has had to be taken into account in the diagnosis.

Another set of symptoms, which are of importance because they might cause the disease to be mistaken for measles, are those of common catarrh, sneezing, epistaxis, intolerance of light, lachrymation, sore throat, and hoarseness. The tonsils and the palate may be seen to be reddened.

Lastly, the initial stage of smallpox may be attended with two distinct forms of cutaneous rash, which differ in their characters and still more in their significance. One of them is, I think, best described as the *roseola variolosa*, a name employed for it long ago by Rayer, and in 1853, by Eimer. To attempt to trace back the history of the knowledge of this remarkable eruption is, indeed, almost useless, because, until recently, most observers regarded it as a proof that one of the other exanthemata was present, in addition to the variola. Reinbold, in 1840, seems to have originally propounded in express terms the doctrine that it is only a preliminary symptom of that one disease alone. In this country, Dr. Wilks, I believe, deserves the credit of having first drawn attention to it, in the "*Guy's Hospital Reports*" for 1857 and for 1861. Watson, however, also mentions it in 1857. But Th. Simon, of Hamburg, is the writer who has most fully described it, and has recorded the largest number of cases. As a rule, it comes out on the second or the third day; but sometimes it immediately follows the initial rigor, and sometimes it is the very earliest sign that anything is amiss with the patient. There seem to be two varieties of it. One is maculated, and is more or less like measles; it may come out all over the body, perhaps appearing first upon the face. The other is an evenly diffused red blush, resembling scarlet fever, but (according to Simon) darker and of a more bluish tint. This often has a distribution peculiar to itself, which was, I think, first pointed out by Hebra. It is then limited to a triangle having for the base a horizontal line drawn across the abdomen about the level of the umbilicus between the two iliac spines, while the apex is formed by the contact of the two thighs. Or it may also be visible in the axillæ, and on the adjacent parts of the upper arms and of the chest. And on the limbs it may affect the extensor surfaces of the elbows and of the knees, the backs of the hands and fingers, as far as the first pharyngeal joints, and the back of each foot in a line corresponding with the course of the extensor tendon of the great toe. Its occurrence in the pubic and inguinal regions especially is so characteristic, that it enabled me several years ago to diagnose correctly a case in which there was no other reason to suspect smallpox. Even if no other eruption should afterward develop itself, one ought probably still to maintain such a diagnosis, for Simon has recorded an instance of this kind in which the disease aborted, notwithstanding that its real nature was

proved by the patient's sister being attacked by variola at the very same time. Sometimes, however, the seat of a *roseola variolosa* is irregular. We have in our museum at Guy's Hospital, models of a case in which it affected only the flexor surfaces of the forearms; and Curschmann has seen it in women limited to the skin about the nipples. It often lasts for a day or two, and sometimes not longer than a few hours. But in a great many cases it assumes a petechial form; and leaves green or brownish stains which may remain visible the whole course of the disease. A point of importance is that, even when it thus becomes hemorrhagic, the prognosis need not be modified in an unfavorable direction. Hebra and Trousseau seem to have independently remarked that the parts affected by a *roseola variolosa* afterward remain free from the proper smallpox rash. All subsequent observers have confirmed this statement, reserving, however, some few cases as exceptions to it.

Very different is the second form of initial eruption, which, in fact, constitutes the most fatal of all the varieties of the disease,—the *Variolæ nigræ* of older writers, now generally known as *Purpura variolosa, Malignant smallpox, or Hemorrhagic smallpox.*

After the usual early symptoms have been present during from eighteen to thirty-six hours, there appears on the trunk and the limbs a diffused scarlet redness, which at first can be made to disappear by pressure with the finger. This soon becomes the seat of most extensive effusions of blood, in spots and patches of all sizes and shapes. Large black rings now form round the eyes; the conjunctivæ are ecchymosed (this, according to Marson, is often seen very early in the case and is always a most dangerous symptom); the whole face may be red and swollen. The tongue is thickly coated. The breath has a horrible fetor, from the presence of a diphtheritic affection of the throat. Hemorrhages may occur from any or all of the mucous surfaces. Bloody liquids are coughed up or vomited, or discharged from the uterus or the bladder; or there may be epistaxis, or a sanguineous flow from the eyes or the ears. The urine is extremely fetid, and it is albuminous even when it contains no blood. The patient generally complains of severe pain at the præcordia, and suffers severely from vomiting. Yet, according to Curschmann, the temperature is seldom over 104°; or this is the case only just before the fatal termination. Both he and Marson remark that consciousness is generally retained almost to the very last; "few patients are so fortunate as to become quickly delirious or comatose." Anæsthesia or hyperæsthesia of certain parts of the surface, and paralytic affections of the limbs, are said to have been observed by Zülzer. Before death the whole body becomes black or of a leaden gray color. According to Marson the smallpox eruption is nearly always confluent in cases of this kind; but, as all writers point out, they often end too rapidly for such an affection to appear. I have, however, seen at least two or three instances in which, although the disease was prolonged for several days, not even papules could be discovered. The first case that impressed this fact upon me was one which occurred in the clinical ward of Guy's Hospital. The patient lived several days, and as no sign of a proper smallpox eruption could be detected, I came to the conclusion that it was not an example of *purpura variolosa;* but a few days afterward the clinical clerk who watched the patient fell ill with a mild form of smallpox. It has often been remarked that strong, muscular men are particularly apt to be attacked with fatal hemorrhagic smallpox; but Curschmann says he has often seen it in delicate persons, in drunkards, and in women recently confined, or in those who are pregnant, and who of course at once abort. According to Marson the blood in this variety of smallpox is "poisoned from the very first, and is rendered very fluid and watery." German pathologists, how-

ever, lay some stress on the fact that in such cases the liver, the spleen, and the kidneys are found after death to be neither swollen nor softened, and that the substance of the heart is firm and of a dark red color.

Hitherto I have said little or nothing about variations in the intensity of the symptoms during the normal initial stage. But sometimes they are very slightly marked, or even altogether absent; the smallpox eruption then appears after a few hours' malaise, or is actually the first sign that the patient is ill. In such cases the disease itself is always very mild; so that Trousseau is not quite right in saying that the greater the duration of this period the less serious is the attack. As a rule, the *third* day is that on which the eruption comes out; but in children it is often the second day. Sometimes, however, nothing is to be seen until the fourth day; and then, again, the prognosis is comparatively favorable. A postponement until the sixth or the seventh day seems to occur only when there is some complication, as in a patient of Trousseau's who had choleraic symptoms. Unlike a slight initial stage, one which is severe is no guide as to the ultimate progress of the case. In women and in children it often happens that the most alarming symptoms are present during the first day or two, notwithstanding that the rest of the disease is to be of a mild character. The most extreme instances of this are afforded by a form of smallpox which was long ago described by Sydenham under the name of *Febris variolosa*, and which de Haen termed *Variolæ sine variolis*. After a well-marked initial stage the disease aborts, and the patient is well in three, four, or six days at the latest. I have already referred to one instance of this kind, which was attended with a characteristic roseola. Another example of it is thus recorded by Marson. A lady walked with a person already affected with smallpox. Twelve days afterward she was taken ill; she was, for a few hours, delirious, but her illness passed off without eruption; twelve days later still, her sister, who had not been out of the house for three months, was attacked with the same disease, which ultimately assumed a confluent form. Curschmann tells of a woman who was seized with shivering, fever, headache, and pain in the back, so that, as variola was epidemic at the time, she seemed without doubt to be passing through the initial stage; but on the fourth day defervescence occurred, no rash could be detected, and by the tenth day she felt perfectly well; however, she gave birth to an infant, which was then covered with an early eruption of smallpox. It afterward suppurated and proved fatal.

The Eruption.—The proper variolous eruption is first papular, then vesicular, and finally pustular. The papule is round and smooth; at first it may be scarcely redder than the rest of the skin, and as it is hard, like a small shot, it can often be felt better than it can be seen. But after twenty-four hours it is always decidedly reddened, besides having increased in size. Unlike the papule of measles and of most other exanthemata, it depends upon a definite change in the superficial and middle cells of the rete mucosum, which, from the very commencement of the morbid process, are swollen and opaque. And in their midst exudation quickly takes place, so that by the end of two days the horny layer of the epidermis is raised to form a minute conical vessel. By the fourth or fifth day of the eruption (seventh or eighth of the disease) the vesicle is generally as large as a split pea, hemispherical in form, and opaline in appearance. As a rule, the *pock* (as it is termed) has a central depression or umbilicus. The origin of this has been much discussed; it generally seems to depend upon the fact that the original papule was developed round the mouth of the hair sac, or else round that of a sweat gland, either of which structures may afterwards form a *retinaculum*, tying down the roof of the vesicle in the middle. Rindfleisch gives a drawing of a preparation in which a sudoriparous duct is plainly to be seen perform-

ing this office, and he says that he has many such specimens in his possession. However, it would seem that this explanation is not always applicable, since the pock does not necessarily bear a definite relation to any of the canals which traverse the cuticle. In all probability a similar function is then discharged by one of many other bands which cross the upper part of every vesicle in a direction more or less vertical, dividing it into a number of separate chambers. This *loculated* character of the pock attracted notice long before its nature was understood; it affords the reason why only a small part of the fluid is evacuated when a needle is introduced into the roof at a single spot. But the idea of the older observers was that the septa between the chambers were new formations, consisting of fibrin. Sir Thomas Watson, for instance, speaks of a "central whitish disk," consisting of "several little cells;" and this, he says, may, by careful dissection, be taken out entire, with the "lymph" which it contains, even when the rest of the fluid in the pock has become yellow and purulent. Auspitz and Basch, however, showed, several years ago, that all the septa in question are in reality formed out of the original cells of the rete mucosum, small bundles of which cohere together, and become stretched out into filaments and bands, as the exudation accumulates around them. In this fluid leucocytes are present in small numbers from the very first; they go on increasing, and it gradually passes from transparent serum into opaque pus; the change is complete in about six or seven days from the first appearance of the papule; that is, in the earliest part of the eruption, by the ninth or tenth day of the disease. The pustule, when at it height, is often quite hemispherical, the umbilicus having disappeared in consequence of the rupture of the retinaculum which formed it.

But while the roof and the cavity of the pock are thus being developed out of epidermic structures, a morbid process is at the same time going on more or less actively in its floor, which, of course, consists of the papillary layer of the derma, with at least the lowest cylindrical stratum of the rete Malpighii. Whether a swollen state of the papillæ contributes to the formation of the original papule of the disease appears to be doubtful; but there is no doubt that its redness is due to a hyperæmia of them; and (according to Bärensprung) this extends down through the whole thickness of the skin. The exudation which fills the vesicle and afterwards the pustule, is necessarily derived from these vascular tissues. But Curschmann says that, so far from the papillæ being always enlarged at this stage, they are often rather flattened by the pressure to which they are subjected, and may even be, to some extent, permanently atrophied by it. In some cases, however, they become the seat of an infiltration of leucocytes, which is so intense that it obliterates their blood vessels, and, indeed, destroys their structure completely, converting them into a white or ash-gray substance. German histologists, in accordance with their usual terminology, describe this form of pock as *diphtheritic* (*vide supra*, pp. 73, 74). The contrast is the greater because the surrounding skin is of a bright red color for a considerable distance, making what is termed the *halo* or *areola*. The infiltration of leucocytes into the floor of the diphtheritic pock may extend to a varying depth in the derma, or even through it into the looser tissue beneath. Rindfleisch gives a drawing from an injected preparation, in which the affected area had failed to receive any of the coloring matter.

Many of the pustules of smallpox undergo destruction almost as soon as they are fully formed. They break, or are ruptured, giving exit to a honey-like matter, which collects, in drops, upon the face and upon other exposed parts of the patient's skin, and which saturates his shirt, his pillow, and his sheets. Others, however, remain uninjured, and ultimately dry up without discharging their contents. First, a yellow-brown spot appears in the roof of

the pock; this sinks in, so as somewhat to resemble the earlier umbilicus; gradually it enlarges and extends to the periphery. The process of desiccation, both in ruptured pustules and in those which are entire, begins about eight or nine days after their first appearance in a papular form. The crusts vary in color from yellow to brown, or even black; this depending upon the admixture of more or less blood with the pus of which they are formed. It is now that the extent to which the cutis vera has been involved in the inflammation affects the result. If it has escaped, the crusts fall off in four or five days (about the fifteenth or sixteenth day of the disease), leaving purple-red stains, but little or no signs of actual cicatrization. But if pus cells have infiltrated the papillæ, or the whole thickness of the skin, these structures, to whatever depth they may have been affected, slough away and become detached, as shreds, adhering to the under surface of the crusts. Their separation takes longer, and may not begin until the eighteenth or twentieth day. Moreover, when it occurs, granulating surfaces are exposed which may take a considerable time in healing over. The resulting cicatrices are for a time of a brown color, but ultimately they become whiter than the skin around them. They may either be so faint as to be scarcely perceptible, or more or less deeply scarred and pitted. It often is not until many months after recovery from the disease that they become depressed below the surface to the full extent.

So much for the appearance and structure and course of the individual pocks of variola; the characters of the eruption as a whole have still to be considered. Like the rest of the exanthemata, it does not develop itself over the whole of the body at once; and it resembles others among them in appearing first upon the face and scalp, especially on the forehead, and about the eyes, the nose, and the upper lip. Marson also mentions the wrists as among the earliest parts affected. A few hours afterward it is to be seen upon the trunk and the arms; this writer says that on the legs and the feet it is generally two days later than elsewhere. He also lays stress on the fact that the papules come out "in threes and fives, forming crescents," or even a complete circle if two crescents happen to coalesce. During the first day or two, fresh spots keep appearing, even on those parts which are already more or less thickly covered; but by the end of this time the eruption may be said to be complete, even if a few more should afterwards show themselves, they soon abort and die away. A curious circumstance is that smallpox specially affects any parts of the skin which may happen to have been recently irritated; the red patch from a mustard plaster applied during the incubation or shortly before, will present many more pocks than the regions adjacent. Curschmann mentions the case of a man who was infested with clothes lice, and whose body was consequently covered with scratches; on his becoming affected with a mild form of variola, these scratches were found covered with pustules, which were closely arranged in lines, like strings of pearls, some of them three or four inches in length.

Certain *mucous membranes* take part in the eruption of smallpox, but not in quite the same way as the skin. There first appear raised spots, whitish or gray in color, contrasting with the reddened state of the surface around them, but these quickly pass into excoriations. They are sometimes present in large numbers on the inside of the lips and of the cheeks; sometimes they rather affect the tonsils and the palate, which may then become greatly swollen and the seat of deep phlegmonous inflammation, ending in abscess. On the other hand, the tongue very seldom presents any traces of vesicles, but sometimes it is enormously enlarged, protruding from the mouth, rendering the patient unable to close his jaws, and apparently helping to bring the disease to a fatal issue. The air passages are said to show more or less distinct pustules as low as the

bifurcation of the trachea, or even (according to Wagner), down to bronchia of the second or third order; there may also be deep ulcers in the larynx, with perichondritis and necrosis of cartilages, œdema of the ary-epiglottidean folds, etc. In the alimentary canal nothing resembling a pock can be seen below the upper part of the œsophagus, except perhaps in the rectum, close to the anus. They are also said to occur at the entrance of the vagina. Marson speaks of twenty-six cases at the Smallpox Hospital out of a total of over fifteen thousand, in which the conjunctiva showed a pustule, which was generally situated midway between the cornea and the internal canthus. It did not in any way affect the sight or lead to any ill result.

Varieties.—Before I attempt to describe the general symptoms of the eruptive stage of smallpox, I must distinguish certain varieties of the disease which have long been known to physicians by separate names, and the recognition of which is of no little practical importance. Roughly speaking, the severity of variola depends upon the number of the pustules, but for obvious reasons the differences between different cases in this respect could not be directly used to arrange them into groups. It is, therefore, most fortunate that in the relation of the pustules to one another we have a criterion which, while it is itself based upon their number, affords a natural division of the most marked kind. Whenever they are so crowded that they run together the disease is said to be *confluent*. This may either be the case over the whole of the body, or only over certain parts of it, or even on but a single region, which is then always the face, since the eruption is never more abundant than upon the face. On the other hand, if the pustules remain everywhere distinct from one another, the smallpox is described as *discrete*. It will be noticed that what constitutes the boundary line between the two forms is in reality the state of the face alone; I believe that there is very seldom any difficulty in drawing it sharply and decisively. Watson, indeed, speaks of some cases in which the pustules stand just thick enough to touch one another without absolutely coalescing, and would then call the disease *cohering*. And Trousseau alludes to instances in which a few isolated patches are found confluent, but which he would term discrete.

In ordinary discrete, as well as in confluent, smallpox, the pustules run through all their stages, unless, indeed, the disease should happen to be cut short by the patient's death. But there is a third variety, consisting partly of discrete and partly of confluent cases, which is characterized by the premature subsidence or abortion of the eruption. For this, unfortunately, there is no satisfactory name. German writers* describe it under the name of "*variolois*," which is etymologically incorrect, since it is not like variola, but is a mere modification of that exanthem and capable of generating the severest form of it by contagion. In this country it is generally termed *modified smallpox*, because it is comparatively seldom seen except in persons who have already passed through a former attack, or who have been vaccinated. But it is important to know that variola sometimes passes off in exactly the same manner in a patient who is altogether unprotected. Probably this depends upon some peculiarity in his predisposition, whereby his susceptibility to the

* Strictly speaking I think that this is not quite correct. It is true that German writers give *variola modificata seu mitigata* as a synonym for their "*variolois*." But in practice they confine the use of the latter term to cases of a certain moderate degree of severity, and apply it to all such cases, whatever may be the course of the individual pustules. Thus Curschmann speaks of *variolois* as sometimes leading to destruction of the papilla and to the formation of deep cicatrices, "so that what distinguishes the case from one of *variola vera* is merely the very much smaller number of pustules." We should call such a case one of very mild discrete *un*modified smallpox. On the other hand, I do not find German writers mentioning under *variolois* the examples of modified confluent smallpox which will presently be described.

contagious principle is diminished without being annulled. Trousseau mentions a case which goes far to establish such a view. A person had been vaccinated (as I presume, for the first time), but the operation did not take effect. Some time afterward, when the doctor was thinking of repeating it, he was called to see the patient, whom he found to be suffering from a discrete smallpox which ran a modified course. I do not know that the *confluent* form of the disease ever runs this modified course in those who have not been vaccinated and who have not previously had variola.

1. In *discrete smallpox* the eruption presents in their most typical forms the characters which have been described as belonging to the individual pustules. Its amount, of course, varies infinitely in different cases, from a few spots that can almost be counted on the fingers, up to many thousands. At the time when they are acquiring their areola there is often a considerable degree of swelling of the surrounding parts, especially when the subcutaneous tissue is loose. Even three or four pustules upon the eyelids may cause them to be puffed out like bladders, so that on the ninth and tenth days the patient may be unable to open his eyes. Trousseau cites a case of Van Swieten's in which a single pustule on the prepuce produced phimosis and great difficulty in micturition. He remarks that the absence of a corresponding degree of tumefaction of the rest of the face renders the swelling of the eyelids more conspicuous in some cases of discrete smallpox than in the confluent variety of the disease. At about the same time the pustules first begin to be painful.

As regards the general symptoms of discrete smallpox, the first thing to be noticed is that as soon as the eruption has fairly begun to develop itself the fever which has existed during the initial stage subsides, and the temperature falls within thirty-six hours to normal, or even lower. Its decline is not always quite continuous, being perhaps interrupted by a slight evening exacerbation. At the same time the pain in the back and the sickness disappear, and the patient often feels perfectly well, with as good an appetite as when in health. This lull in the symptoms commonly lasts for three days; at the end of it he is, in all cases except those in which there are but very few pustules, again attacked with shivering and with febrile disturbance, which is known as the *secondary fever*, or the *suppurative fever*, or the *fever of maturation*. The date at which this sets in seems to be differently stated by writers; Trousseau puts it at the seventh or eighth day of the disease, Watson at the eleventh. It is generally remittent in type, the daily variations amounting to one or two degrees Fahr. The height to which the thermometer now rises varies with the extent of the inflammation in the cutaneous tissues, and therefore roughly with the number of the pustules; even in severe cases it is seldom above 102° or 103°. The pulse is quickened, being at from 110 to 120. The patient at the same time complains of headache, and is restless and sleepless. He is not unfrequently delirious, especially during the first night or two.

It must not be supposed that discrete smallpox is always unattended with danger. Trousseau relates the case of a girl, aged twenty-one, who had passed through a remarkably mild attack, but who was one evening suddenly seized with cerebral symptoms and difficulty of breathing, and in an hour she was dead; and he remarks that when this form of the disease does prove fatal, death occurs at an earlier date than in the confluent form, namely, about the eighth or the ninth day.

2. In *confluent smallpox* the eruption necessarily fails to some extent to present its typical characters, at least upon those parts of the body where an actual fusion of its elements has taken place. Even before any definite papules can be recognized, there is often a diffused redness of the face, which in itself could hardly be distinguished from the rash of measles. Watson

mentions a case in which the appearances of urticaria, with its characteristic sensations, were at the outset so intermingled with the papules of variola that for twenty-four hours he doubted which of the two diseases was developing itself; and in other instances smallpox has been mistaken for erysipelas. Trousseau remarks that even where there are, in fact, multitudes of papules, the complete absence of intervals between them may render one almost unable to detect any unevenness of the surface by passing the hand over the patient's forehead or cheek. The papules are always smaller than in the discrete form; they coalesce so as to form the most irregular figures and patterns. At a very early period a milky fluid collects in their summits, giving rise to minute flat vesicles, which have all sorts of zigzagged shapes, or may even run together into a kind of open network, with intervals here and there of healthy skin. The whole of the face then has a whitish-yellow color, and its appearance is exactly like that of a mask of parchment, with which substance Morton, in his "Pyretologia," long ago compared it. By this time, however, there is a good deal of swelling, and it goes on increasing up to the ninth day of the disease, and until the features are so altered that it is impossible for the patient to be recognized by his nearest relations. The parts about the ears and the sides of the neck become enormously bloated; the eyelids cannot be opened. This condition is necessarily very painful, and the scalp is often so tender that the pressure of the head upon the pillow can hardly be borne.

Somewhat later, about the eleventh or twelfth day, the *hands* and the *feet* become red, swollen, and painful. Trousseau, following certain of the old writers, attached a high prognostic value to this symptom; according to him its absence is almost invariably followed by the death of the patient: in fact he had only seen three instances of recovery from a confluent form of the disease without it.

The *mucous membranes* generally suffer severely in confluent smallpox. The interior of the mouth not only presents the eruption already described, the fauces may become covered with a diphtheritic membrane. Laryngitis often renders the voice very hoarse, and it sometimes causes a sudden fit of suffocation, which proves fatal in a few seconds, before anything can be done to relieve it. Trousseau mentions three cases of this kind, in each of which death seems to have occurred quite unexpectedly on the eighth day of an illness that had previously run a perfectly normal course.

Another complication, perhaps due to extension of inflammation to the parotid gland, is *salivation*. Trousseau speaks of this as a characteristic feature of confluent smallpox. It begins, he says, about the fourth or fifth day, and goes on increasing, until by the ninth or the tenth day one or two quarts may run from the patient's mouth within the twenty-four hours. Even during sleep there is a constant flow of saliva. Again, ophthalmia is of frequent occurrence, which (unlike the harmless discrete pustules of the conjunctivæ) is often attended with sloughing of the cornea, and leads to permanent blindness.

From the very commencement of the eruptive period, the general symptoms of confluent smallpox run a course which differs from that of the discrete form of the disease. Instead of the temperature falling when the papules come out, it remains at 103° or 104°. There is often violent *delirium*, even during the daytime. It may be quite impossible to keep the patient in bed, except by tying him down with a folded sheet across the chest. If free, he must be most carefully watched, or he is very likely to throw himself out of a window or over the stairs, or to cut his throat with a razor. In those who have been intemperate, the nervous disturbance often assumes the form of delirium tremens.

Retching and vomiting commonly run on throughout this period of the

disease; and diarrhœa is often obstinate and troublesome. The urine contains albumen in a large number of cases.

When suppuration is established, there is a further rise of temperature. Indeed, it becomes higher than in the corresponding stage of discrete smallpox, although from the patient having all along been feverish, the development of a fever of maturation is less conspicuous.

This is the most dangerous period of variola. Few patients die before the eleventh day; the most fatal days are the twelfth, the thirteenth and the fourteenth. Toward the last there is generally coma; sometimes hyperpyrexia is present, as in a case of which Wunderlich gives a chart, and in which, before its termination on the eleventh day, the thermometer registered 109°. Again, in many instances the immediate cause of death is bronchitis, pneumonia, pleurisy, or pericarditis. Even under the most favorable circumstances, the fever runs on for at least ten days longer. Not infrequently, during the third week, the patient falls into a typhoid condition, with sordes, a dry, brown tongue, muttering delirium and subsultus; and he is then very likely to die of exhaustion.

When recovery is to take place, the redness and the swelling of the face subside, as the pustules undergo conversion into crusts. The pain from which the patient has been suffering passes off, but only to be followed by the most intolerable itching. The fever also declines more or less rapidly, being now proportionate to the amount of inflammatory action which is still going on. He regains his appetite, and becomes once more able to sleep. He opens his eyes, and gradually his features begin to re-assume their natural outlines. At the time when the crusts are falling off, or a little later, he generally loses almost all his hair, and if the deeper layers of the skin of the scalp have been involved in the morbid process there is always reason to fear that permanent atrophy of the hair sacs may result.

3. I have still to describe two minor varieties of smallpox, which, on account of their dangerous tendencies, deserve to be ranked with the confluent rather than with the discrete form of the disease. One of them is characterized by the occurrence of hemorrhage into the pustules. This must not be confounded with the *hemorrhagic variola*, which proves fatal at an early period before the proper eruption has begun to develop itself. By way of contrast, Curschmann calls it *variola hæmorrhagica pustulosa*. According to that writer, the pustules generally attain their full size before any blood is effused into them; but sometimes this begins even during the papular stage. As a rule, those pustules which are seated upon the lower limbs are the first to show a purple color. Bleeding presently takes place from the nose, kidneys, intestines, and other mucous surfaces. In women menstruation sets in; or abortion occurs if they are pregnant. The gums become spongy and bleed, as in scorbutus; the fauces show a kind of "diphtheritic" affection which is attended with a terrible fetor. The constitutional symptoms are in most respects severe; but although the pulse is very rapid the temperature during the eruptive stage is seldom above 102°, and it not rarely falls to 95°, or even lower, before the patient's death. Curschmann says that this variety of smallpox occurs chiefly in persons over forty years of age, and in such as were previously out of health. He describes it as being almost always fatal. He points out, however, that in patients who have, in consequence of delirium, got out of bed and walked about during the early part of the eruptive stage of smallpox, the pustules on the legs may become filled with blood, without there being anything particularly serious about the case; and I think I recollect at least one case in which a similar appearance of the pustules on the forearms has brought with it no evil consequences.

4. Another and a very rare variety of smallpox is that which is called

corymbose. I have never seen an instance of it. Marson describes it as generally presenting two or three patches or clusters, of the size of the hand, upon which the eruption is as thickly set as it possibly could be, while the skin around is, for some distance, almost, if not entirely, free. They are often developed symmetrically upon corresponding parts of the limbs. Sometimes there are numerous clusters, about as large as half-crowns, seated on different parts of the body. Elsewhere the pustules may be but sparsely scattered, and one would have expected the disease to be attended with little danger. The contrary, however, is the case. Marson collected from the register of the Smallpox Hospital one hundred and four cases of this kind which had occurred during a period of thirty years; and he found that there was a mortality among them of no less than 41 per cent. Even if only a single cluster was formed, the disease yet manifested its fatal character, and, what is perhaps most remarkable of all, it was scarcely less destructive to vaccinated persons than to those who were unprotected. In many cases the patient died from some complication when he had appeared to be doing well; and, at the best, his convalescence was long and tedious.

5. In *modified smallpox*—which, as we have seen, is not exclusively confined to protected persons—the course of the eruption is much less regular than in the ordinary forms of the disease, whether discrete or confluent. During the initial stage, however, no distinction can be drawn between it and them; no doubt there is a larger proportion of cases in which that stage is but little marked; but in some instances, at least, it lasts the usual time, and is attended with severe symptoms. But when the papules begin to appear, it is said that the characteristic irregularity is often shown by their being visible upon the trunk or elsewhere before there are any of them on the face; and it is also said that the period during which fresh ones develop themselves is variable, the number being sometimes complete in a few hours, while in other cases they keep coming out even when those which were first formed have already become pustules. And what appears to be still more indicative of this variety of smallpox is the course of the fever at the commencement of the eruptive stage. The temperature falls, as in unmodified discrete variola, but with still greater rapidity, and without any interruption in its downward path, so that in less than twenty-four hours it is normal. Trousseau and others, however, speak of confluent smallpox as running its normal course for ten or twelve days, and then suddenly subsiding, so as to show that it in reality belongs to the modified variety of the disease.

It is thus evident that no definite period can be fixed at which the modified character of a given case of smallpox first becomes apparent. Sometimes the eruption dies away before it has passed out of the papular stage. Sometimes the papules undergo a partial conversion into vesicles, which then rapidly dry up. A special modification of this variety of the affection has long been known as the "horn-pox" (*variola verrucosa*); but writers by no means agree in their descriptions of it, for while Trousseau speaks of the vesicles as drying up and leaving in their places small, hard, corneous projections, which fall by a sort of desquamation, between the tenth and the fifteenth days, Curschmann says that after the scabs have become detached, the solid bases of the pocks remain for a considerable length of time as warty elevations, especially on the face, to which they give an ugly appearance.

Again, in yet other cases of modified smallpox, the vesicles pass on into pustules. The change then appears to take place unusually rapidly, and the pustules themselves are often extremely small, and probably never reach so great a size as that to which they may attain in the unmodified forms of the disease.

The *complications* of smallpox are comparatively unimportant. They mostly concern the respiratory organs; and bronchitis is frequent. Œdema of the larynx has occasionally proved fatal.

The *sequelæ*, besides the pitting, are chiefly various forms of local suppuration, like those which follow enteric and scarlet fevers; abscesses in the skin and deeper organs, furunculi and ecthyma, local gangrene and phlegmonous erysipelas. In severe cases the cornea may ulcerate, and the eye be lost.

Convalescence is, however, usually rapid, and the health and strength are not impaired as after typhoid fever.

The *prognosis* depends first upon the number and quantity of the marks of vaccination; and secondly, in unmodified cases, upon the eruption being slight, discrete, hemorrhagic, or confluent.

In the *treatment* of this disease, the special points to bear in mind are the importance of immediate isolation on account of the virulence and persistence of its contagion; and the fact which seems established that, in unprotected persons, vaccination has some power in modifying the severity of the disease, even if performed after the presence of variola is recognized.

VARICELLA.

HISTORY OF THE RECOGNITION OF CHICKEN-POX—ITS DISTINCTION FROM SMALL-POX—VARIETIES OF THE ERUPTION—SYMPTOMS AND COURSE—A DISEASE OF CHILDREN—DIAGNOSIS—PROGNOSIS AND TREATMENT.

At the end of the seventeenth century, soon after the final separation of measles from smallpox by Sydenham, two other English writers, in treating of the latter disease, mentioned a variety of it as being popularly called "chicken-pox." In all probability the allusion in that name is to *chickpease* (French *chiche*, Latin *cicer*). The same affection seems to have been described nearly one hundred and fifty years before, by Vidus Vidius, and by Ingrassias, under the designation of "Crystalli." Vogel (1764) is said to have introduced the name of Varicella (dimin. of *varus*, a pimple). In 1730, Fuller asserted that chicken-pox and smallpox were really distinct diseases; but Heberden, in the first volume of the "*Medical Transactions of the College of Physicians*" (1767) first indicated fully the differences between them. He also pointed out the chief reason which made the recognition of chicken-pox a matter of importance, namely, that those who had it might otherwise be deceived into a false security, "which might prevent them either from keeping out of the way of the smallpox or from being inoculated."

After the introduction of vaccination, it became of great consequence to distinguish the two diseases, since every case of varicella occurring in a vaccinated person would otherwise have been regarded as an instance of the failure of the operation. But it is to be regretted that some of the earliest advocates of Jenner's method, being anxious to show that the protection afforded by it was absolute, referred every suspicious-looking eruption in persons who had been vaccinated to chicken-pox. This, in its turn, led to a reaction, since it necessarily involved the giving up of all the distinctive characters of the affection, and the consequence has been that its claim to be regarded as an independent disease has been disputed by several eminent physicians, from Dr. John Thompson, of Edinburgh (1820) to Hebra, of Vienna. Unfortunately, too, the great German dermatologist has helped to throw the subject into confusion by employing the term varicella in an entirely new sense, namely, for all very mild cases of smallpox. Thus, when he says that "varicella" may generate variola, he really means nothing that would not be admitted by every one. It is, however, perfectly certain that the varicella of other writers is altogether unconnected with smallpox.

The proofs are (1) that it occurs in those who have been vaccinated, or who have had variola, just as readily and with the same characters as in those who are unprotected; and (2) that a person who has passed through it remains as susceptible as before to the vaccine virus or to that of smallpox. In the "*Lancet*" for 1877, a case is recorded of an unvaccinated child, who was admitted into St. Thomas's Hospital for chicken-pox, but who was placed on the floor containing the smallpox wards, because the diagnosis was at first uncertain. Two days afterward vaccination was performed, which suc-

ceeded. Eight days later still the child fell ill with modified variola. According to Thomas, Czakert vaccinated with success a boy in whom the inoculation of the cow-pox had failed on three previous occasions, by introducing the lymph into the interior of the vesicles of a varicella with which he happened to be attacked. Indeed, if it were not that, at the present day, almost every one is vaccinated in infancy, we should have in the cow-pox virus an almost infallible test, which we could apply at our own discretion, and which would soon clear up all doubts as to the nature of any individual case that might be difficult of diagnosis. I think it is not too much to say that we have really more positive grounds for declaring that smallpox and chickenpox are distinct, than for making the same statement about almost any other two acute diseases. What makes the case the stronger is that, whereas the fluid from even the mildest eruption of variola is capable of conveying the disorder by inoculation, almost all of those who have tried to inoculate varicella in the same way from its vesicles have failed. Early in the present century Bryce performed this experiment upon children who had never had either smallpox or cow-pox, to the number of thirteen, without any result. It is true that in a long series of cases collected by Hesse, in 1829, there was a small minority in which inoculation appeared to succeed; but according to Thomas there are reasons for thinking that some error crept into these observations. In order to negative one of Dr. Thomson's principal arguments on the other side, most writers cite Möhl's statement that from 1809 to 1823 chicken-pox was annually observed at Copenhagen without concomitant smallpox, "and that afterwards both diseases prevailed at intervals epidemically, but always under circumstances which convinced the physicians of the town that their sources were distinct." But it seems to me that evidence of this kind, based only upon opinion, tends rather to weaken a position which is otherwise altogether impregnable.

Nor are there wanting numerous points of difference from variola in the characters of the eruption of varicella, and in the way in which it develops itself. It begins as a series of small, slightly-pointed red spots, which Trousseau compares with the rose rash of enteric fever; Dr. Gee says that they disappear when the skin of the part is stretched, this being a proof that there is no exudation into the tissue of the cutis, but only a hyperæmia. In a few hours they pass into as many transparent, tense vesicles, round or oval in form, and about as large as split peas. These sometimes have a red base, sometimes they are seated upon a perfectly colorless surface, so that the patient looks exactly as if he had been sprinkled with drops of clear water. They are generally scattered quite irregularly, but it is said that they may be somewhat clustered together, as in herpes. A chief distinction between them and the vesicles of smallpox is their superficial position; they have no thickened floor, and they do not consist of a series of separate chambers; thus they are not umbilicated, and when they are pricked they almost completely collapse. Thomas, however, says that when they first appear this is not the case, and that they are at that time intersected by delicate septa. At length they acquire a yellowish appearance, and the fluid in them turns slightly opalescent, but never actually passes into pus. After from twelve to twenty-four hours they begin to dry up and become flaccid; they comparatively seldom escape being ruptured, either by the nails of the patient (for there is often considerable itching) or in some other way; but they often fall in first at their centres, so that they now have a sort of spurious umbilicus. Ultimately they form thin, brownish-yellow scabs, which in a few days crumble away, leaving reddish pigmented spots, and even transitory and very superficial cicatrices.

The *eruption* of varicella generally appears first on the upper part of the body, on the chest, or (according to Thomas) on the face. Formerly it was

said rather to spare the face; and although this is incorrect, it certainly does not come out more thickly there than elsewhere, as is the case with variola. On the scalp it is almost always present. Afterwards it spreads to the limbs. One of its most important characters, however, is that even on the body the vesicles come out, not in a single crop, but in a succession of crops, which may be prolonged over three or four days, or even a whole week. Thus one sees papules and vesicles and crusts side by side at the same time; in this it is very different from smallpox. The total number of vesicles is sometimes not more than from ten to thirty, but according to Thomas it is generally two hundred or two hundred and fifty; as many as eight hundred are said to have been observed.

The mucous membranes take part in varicella. Thomas says that complaints of pain in micturition have often led to his observing vesicles upon the labia of girls, but that on the prepuce in boys they are less commonly to be found. On the palate they often persist for some time, having slightly reddened bases, but on the lips, the tongue and the cheeks they are to be recognized only in the form of excoriations.

Some of the papules which, as we have seen, constitute the earliest stage of the eruption of chicken-pox, not infrequently abort and disappear, especially those which come out toward the end of the disease. Thomas has recorded a case—the nature of which was established by the fact that the patient's sister had just before had varicella—in which every single papule died away after thirty-six hours, so that no vesicles were formed. On the other hand, the vesicles sometimes increase in size until they deserve the name of bullæ; such an enlargement of them does not always begin until they have already become scabbed over, and they may go on spreading at the periphery of the crusts for a considerable time. Indeed, it would seem that in exceptional instances varicella lasts much longer than any other exanthem. Trousseau speaks of an epidemic in the Necker Hospital, in which, during from fifteen to forty days, blebs like those of pemphigus kept appearing on different parts of the patients' bodies, leaving ulcerations which lasted for six weeks or two months. Mr. Hutchinson ("Lect. on Clin. Surgery") saw a rash exactly like fading chicken-pox, which was said to have been out during a month, and which, in fact, vanished spontaneously, almost immediately afterwards. Some writers speak of relapses of varicella, and Thomas, although he denies that the disease is ever followed at once by a second complete attack, admits that he has seen a few vesicles appear as late as a month from the commencement of the eruption. The question is of some importance, on account of its relation to a remarkable cutaneous affection which Mr. Hutchinson believes to arise out of chicken-pox, and which he therefore terms "varicella-prurigo." He has recorded notes of sixteen cases, all of which are said to have begun with the characters of varicella, most of them having, in fact, been diagnosed by medical men as examples either of that disease or of modified smallpox. But it is curious that it never affected several children in the same family; indeed, there is only one instance out of the whole number in which more than one child suffered from the original disease; and I must confess that my doubts as to the correctness of Mr. Hutchinson's views are strengthened by the fact that he gives twelve other cases in which the same skin disease was supposed to have been caused by vaccination. In fact, I believe it to be an exaggerated form of strophulus.

The general *symptoms* of varicella are very slight. As a rule, there is no prodromal stage, the child feeling perfectly well until the eruption appears. Thomas speaks of having carefully taken temperatures once or twice a day (doubtless in children who had a brother or a sister already ill with the disease), and of having always found them either normal, or at most raised

to the extent of half a degree Fahr., which at an early period of life is of common occurrence, even in health. Thus the whole period up to the outbreak of the exanthem is included in the incubation. This seems to be of variable duration, so far as has been ascertained, but I believe that in general it has been calculated upon the very precarious basis of the interval between the dates at which different children of the same family have been successively attacked. Dr. Gregory stated it from four to seven days; Bristowe says that in some cases it lasts exactly a week, but perhaps more commonly a fortnight; according to Thomas, it may be from thirteen to seventeen days; according to Trousseau, from fifteen to twenty-seven days.

In some instances, however, Thomas found that the child was feverish for a few hours before the disease made its appearance, and sometimes the fever lasted two or three days, and was accompanied with delirium or (as in a case which I saw some years ago) with convulsions. Once Thomas observed a transitory general erythema, with a temperature higher than 106°. In other patients, however, the thermometer failed to rise above normal during the first twenty-four hours, even while the vesicles were coming out. Altogether, he says that in four cases of varicella out of six, there would be fever of two or three days' duration, while in a fifth case it would pass off in one day, and in a sixth case it would last four days, or even still longer. It is particularly to be noticed that the temperature in chicken-pox does not fall, as it does in variola, when the eruption begins to develop itself. Trousseau, indeed, speaks of successive onsets of fever, occurring regularly at night, and accompanying the different crops of vesicles; and Thomas seems to have observed something of the same kind, although he speaks of such exacerbations rather as tending to interrupt the nocturnal rise and the morning fall of temperature which would generully occur in this disease, as in most others. The maximum temperature is about 102°. In some very slight cases there is a complete absence of fever. The defervescence, when it occurs, is usually rapid.

All that need be said about other symptoms is, that the patient may be a little restless or drowsy, but that he seldom complains of headache or wishes to be kept in bed. The tongue is clean, but there may be some thirst and loss of appetite. In a child who was previously suffering from chronic laryngitis, Dr. Gee noticed a decided increase of dyspnœa during an attack of varicella.

I have throughout spoken of *children* as the subjects of chicken-pox, and in truth the disease is almost confined to them. Dr. Gee gives a table drawn up from the records of the Great Ormond Street Hospital, according to which infants under six months of age were often attacked, while there was absolutely a much larger number of cases among children between six and twelve months old, than during any other period of equal length. After ten years of age it very rarely occurs. Most writers say that they have never seen it in grown-up patients, but Heberden relates one case in which a mother caught it from her children, and another instance of it in an adult female was observed by Gregory.

Varicella is sometimes sporadic, sometimes epidemic. Thomas remarks that in large towns epidemics are not separated by intervals of several years (as is the case with measles and smallpox), but occur once every year or once every half year—in Leipzig regularly, a short time after the opening of the infant schools.

This disorder does not affect any particular season. It is highly infectious. The contagion is probably communicable through the air, and is also capable of adhering to solid bodies. That it possesses comparatively little tenacity may, perhaps, be inferred from the fact that the extent of an epidemic is not generally very large.

As a rule chicken-pox does not occur more than once in the same child, but according to Trousseau, second attacks are not uncommon, and Gerhardt is said to have seen it recur for the third time.

I have already indicated the differences in the characters of the eruption and in the course of the fever which distinguish varicella from the milder forms of variola, but it must be confessed that in individual instances a positive diagnosis is not always possible, particularly now that almost all children have already been vaccinated. In our collection in Guy's Museum there are models from three cases, which occurred in 1865, in the same family, of an eruption the nature of which seems to be still open to doubt.

It is a question whether the early stage of vesicular strophulus is to be regarded as distinct from chicken-pox. If so, the points of difference have still to be worked out; and if not, the favorable prognosis which is allowed by all writers must to some extent be modified, for whereas they speak of varicella as always ending in recovery, one of Hutchinson's cases of "varicella-prurigo" terminated fatally. At any rate, I cannot agree with Thomas that it is not worth while to isolate a child affected with chicken-pox from its brothers and sisters.

It does not seem to be necessary to adopt any medical treatment beyond keeping the patient on light diet for a few days, but the vesicles should as far as possible be protected from injury by scratching, lest the resulting scars be made deeper than they otherwise would be.

AGUE.

INTERMITTENT AND REMITTENT FEVERS—INCUBATION—THE COLD, HOT, AND SWEATING STAGES—REGULAR AND IRREGULAR VARIETIES—MALARIAL CACHEXIA—NATURE AND LAWS OF THE CONTAGION—DIAGNOSIS—TREATMENT.

We have now to discuss a disease which seems to stand apart from the specific fevers in some very important respects. In its most typical forms its course is interrupted by definite intervals of apyrexia, so that instead of appearing as a continuous fever it consists rather of a succession of independent paroxysms or "fits," which recur with marvelous regularity. For such cases the name of *intermittent fever* is commonly employed. But they must not be separated from others which are due to the same cause, and in which the subsidence of the pyrexia is only partial, so that they are said to be examples of *remittent fever*. And yet further varieties are met with, to which neither of these terms is applicable. I therefore prefer the homely word "Ague," as fairly taking in them all. Another name sometimes used for them is that of *paludal* or *marsh fevers;* but it is advisable to avoid such expressions, for reasons which will appear when I have to speak of the ætiology.

The poison which excites ague is best called *malaria*, for this term can now be kept strictly to that meaning. It enters the human body from without; but, unlike the virus of most other specific diseases, it never passes directly from one person to another. In other words, ague, though in a broad sense one of the *infective* diseases, is not *contagious*, nor even *infectious* in the narrow sense in which that epithet is generally employed. Whether the poison does not multiply in the blood in such a way that the direct inoculation of the blood of a patient affected with ague upon a healthy individual would convey the disease, is a problem that has not, so far as I know, been hitherto solved.

It is a curious circumstance that malaria appears to be almost without influence upon domestic animals, at least in those countries where its effects have been most studied. In Italy, however, a few instances have been recorded in which horses or cattle have had intermittent attacks of fever, or have suffered from a cachexia attended with enlargement of the spleen, such as we shall see to be another manifestation of the ague poison.

I may note that very complete descriptions of the different varieties of ague are to be found in the writings of Celsus and other ancient writers. The first observer who took a clear view of malaria as a noxious effluvium given off by marshes, appears to have been Lancisi, in a work published at Geneva, in 1716.

That ague has a period of incubation is not a prominent fact in its history, for, as a rule, those who are attacked by it have resided for some little time in a region infested with malaria, and seem to succumb to the influence of repeated doses of the poison, instead of taking the disease on any particular occasion. But when a single exposure alone is known to have occurred, an interval of from six to twenty days is said generally to elapse before the paroxysms begin. Dr. Maclean, however, in "Reynolds' System,"

mentions the case of a party of German missionaries, fresh from Europe, who passed a night in an uncleared and unhealthy spot, at the foot of the Segoor Pass, in India; next morning they pursued their journey, but within less than twenty-four hours three out of four of them were stricken with fever. And Hertz, of Amsterdam, writing on this subject in "Ziemssen's Cyclopædia," declares that on several occasions, having purposely placed himself in a marshy ditch at a time when it was drying up, he has been attacked within half an hour by giddiness, shivering, nausea, and other symptoms, which ended in a slight paroxysm of fever a few hours afterward. On the other hand, it is well known that the onset of the disease may be much longer delayed. Sailors have fallen ill upon the open sea, weeks or months after leaving a port infected with malaria. In London the Irish poor often apply for hospital relief in the spring, on account of an ague which, although only just declared, must have been contracted during their autumnal migrations into Kent for hop picking.

Among strangers who have recently entered a malarial district, and also among persons who have left such a district before being attacked, the occurrence of a regular paroxysm is, according to Hertz, in most cases the earliest symptom of ague. He says that it is chiefly when the patient has been living for a long time in the same place, so as to have repeatedly run the risk of catching it, that a *prodromal* stage is observed. It lasts a week or more, and consists of vague malaise, fatigue, headache, and pains in the limbs, in association with nausea, fullness at the epigastrium, loss of appetite, and other symptoms of disordered digestion. There may also be occasional sensations of chilliness, alternating with slight flushes of heat.

The actual ague fit itself is divided into three periods, which have long been known as the *cold*, the *hot*, and the *sweating* stage respectively.

I. The *cold* stage begins with the patient's feeling tired, weak and listless. He may yawn and stretch out his limbs. He complains of an uncomfortable sensation at the epigastrium, of headache, of giddiness. Before long these symptoms merge into those of a "shiver" or *rigor*. He experiences a chilly feeling along the spine, which quickly spreads all over him. He shudders, his teeth chatter, his knees knock together, his whole frame may be so violently agitated that the bed into which he has crept shakes beneath him. His voice is feeble and his speech is interrupted by the quivering of his lips. At the same time his appearance undergoes a remarkable change. He grows pale, his features shrink, his ears and nose and finger tips turn livid, his skin becomes dry and rough with the condition known as *cutis anserina* or "goose-skin." Yet the thermometer, as de Haen pointed out more than a century ago, shows that in the central parts of the body the temperature is not lowered, but raised several degrees above normal. It must not, however, be supposed that the patient's sensations deceive him as to the condition of the surface, and especially of the more distal regions. One has only to feel his nose or fingers, in order to assure one's self of the fact that they are really cold. And a surface thermometer, or an ordinary thermometer held in the patient's hand, will remain far below the natural standard. But in the axilla, the mouth, or the rectum the reverse is the case. The temperature even begins to rise before any subjective symptoms of the onset of the ague fit is experienced by the patient. At first its increase is gradual; but with the development of the rigor it makes a sudden leap upward, perhaps passing through four or five degrees Fahrenheit in the course of an hour. The patient, during this stage, feels dry and parched; his tongue is white; he may complain of nausea, and may even vomit. His pulse is quick, but it is small and sometimes irregular. His respiration is hurried, but it is short and distressed. His urine, although pale, is often very irritating, so that Dr. Maclean has

found it desirable to administer bicarbonate of potass and even tincture of opium for the relief of this symptom alone. Sir Thomas Watson spoke of the urine as scanty, although passed frequently; but the careful measurements and analyses of Redtenbacher and of Ringer have shown that it is in reality considerably increased in quantity, when compared with the amount passed each hour during the apyretic interval, and that there is also a marked augmentation of the urea and of the chloride of sodium. Indeed, the excess, both of the water and of these solid ingredients, not only lasts throughout the whole length of the fit, but actually begins before the patient experiences any subjective sensations of chilliness, and even a little before the commencement of the rise of temperature.

II. The *hot* stage gradually succeeds the former one. The patient ceases to shiver, and begins to experience flushes of heat about the face and neck, which presently diffuse themselves over his body. But if he attempts to throw off the bedclothes which have been piled up over him, slight rigors for a time reappear. At length, however, even the extremities become permanently hot and glowing. His aspect is now again altered. His face is flushed, his eyes are injected, his skin becomes level and turgid. The temperature, as measured by the thermometer, still continues to rise for some time, and may now reach its maximum, which is generally 104°, but sometimes 106.5°, or even higher. The surface of his body, and even of the distal parts, now feel harsh and pungently hot to the hand. Evidently, there is not only an increased generation of heat, but it is now freely carried to the periphery, and is abundantly dissipated. Accordingly, the pulse is not only rapid, but full and throbbing; and the breathing is deep as well as frequent. The patient complains more than ever of headache, which is now of a throbbing character; and he is exceedingly restless and uncomfortable. The urine during this period is described by most writers as differing in appearance from that of the cold stage, being now high colored and concentrated; and both Hertz and Sir Thomas Watson speak of it as passed in but small quantity. There is often an eruption of herpetic vesicles upon the lips, or the nose, or the tongue.

III. The *sweating* stage, in its turn, arises out of that which precedes it. The surface gradually becomes soft; then a little moisture breaks out upon the forehead and the face, and before long the whole skin is bathed in the most copious perspiration, which saturates the patient's linen and even the bed clothes. It is said that the thermometer sometimes still continues to rise for a little while, so that the maximum temperature may occur during this, and not during the hot stage. But, as a rule, the pyrexia begins to decline from the moment when the skin ceases to be dry. The fall is at first slow, but after half an hour or an hour it becomes more rapid, and goes on until the normal temperature is reached. According to Wunderlich, this takes place by a series of steps, there being a drop of from one-fifth to one-third of a degree Fahr. about every fifteen or thirty minutes. All observers speak of the urine during this period as differing from that passed in the previous stages in showing a thick deposit of urates. The breaking out of the perspiration is attended with complete relief of the patient; he loses his pains, the throbbing of his arteries ceases, he is no longer tormented with thirst, and his tongue becomes moist. After a time he perhaps falls asleep; and, when he wakes, he feels perfectly well, except that he may be somewhat exhausted.

Recurrence.—The subsidence of the ague fit or paroxysm by no means brings the disease itself to an end. As we have already seen, its most remarkable feature is that precisely similar attacks are repeated again and again, recurring at perfectly definite intervals. Some patients have a fit every day; the ague is then said to be *quotidian*. In other cases it occurs every other

day; and this form of intermittent fever is styled a *tertian.* If there are two entire days between the paroxysms, the disease is a *quartan* ague. In other words, an interval of about twenty-four hours characterizes a quotidian, one of forty-eight hours a tertian, and one of seventy-two hours a quartan. Nor does this exhaust all the possible varieties. In some cases in which there is an attack every day, each paroxysm differs in severity from that which occurred the day before, and resembles exactly that which occurred two days previously. Thus a tertian type is clearly perceptible, and the affection is then called a *double tertian.* So, again, it is possible to have a *double*, or even a *treble quartan* ague. Or a patient may have two ague fits one day, and a single fit on the following day, this series being regularly repeated; and one may then suppose that a quotidian and a tertian are combined. Such a case constitutes the ἡμιτριταίος of Celsus, or, as subsequent Latin writers have termed it—a *semi-tertian.*

The return of the paroxysms of ague is often so regular that it can be foretold with absolute certainty. But even then the hour at which the fits begin is not always the same on each occasion. Sometimes it is earlier, each time by a constant difference; sometimes it is later. In the former case, the disease is said to *anticipate;* in the latter case, to *postpone.* And whereas the one is a sign that the severity of the case is on the increase, the other indicates that it is becoming milder. Such variations may even bring about a change of type; thus an anticipating tertian may pass into a quotidian, whereas a postponing quotidian may be converted into a tertian. Sometimes the attacks of a quotidian fever follow one another so rapidly, and are so prolonged, that before the sweating stage of one fit is over the cold stage of the next one begins; the disease is then said to be *subintrant.* With regard to the relative severity of the three principal forms of ague, writers state that quartans are peculiarly obstinate. This variety, which is comparatively seldom seen, is especially apt to occur in the autumn; and it has long been well known that autumnal agues are worse than those contracted at other seasons of the year. On the other hand, it is said by Hertz that in the tropics the disease never assumes the quartan type; and yet ague is certainly in every other respect far more severe there than within the temperate zone. In hot countries, indeed, the quotidian appears to be the most common form of ague, whereas in milder climates the tertian is more frequent.

There are some other differences in the course of the several varieties of intermittent fever. Thus the fits of a quotidian are said generally to set in during the morning, those of a tertian at about noon, those of a quartan still later in the day. The average duration of a paroxysm of quotidian ague is stated at from ten to twelve hours, that of a tertian at from six to eight hours, that of a quartan at from four to six hours; but it is added that in the last-named variety the cold stage is more prolonged than in the others.

Subsequent Course.—Ague, in its intermittent forms, appears to be never directly fatal. Its duration is very variable. Sometimes, even though the patient remains in the place where he acquired it, it ceases after a few paroxysms. In other cases they come to an end when a change of season reduces the malaria to a state of inactivity. In others, again, they recur for an indefinitely long period, until he is compelled to remove to some other district. There is always a strong tendency to relapse, not only under a fresh exposure of the ague poison, but even independently of it; and it is a very remarkable fact that, whether the type be tertian or quartan, the paroxysms of a relapse often occur on those very days on which they would have been due if the disease had gone on without break from the first. Graves has related an instance of quartan ague which he watched for twenty-seven months, and in which the periodic rate was maintained through thirteen out

of sixteen intervals, some of which lasted more than two months. A similar observation had previously been made by Dr. James Gregory, in the case of a relative, who had marked on an almanac the days on which the fits of a tertian ague might be expected to recur, and who found that for a long while they did so whenever the east wind blew. Since the introduction of the thermometer into clinical practice, however, it has been shown that there is sometimes a distinct rise of temperature at the proper time for the paroxysm, although the patient himself may be altogether unconscious that anything is the matter with him. Ringer even states that, in spite of the absence of all febrile disturbance, the periodicity of the disease may be maintained by the voiding of urine undue in quantity, and containing an excess of urea.

Indeed, well-marked ague fits are not always characterized by the whole series of phenomena described above. Sometimes the cold stage is absent, sometimes the sweating stage. It is said that sometimes the usual order is reversed, as in a case cited by Sir Thomas Watson from M. Maugenet. The patient in question was always first attacked with profuse sweating; then he became dry and hot, finally he felt cold, and had distinct rigors. As an unusual symptom may be mentioned the presence of albumen in the urine, accompanied by casts and even by blood; this has been observed by Griesinger and Hertz. In certain exceptional cases the paroxysms differ *in toto* from those which are ordinarily seen, and they may be attended with great danger to the patient's life, so that Trousseau and Hertz both classify them apart under the name of *pernicious malarial fevers*. The most strange form of all is that which has been termed *syncopal*, in which there is a condition of suspended animation, so that the patient runs a great risk of being buried alive. Trousseau relates two instances of this. One occurred in a station master on the Avignon Railway. He had been subject for some time to paroxysms of intermittent fever, and had repeated fainting fits; once he became pulseless, and, being supposed to be dead, he was carried to the mortuary. After some hours a servant happened to enter the place and found him groaning; he was, therefore, taken back to his bed, and under large doses of cinchona he regained his health. It is an extraordinary circumstance that M. Chauffard, the physician who observed this case, also met with another one of the same kind. A man had fallen into a faint, was taken for dead, and had his face covered with a sheet; M. Chauffard, however, detected slight movements of his heart, although the radial, axillary, and carotid arteries had ceased to beat; he immediately administered quinine, per rectum, and the man was saved. Scarcely less remarkable is another variety which is attended with coma. Hertz says that instances of it are from time to time sent into the hospital under his care, for apoplexy, and Graves placed on record a case in which he made this mistake. A gentleman awoke at about 4 A. M. with sensations of malaise, chilliness, nausea, and headache. After an hour he became extremely hot, the pain in the head was intense, and he passed from a drowsy condition into one of complete coma, with deep snoring, so that he "appeared to be laboring under a violent apoplectic fit." He seeemed to derive much advantage from bleeding and other remedies, and in the evening he was perfectly well. The day but one after the same symptoms returned, and were removed by the same treatment, but when a third attack came on, Graves saw that it was an example of *tertiana soporosa*, and cut it short by large doses of quinine. In other cases it is said that epileptiform convulsions or tetanic spasms have been present, or that the symptoms have simulated those of hydrophobia. It even seems that the paroxysms of ague may imitate various abdominal or thoracic diseases—cholera, dysentery, peritonitis, pneumonia, pleurisy, are among those enumerated by writers. Hertz tells us of a man who was attacked at two o'clock in the morning with shivering and pain in the left

side ; at eight there was a distinct pleural friction sound, but twenty-four hours later he was perfectly well. However, on the following night, at twelve o'clock, all the symptoms returned, and by nine the friction sound was more evident than before ; at four in the afternoon he was sweating and free from pain ; full doses of quinine were, therefore, given to him, and he had no further attacks. But it seems to me that it is impossible to be too cautious in accepting the doctrine that such a case is the mere result of malarial poisoning ; it is an established fact that in those who live in districts in which ague prevails all kinds of diseases assume a more or less distinctly intermittent character, as though the operation of their several exciting causes were modified by the existence of a tendency to ague.

Nor is it less difficult to determine the real nature of what have been termed *masked agues*, in which the paroxysms are stated to be of the most diverse descriptions, and attended with scarcely any febrile disturbance, or even with none at all. The most important of these is neuralgia, occurring especially in the region supplied by the first division of the fifth nerve (and thus often called "brow-ague"), but sometimes assuming all sorts of other forms. Not only have cases of sciatica and of other neuralgic affections of the limbs been attributed to ague, but the same view has been taken of painful affections of the mammary gland or of the testicle, and even of alarming seizures attributed to cardialgia or to neuralgia of the vagi ; but, for my own part, I must confess that I do not know what evidence in favor of such interpretations of clinical facts could be taken as conclusive. Obviously it is not enough to show that the attacks recur with a certain degree of regularity, and pass off under the influence of quinine, for as much as this may be said of almost every kind of neuralgia. The observance of a tertian or quartan type would no doubt be very significant, but Hertz says that masked agues are usually quotidian. It must be borne in mind that the more extensive a man's experience in regard to intermittent fevers, the more likely he is to be led astray by tradition, or by personal bias, until he has recourse to the same convenient hypothesis for the solution of every obscure and indeterminate affection, such as occur frequently enough in clinical practice, even in districts where no ague is met with. What, for example, is to be said of the case of Dr. Macmichael, cited by Sir Thomas Watson, who "caught an ague, many years before his death, by sleeping on a rock, somewhere in Greece, and was ever after subject to occasional attacks of periodic headache and other aguish symptoms, for which he was obliged to have recourse to bark or arsenic"? And what value can be assigned to Trousseau's statement that there are cases in which insomnia, unaccompanied by fever and not preceded by rigors, recurs every two or three nights, that these cases are of the nature of masked ague, and that they are cured by similar treatment? This writer mentions, in addition, spasmodic cough recurring in paroxysms at the same hour, and certain periodical mucous or sanguinolent fluxes from the nasal fossæ, the uterus, or the intestines.

Remittent Fever.—The effects of malaria are not limited to the production of the different forms of intermittent fever. In hot countries, and during the hot seasons of the year, even in temperate climates, it causes fevers which are either continuous or at most interrupted only by *remissions*, intervals in which, instead of there being a complete apyrexia, there is only a partial lowering of the temperature and of the pulse, with some degrees of abatement of the other symptoms. For cases of this kind the technical name is *remittent fever*, but in India a common term for them is *jungle fever*, and another is *bilious remittent fever*. They are, in fact, characterized by a series of phenomena which do not belong to the milder effects of the marsh poison, as well as by the absence of some of the more distinctive features of ordinary

ague. Thus the cold stage is slight and ill defined, and the hot stage is followed by little or no sweating. The remissions themselves, which often begin about midnight or in the morning, and which last for twelve hours or longer, may be so slight as to require careful clinical observation for their detection. A feeling of oppression at the epigastrium is present from the commencement of the patient's illness, and vomiting is usually a marked symptom throughout its course. The vomited matters may at first consist of food, but afterwards a watery fluid is ejected, often in surprising quantity, and it may ultimately become greenish yellow, brown, or even in rare cases black. Headache is generally a prominent symptom, but not delirium. In the worst cases, however, in which the disease is said to be of an "adynamic" character, the patient rapidly becomes very prostrate and insensible, his skin is yellowish and covered with petechiæ, his tongue is black, his teeth are covered with sordes, and hemorrhages may occur from the nose, the mouth, and the limbs; or hematuria may be a prominent symptom. French writers have especially described such cases under the name of *fièvre bilieuse hématurique*. Dr. Maclean, from whose description of remittent fever in "Reynolds' System" I have taken most of the preceding details, says, that except in such cases the urine seldom contains albumen; it is often abundant, and there may be but little pigment in it.

The duration of remittent fever is said to be generally from five to fourteen days. Even its worst forms ought, according to Maclean, to do well in most cases, if seen early and skillfully treated. Sometimes it ends with a critical perspiration, sometimes it subsides gradually, sometimes it passes into one of the regular types of intermittent fever. Maclean says that death seldom occurs before the seventh or the eighth day, a point which may distinguish this disease from yellow fever, in countries where they both prevail.

Cachexia.—In persons who have suffered from protracted ague, or who have had repeated relapses, a chronic condition of ill health is apt to develop itself, which is known as the *malarial cachexia*. And a precisely similar condition is not infrequently seen in those who have lived for a long time where the disease is prevalent, even though they may never have had any febrile symptoms. One of its most characteristic signs is a peculiar earthy, sallow complexion; another is a damp, clammy state of the skin, especially noticeable in the hands. The patient is depressed in spirits, and wakes unrefreshed by the night's sleep; he may suffer from giddiness, noises in the ears, tingling in the hands and feet, pains in the lower part of the back, palpitation, loss of appetite, a furred tongue, and digestive disorders.

In all cases of this kind, enlargement of the *spleen* can be made out on percussion. Very often the organ is to be felt beneath the left costal cartilages, extending down to the umbilicus or even lower. Sometimes it forms a conspicuous tumor, which used to be called an "ague cake." Indeed, although in some very rapidly fatal forms of malarial fever the spleen has been found of unusual size, the rule appears to be that it swells with every paroxysm of the disease, subsiding more or less completely during the intervals. It is generally supposed that the yielding capsule of this viscus allows it to become the receptacle of an undue proportion of the blood which is driven inward from the surface of the body in the cold stage. Nor do I think that Hertz is right in rejecting this hypothesis altogether, on the mere ground that a malarial cachexia in which there have been no febrile attacks is also attended with splenic enlargement. As might be expected, the state of the organ is very different under different circumstances. In acute cases it is soft or diffluent. Hertz says that it may even rupture, pouring a quantity of blood into the peritoneal cavity, so as to bring the case directly to a fatal issue. This writer also states that infarcts are sometimes formed

in it, which may lead to suppuration, or to gangrene, followed by intense peritonitis. On the other hand, in cases of chronic ague or of malarial cachexia, the spleen becomes extremely indurated. Not only is the hardness of it discoverable by palpation during life, but after death it is found to have little or no pulp, while the fibrous elements of its substance have undergone an immense increase. Its capsule is often very much thickened, and it may be fixed by adhesions to adjacent parts. At the same time the *liver* is often found enlarged and indurated, and there appears to be some doubt whether it may not undergo a change identical with cirrhosis. Clinically these conditions are further indicated by the presence of pain and tenderness in the hypochondriac regions, and by the occurrence of slight ascites with which œdema of the lower limbs may also be associated. Indeed, I ought to have noticed that uneasy and painful sensations over the spleen are sometimes complained of, even during the hot stage of an ague paroxysm, being no doubt dependent upon the temporary enlargement of the organ which we have seen to occur. German writers commonly mention lardaceous changes in the abdominal viscera among the results of chronic poisoning by malaria. The only authority that I have found for such statements is the Swedish physician, Key, who, in 1862, described a form of renal disease, consisting mainly in a lardaceous affection, as a sequela of ague. Maclean cites Parkes as having often observed chronic Bright's disease in those who had suffered from ague, and confirms the statement from his own experience. But can one be sure that sufficient care was taken to eliminate other possible causes?

There is, however, another affection which results from severe or protracted ague, and which is altogether peculiar to this disease. It is called *melanæmia*, and consists in the presence of a brown or black pigment, partly free, partly enclosed in leucocytes, which is found not only circulating in the blood, but also lying in the substance of the solid tissues. The pigment in question is, no doubt, derived from the coloring matter of the blood. Klebs states that it yields Prussian blue when tested with ferrocyanide of potassium, so that it must contain iron which no longer forms part of an organic compound. It occurs either in the form of minute granules, or in that of larger masses, irregular in shape, and sometimes semi-crystalline in appearance. Where there is a very considerable quantity of it, the organs are very conspicuously discolored. Thus the liver and the spleen and the kidneys assume a slaty-gray color, the medulla of bones becomes chocolate brown, the cortex of the brain may appear as if rubbed over with black lead. A most striking example of pigmentation of the brain, no doubt malarial in origin, occurred at Guy's Hospital, in 1829, in the practice of Dr. Bright, who figured it in his "Medical Reports." The case was that of a man who, with his wife, died of severe fever immediately after they had walked up to London from Horncastle in Lincolnshire. I do not know of any other similar instance as having been observed in this country. In Germany, too, melanæmia with abundant pigmentation of the organs appears to be very rare; the well-known cases recorded by Frerichs all of them belonged to an epidemic of ague which arose in Silesia after an inundation in 1854. But more than one observer has stated that he has constantly been able to detect pigment in the blood, by microscopical examination, in cases of severe or *pernicious* ague, or in those of malarial cachexia.

In 1877, Dr. Stephen Mackenzie made a similar observation several times in the case of a man who had brought ague with him from India. The red discs were normal; but many leucocytes contained granules which were arranged round their nuclei, or sometimes completely filled them, so as to give them a uniform brown or black color. After treatment with

quinine the melanæmia was no longer discoverable. The blood of the portal vein has been said to be specially affected with this change; Dr. Hammond has even punctured the spleen during life, and drawn off a few drops of blood from it for examination. The pigment is generally supposed to be originally formed mainly in the spleen, and to pass from this organ into the blood, so as to be carried all over the body. It often blocks up the capillaries, but it is also found in the walls of larger blood vessels, and even outside them, in their sheaths. These facts do not prove that it may not have come from elsewhere, for wandering leucoctyes may have carried it with them. But many pathologists think that, in some cases at least, it is developed *in situ* from extravasated red corpuscles. Whether melanæmia directly gives rise to any symptoms appears to be doubtful. Dr. Mackenzie's patient had hemorrhages into each retina, and the same thing has been observed in some other cases of ague. It is natural to suggest that they might have resulted from plugging of minute vessels with pigment, but the anæmia which is so commonly present in such patients would equally well account for them. In 1880 a man who had caught ague in the Black Sea, was in Guy's Hospital, under my care; he had a large hemorrhage in one retina; but no pigment could be found in the blood. Capillary hemorrhages in the brain have, however, been attributed to pigmentary embolism of this organ; and a variety of cerebral symptoms have been supposed to result. But Hertz asserts that there is, in individual cases, no close correspondence between the occurrence of such symptoms and the demonstrable presence of the pigment in the minute vessels of the brain. Perhaps the most interesting question of all is whether there may not be something in common between melanæmia and that remarkable affection, paroxysmal hæmatinuria.

Pathology.—We have now to consider what is the nature of the *malaria* or exciting cause of ague, under what conditions it is developed, and how it gains access to the human body.

That the disease is especially apt to prevail in marshy districts is universally admitted, and the fact could not possibly be disputed. Yet it is no less certain that something more is needed than the mere saturation of the soil with water. For countries like Ireland, which are widely covered with peat bogs, do not breed malaria abundantly. Nor, according to Parkes, are salt marshes infested with ague, when they are regularly overflowed by the tide; and this, notwithstanding that for a large part of each day their surface is exposed, which (as we shall presently see) might have been thought likely to favor the development of malaria in them. Again, the most virulent forms of the poison have sometimes been met in districts that appeared to be perfectly dry and arid. Such, according to Hirsch, are the table land of Castile, the plain of the Araxes, and certain lofty plateaus in India and Persia, all of which are highly aguish; he even says that careful inquiry has proved that the malarial regions of Italy are in large parts of their extent devoid of water and sterile. Again, instances in which British troops have been attacked with the disease while encamped upon dry, sandy soils, both in Holland and in Spain, are cited by Sir Thomas Watson from observations made by Dr. William Ferguson nearly a century ago. And Maclean lays stress upon the development of malaria in Hong Kong, and in other places, situated upon granite rocks which are undergoing disintegration. These exceptional cases are fatal to an hypothesis which at one time was generally upheld, namely, that the poison of ague is nothing more than a product of putrefying vegetable matters. But there is no real difficulty in bringing them into harmony with those in which the disease is developed in marshes, along the estuaries of rivers, and in smaller regions. For accurate investigations have shown that even there the malaria is developed,

not in the wet part of the year, when the ground is entirely flooded with water, but rather during those seasons at which large parts of it are exposed to the air, and become more or less dry. And as Maclean has pointed out, it is tolerably certain that, in every instance supposed to be of an opposite kind, water might very easily have been reached, either by the form of subterranean streams, or upheld by a bed of clay or other impermeable material, or only saturating the soil up to a certain level. So, again, it has been noticed that in Italy an occasional overflowing of fresh water marshes by the sea has been followed by a great development of malaria; but this is not, as has been supposed, inconsistent with the fact that marshes which are always saturated with salt water are healthy, since it is obvious that the conditions in the two cases are altogether different. One way in which a hot sun probably favors the development of ague is by cracking the dry surface of the ground and allowing emanations to escape from the moist layers beneath. And so, again, it has repeatedly been found that the disease has reappeared in places that had long been free from it when the soil has been extensively disturbed for building purposes, or during the construction of canals or of fortifications. Conversely, one can do a great deal toward preventing the exhalation of malaria by draining the ground thoroughly, and by then spreading a layer of fresh soil over its surface, or even, in the case of inhabitated villages or towns, by paving it.

Of course, many districts are decidedly damp without being aguish. But this merely proves that other conditions besides a wet state of the soil are necessary for the production of malaria. For at certain times the disease spreads to regions which lie far beyond its usual limits, assuming, indeed, somewhat of a *pandemic* character. The years 1558, 1678–79, 1718–22, 1807–12, 1824–27, 1845–48 are mentioned by Hertz as having been characterized by such a wide diffusion of it. Sir Thomas Watson says that he never knew ague at all widely prevalent in London except in 1827. From 1866 to 1868 there was an epidemic in Mauritius, which island had previously been so free from malaria that the sufferers from Indian fevers had been accustomed to resort there. And in 1869 it broke out for the first time in Réunion.

Distribution.—With regard to the nature of the various conditions which may thus favor the development of the ague poison, we at present know very little. It is a curious circumstance that there has often been an antecedent or simultaneous epidemic of influenza, or of some contagious disease such as typhus, cholera, or the plague. One very important feature is undoubtedly a high temperature. Ague prevails with far greater intensity in the tropics than in temperate climates, while toward the poles it is unknown, its boundaries in either direction being about 63° N. and 57° S. Hirsch has endeavored, upon very defective data, to determine the corresponding limits of temperature, and finds that mean summer temperatures rather than mean annual temperatures, must be taken into account. In this hemisphere the highest range of malaria toward the North is fixed by him at a line between the isotheral lines of 59° and 61° Fahr. Again, in particular districts, the occurrence of malarial fevers is governed in a very marked manner by variations of temperature. During the winter they disappear entirely; and, other things being equal, they are always more severe when they break out again, in proportion as the season happens to be a hot one. In the tropics the exact period of the year at which ague is most prevalent varies in different localities; generally speaking, it closely follows the rainy season. Hertz lays down the rule that, in order to escape malaria, one should arrive in the East Indies between November and January, in the West Indies between January and March. In temperate climates malaria is usually more active in the spring and in the autumn than at the height of the

summer. Wenzel made the important observation that among the men employed upon the works at Wilhelmshafen the development of ague followed the summer heat by an interval of twenty to twenty-five days, of which from six to eleven days appeared to be occupied in the generation of the poison in the soil, while the remaining fourteen days correspond with the period of its incubation within the bodies of those about to be attacked by the disease. The liability to ague is naturally greater near the sea level than in cooler places up higher; but Parkes mentions some instances in which marshes at elevations of five thousand to six thousand feet have, in hot countries, been found to give off malaria. It is, of course, quite a different question to determine what is the lowest point on the hills above a marsh at which one may reasonably expect immunity from the disease. The fact is well ascertained that the wind sometimes carries the ague poison to considerable distances from its source, not only on level ground, but also up the slope of a range of mountains. Parkes says that in temperate climates an elevation of at least five hundred feet above a malarial spot should be reached in order to escape its influence; in the tropics, one of one thousand to fifteen hundred feet, or even higher still. On the other hand, at the level of a marsh itself, the injurious emanations seem to have but little tendency to rise and diffuse themselves into the air. There is evidence that, both in barracks and in private houses, persons sleeping on the ground floor are more apt to be attacked than those who occupy upper stories. It is at night, and for a short time after sunrise, that malaria is especially to be feared; probably the poison is harbored by the stagnant mists which are so apt to lie over the surface of damp ground, until the sun disperses them. The presence of actively growing vegetation appears to be adverse to the development of malaria. There is also reason to believe that the inhabitants of houses in the neighborhood of a marsh are sometimes protected by the interposition of a belt of trees between it and them. The poison also seems to be incapable of being carried across a surface of water without undergoing absorption by it. When English troops occupied Walcheren and other parts of Holland it was several times noticed that only those soldiers who disembarked were attacked by ague; those who remained on board ship, even in narrow channels, escaped.

I have already incidentally mentioned many of the countries in which ague prevails. In England, its chief seats are along the eastern coast; Romney Marsh, in Kent, the estuary of the Thames, in Kent and Essex, the fens of Cambridgeshire and Lincolnshire, the marshy lands of the East Riding of Yorkshire. And in all these districts the disease has become far less frequent than formerly, since population has increased and improved drainage has brought more and more land under cultivation. Not many years ago cases often arose in London. Sir Thomas Watson cites without question the statement that both James I and Oliver Cromwell died of ague contracted in this city. At present its occurrence is altogether exceptional, even in the low-lying parts of Southwark. In Scotland I believe that there is now but little malaria; Sir Robert Christison, I think, has stated that it had begun to disappear before any drainage works were carried out. On the continent of Europe the regions in which the ague poison is most prevalent are, the great plain of North Germany, with some of the Baltic provinces of Russia; Holland, with the adjacent parts of Germany and Belgium; the southwestern departments of France; the western side of Spain and Portugal; almost the whole western side of Italy; a great part of Greece and Turkey; the plains of Hungary; and the shores of the Black and of the Caspian Seas. Nowhere is it so severe as in Italy, especially in the Maremma of Tuscany, the Campagna of Rome, and the Pontine Marshes. And what is of especial interest is that in these districts it has greatly in-

creased in intensity within the last four centuries. Places which were at one time thickly populated and well cultivated have become waste and deserted; and coincidently with these changes they have grown in the highest degree unhealthy. In Asia, malarial fevers abound not only in many parts of India, but also in Persia, and in China, and elsewhere. In Africa, the west coast is too well known for the malignant character of the agues which prevail there; and they are also of frequent occurrence in Algeria and in Egypt, as well as in Mozambique and Zanguebar. On the American continent, the States which surround the Gulf of Mexico are those in which the effects of malaria are most frequent and severe; they are also seen in parts of Brazil and of Peru, as well as in many of the West Indian islands. It is a remarkable fact that in Australia ague is everywhere of a very mild type, if it prevails at all; it is said to be altogether absent from New Zealand and from Tasmania.

Infection by Water.—Hitherto I have assumed that malaria enters the body with the air which is breathed; and, no doubt, this is usually the case. But there is good evidence that the poison is sometimes introduced in drinking water. The most striking case of all seems to be that recorded by M. Boudin, of the one hundred and twenty soldiers conveyed by the French transport ship "Argo" from Algiers to Marseilles, in 1834. In the hurry of embarkation, the water which was to be supplied to these men was taken from a marshy place near Bona. Thereupon, all but nine of them became attacked with various forms of ague; and it turned out that these nine, instead of drinking the same water, had purchased wholesome water from the crew of the vessel, all of whom remained well. In two more ships, which made the voyage at the same time, there were other six hundred and eighty soldiers, and they, too, escaped. Another instance is that of a farmer's family at Houghton, near Bedford, who drank well water, and who were at one time almost the only persons free from ague in the parish, the other inhabitants of which had only ditch water. In another village close by, it was noticed that the digging of wells, by which a supply of fresh water was yielded, was followed by a great diminution of the disease. And it seems not unlikely that to the same cause may be attributed whatever part of the decline of ague during the last few years, both in England and in Scotland, cannot be accounted for by the improved condition of the soil. I rather doubt, however, whether any statements with regard to the prevalence of malaria in former times can be universally accepted, since it is certain that until recently its effects were frequently confounded with enteric fever.

Predisposition.—Of the conditions that may render one person more susceptible to malaria than another we at present know but little. That more cases of ague are seen in men than in women or in children is doubtless due to the circumstance that men are more exposed to its exciting cause. A similar explanation may be found for the fact, if it be a fact, that during pregnancy women possess a certain degree of immunity. Sir Thomas Watson, however, cites an instance in which a child *in utero* had tertian ague with its mother, who herself shook one day, and felt the child shake within her on the following day. And Hertz speaks of infants born with enlarged spleens, under similar circumstances.

It seems to be well ascertained that negroes are proof against ague; so that black soldiers are invaluable for field service in certain parts of the West Indies. Even if this fact be due to a kind of acclimatization, transmitted by inheritance, it is not the less remarkable. For, although ague has sometimes been noticed chiefly to attack persons who had recently arrived in a malarious district, yet those who had lived there for a long time have commonly suffered in other ways, being, in Sir Thomas Watson's words, "puny,

sallow, and sickly." The negro, on the contrary, is said to enjoy life and health in such regions. It seems to be certain that over-exertion and fatigue render a man more susceptible to the action of malaria; and that those who are convalescent from other diseases, or recovering from the effects of wounds, are particularly apt to suffer. It would be worth while to investigate whether persons whose general state of circulation is such that they are prone to the disease known as paroxysmal hæmatinuria are not more sensitive than others to the ague poison. Hertz says that this is the case with those who are weak and anæmic.

Contagium vivum.—It appears to me impossible to ponder over the various conditions which favor or modify the development of malaria, without coming to the conclusion that no hypothesis accords so well with the facts as that which assumes that it is a living organism. In 1866 an American physician, Dr. Salisbury, declared it to be an alga, of the genus *Palmella*, which he had found in marshy soil in Ohio. But he did not attempt to show that the spores of this microscopic plant were really capable of generating the disease; and the very same alga has since been discovered in regions where no ague exists, and even in the Alps. Nor are any direct proofs brought forward by other observers in Italy and elsewhere, who afterwards took up the subject, and by each of whom some different species was assumed to be the essential agent in propagating the disease. It was, therefore, a step in advance when, in the spring of 1879, Prof. Klebs, of Prague, and Prof. Tommasi-Crudeli, of Rome, carried out together a series of experimental investigations which they believe have determined not only that what constitutes malaria is a particular kind of bacillus, but also that a fever of intermittent type can be generated in rabbits by subcutaneous injection of liquids containing this organism. They constructed a machine by means of which a large quantity of air could be rapidly made to pass over the surfaces of glass slides moistened with glycerine jelly. This they set to work in the Pontine Marshes and in Rome, taking the air always from very near the surface of the ground. Direct microscopical examination of some of the glass slides afterward revealed the presence of rod-shaped bodies and delicate threads; and by suitable methods of cultivation these were made to undergo further development into jointed filaments, with spores in their interior. Similar structures were found in mud taken from the borders of lakes in the same region, but not in the waters of the lakes themselves. I must confess, however, that after reading carefully the details of the experiments in question, and studying the temperature charts which Klebs and his Italian coadjutor give, I fail to see that there is anything definite or characteristic about the fever which occurred in their animals after injection of fluids containing the so-called "*Bacillus malariæ.*" Further points on which these observers lay stress are that the spleen, without being softened, was constantly found enlarged after the rabbits were killed, and that this organ often contained black pigment, analogous with that which is seen after severe ague in man. They also detected the bacillus both in the spleen and in the medullary tissue of the bones of the animals. Finally, they cite Dr. Marchiafava as having succeeded in discovering the spores and jointed filaments of the same parasite in the same structures, and in the blood of two persons who had succumbed to pernicious malarial fever in Rome.

It is, I think, a significant fact that the other disease in which the presence of a peculiar organism in the blood has been most clearly traced—relapsing fever—should itself be characterized by a definite interruption in its pyrexia, which may fairly be taken as bearing some relation to the intervals between the successive paroxysms of an ague. And the analogy tempts one to hazard the hypothesis that during each paroxysm the bacilli, if they really constitute the malarial poison, are in great part destroyed, and

that their reaccumulation is in some way the cause of the next paroxysm. But, if so, it is difficult to understand how the periodicity can be maintained when no well-marked ague fits occur. With regard to the succession of phenomena that constitute the typical paroxysm, I do not think any special explanation is needed. The same order of events, more or less modified in details, is seen in the rigors of pyæmia and under a variety of other conditions. And there can be no doubt that, like an epileptic seizure, it rests upon a physiological basis. The recurrence of ague fits at the same hour on successive days, or every third or fourth day, is doubtless, as Cullen originally suggested, connected with the diurnal habit of body which is so plainly manifested in other ways, both during health and in disease. This is not merely acquired, but has probably been transmitted by long inheritance to every existing member of the human race. One, therefore, need not wonder if, as is stated by Hertz, Griesinger and Duchek failed to modify the times at which ague fits returned by changing the patient's hours of meals and his other conditions of life.

The *diagnosis* of ague is for the most part very easy, but it may present difficulties which have to be considered from two points of view, according as the disease is or is not known to be prevalent in a particular district. In places which are free from malaria there is sometimes no little risk of the real nature of a patient's illness being overlooked, when it has been acquired elsewhere during a visit, or while he was traveling in a country in which he, perhaps, was not aware of the existence of the poison. Again, it is an extremely difficult point to settle whether or not some of the milder and less characteristic effects of malaria may still continue to appear from time to time in places (such, for instance, as the Borough of Southwark) in which the regular forms of ague have long been extinguished. Many very experienced physicians have undoubtedly held this belief; but I cannot determine how far their judgment was biased by the fact that the affections in question are curable by quinine and the other remedies for ague, a fact which certainly is in itself of no decisive significance. On the other hand, in regions notoriously infested with malaria, the physician has constantly to keep in mind not only that ague is apt to complicate all other diseases, but that it is capable of actually simulating an immense variety of them, as I have already pointed out at p. 259. But, further, there is always great danger that he should carelessly mistake for results of malaria different affections which are attended with recurrent rigors or with paroxysms of febrile disturbance. Thus Graves relates an instance in which the intermittent hectic of phthisis was set down as ague, and some years ago I saw the very same error committed in the wards of Guy's. Another disease which is very apt to be overlooked under such circumstances is pyæmia. In reference to this, Hertz remarks that the pyæmic rigor generally sets in more gradually than that of ague; that its subsidence is more rapid than its onset, whereas in ague the reverse is the case; and, lastly, that in pyæmia there is very seldom an interval of complete apyrexia, continuing for twenty-four or even for twelve hours. But, as we have seen, ague itself may be merely remittent. The fever attending internal suppuration may also be attributed to malaria. Not long ago I saw a case in which a medical man was firmly convinced of the correctness of such a diagnosis, until his patient voided a quantity of horribly fetid pus with his urine, after which a rapid recovery took place. Some years since, a gentleman who had stricture was placed under my care by Mr. Cock, on account of the recurrence, at tolerably definite intervals, of rigors that seemed to be due to the formation of an abscess that could not have been of any great size, in the neighborhood of the urethra. Ulcerative endocarditis, too, must be thought of.

Finally, there are the continuous forms of ague, with which other diseases, and especially enteric fever, have often been confounded. Thus I happen to know that in Romney Marsh no kind of fever used to be recognized except that due to the endemic malaria; and in many parts of India it is only quite recently that enteric fever has been proved to occur. The *fièvre bilieuse hématurique* is not always easily distinguished from yellow fever in countries in which both diseases are met with.

This is, perhaps, the most convenient place for brief mention of a complaint to which brass founders are liable, and which was studied by Dr. Greenhow in Birmingham, in 1858, and named by him "brass founders' ague," on account of the resemblance between its symptoms and those of a paroxysm of intermittent fever (*Med.-Chir. Trans.*, vol. xlv). It is in no way dependent upon malaria, being simply caused by the dense white fumes of oxide of zinc, the result of deflagration of the metal, which, at a certain stage in the formation of brass, fill the casting shop, unless it is very well ventilated. Exposure to these fumes is followed by malaise and weariness, by a sense of constriction at the chest, and sometimes by nausea. Afterward shivering comes on, and even chattering of the teeth; and this is succeeded by a more or less marked hot stage, and ends in very profuse sweating. Next day the man feels out of sorts, but not enough to be unable to work. There is no regularity in the recurrence of the attacks, which come on from time to time under the operation of their exciting cause, but especially when the patient has been off work for a few days. They seem not to impair the health, and it is not known that they shorten the life of the patient. Drinking milk is said to be preventive of the brass founders' disease.

Treatment.—With regard to the course taken by the various forms of ague if left to themselves, I have nothing to add to what has already been stated. But the *prognosis* in this disease is not based directly upon its natural course, for it always is, and always should be, submitted to treatment as soon as it is recognized. Indeed, we have so potent an antidote to malaria in cinchona bark, and in the quinine which is derived from it, that writers have sometimes been disposed to class this remedy apart from all others, as the most signal instance of a specific. And I imagine that it really does occupy a unique position in one respect, namely, as being the only medicine of which the efficacy has never, in our day, been challenged by any one, however rash and inexperienced. The bark itself is not now prescribed, on account of its causing nausea and sickness in the large doses required; quinine or one of the other alkaloids is substituted for it. Even as a preventive of ague, quinine has been found very efficacious in the cases of sailors exposed to malaria when sent ashore for a day, of travelers passing through, or of soldiers bivouacked in, a marshy district. From two to five grains are given twice or three times a day. It is less desirable to place the permanent residents in an aguish region constantly under the influence of quinine, since the organism appears to become somewhat blunted to its action in course of time; but they may, at least, take it at those seasons during which the disease is most prevalent.

With regard to the best method of administering the remedy in the intermittent forms of ague there has been much difference of opinion. There is reason to believe that the paroxysm itself cannot be modified by quinine taken after its commencement, except, perhaps, when it is injected subcutaneously. Probably, therefore, if one is called to a patient in an ague fit the best plan is to prescribe a full dose of some ten or fifteen grains, as soon as possible after its cessation, repeating the same dose more or less frequently during the interval, until an hour or so before the next fit is expected. Maclean suggests, as a rule of practice, that the patient should never

have less than thirty grains during this period. It is, he says, best given in solution with a little sulphuric acid and tincture of orange peel. As is well known, pills containing quinine, besides being clumsy, are exceedingly apt to become hardened by time, so as to pass through the stomach undissolved. If vomiting should occur the alkaloid may be administered, per rectum, in a dose of fifteen grains suspended in beef tea or thin starch.

When the stomach is loaded, an emetic is often a useful adjuvant, and if there is constipation a purgative; but it was altogether a mistaken practice which formerly prevailed, of putting off the use of quinine until the tongue had been made clean by other treatment, and the digestive organs regulated.

For the actual paroxysm of ague but little treatment is needed. The patient should go to bed and have plenty of blankets, and hot bottles or hot bricks wrapped in a flannel may be applied to his limbs. Baths are better avoided, and he should not be encouraged to drink warm fluids in any large quantity. Frictions with stimulating liniments, or with a mixture of soap liniment and laudanum, have been recommended. It is only when threatening symptoms of collapse are present that recourse should be had to coffee, wine, ammonia, and ether. As the hot stage comes on most of the bed clothes may be removed, the patient may have cooling drinks if he wishes it, or he may suck a little ice; he may be sponged with vinegar and water. At the end of the sweating stage he should be carefully dried and have a change of linen. He may then get up, but if he should afterward go out of doors he must be very careful not to take cold.

It very rarely happens in recent cases of ague, where quinine is given in the manner indicated above, that the patient has any subsequent attack of the same degree of severity as before; and within a few days, or in a week or two at latest, they almost always cease. But in certain cases, especially such as are of long standing and of quartan type, it seems altogether to fail. The remedy which then proves most successful is arsenic, or the tincture of eucalyptus may sometimes be prescribed with advantage. The application of cold douches to the left hypochondrium is recommended by Hertz as a valuable accessory to other treatment.

For the dangerous remittent and continuous forms of ague, and for those cases in which the symptoms are those of apoplexy or of some other cerebral disease, quinine is still the remedy, but it must be employed very much more boldly. Maclean, indeed, insists on the importance of watching for even a slight remission, using it as an opportunity of giving fifteen or twenty grains of quinine by mouth, and repeating the dose in two hours' time. If the stomach rejects it a similar quantity may forthwith be administered, per rectum, but in the most severe cases of all he does not wait a moment, and not only prescribes quinine energetically, but also gives stimulants and plenty of nourishment. This writer also speaks in high terms of Warburg's tincture, a remedy at one time secret, but now known to contain, in addition to quinine, aloes, rhubarb, camphor and opium. This appears to be a powerful sudorific.

The malarial cachexia requires the administration of iron as well as quinine, but even these remedies are of secondary importance in comparison with the removal of the patient to a healthy locality. Maclean speaks strongly of the value of the ointment of bichloride of mercury in the treatment of chronic enlargements of the spleen and of the liver. He directs that a piece of the size of a nutmeg should be rubbed into the skin of the hypochondriac regions, and that these parts should be then exposed to the heat of a fire until the smarting becomes too severe to be borne.

DENGUE

HISTORY—NAMES—COURSE AND SYMPTOMS—PROGNOSIS—TREATMENT—NATURAL HISTORY AND MODE OF SPREADING.

In 1824 an epidemic of a kind hitherto unknown to Indian surgeons broke out at Rangoon, in Burmah, and quickly spread to Calcutta, and to various places in Bengal or in Madras. In 1827 the same malady appeared in the West Indies, in the Isle of St. Thomas, and a few weeks later in the adjacent Isle of Santa Cruz. Here also it was at first regarded as a new disease; but it has since been found that Dr. Rush, of Philadelphia, described it as prevailing in that city in 1780. During the last fifty years it has from time to time attracted attention both in the Eastern and the Western Hemispheres. The latest outbreak in India seems to have been in 1871–72, when it is said to have been derived from Zanzibar, passing to Bombay by way of Aden; in 1873 it spread to China and to Cochin China, and also to the islands of Mauritius and Réunion. In the West Indies it was last seen at Martinique, in 1874 and in 1875. The only spot in Europe in which this malady has hitherto appeared is Cadiz, where it was observed by Poggio in 1867; an earlier epidemic is said to have occurred in the same town as far back as 1784.

In St. Thomas the English negroes called it "dandy fever," apparently in ridicule of the attitude and step assumed by those who were seized with it. Other absurd names for it have been break-bone fever, broken-wing fever, and giraffe, the latter because the neck is held in a stiff position. Several later terms have been applied to it, one of which is "scarlatina rheumatica," but it is now universally known as Dengue, not only in England, but also in France and in Germany; this is in reality a Spanish word, which is very analogous to dandy in its meaning, except that it is applied to women and not to men.

Course.—Dengue sometimes sets in with lassitude, drowsiness, vertigo, a sensation of chilliness down the back, and other symptoms of commencing pyrexia, but very often it begins quite suddenly, with pain in some particular part of the body, perhaps while the patient is walking about, or during the night, waking him from sleep. If the pains affect the knees while he is standing, he may fall down. Dr. Stedman, who in the "*Ed. Med. Journ.*" for 1828, gave an excellent account of the disease as it occurred in St. Thomas, says that the first thing noticed was often a stiffness in one finger, especially the little finger; this would increase and be accompanied by intense pain, which spread over the whole hand and up to the shoulder, and in a few hours the fingers of both hands would be swollen, stiff, and painful, and their joints would be incapable of being bent. I do not find that any writer states that effusion can be detected in the articular cavities, as is the case in rheumatic fever. Hirsch says that in those cases in which autopsies were made serous infiltration was found to have occurred into the connective tissue round certain joints, and that in one instance the crucical ligaments of the knee were reddened. Cotholendy, in describing dengue in the Isle of Réunion, suggests

that there is an exudation into the sheaths of the tendons, and especially of the extensor tendons, and that this renders movements of the joints so exquisitely painful. He also speaks of "a slight fullness, a sort of œdema," of the hands and feet. There is often a very violent pain in the eyeballs, which feel too large for their sockets, and as though they would start from the head. Before long every part of the body becomes the seat of the most extreme suffering, which is aggravated by restlessness, compelling the patient to be constantly changing his position, while he also feels extremely prostrate and distressed. Sleeplessness is a marked symptom, and children are sometimes delirious.

In the meantime the temperature rises until it reaches 102°, 103°, 104°, or even (though very rarely) a higher point still. Observers who have employed the thermometer during some of the more recent epidemics, have attached some importance to the occurrence of remissions, which follow one another three or four times in the twenty-four hours; but I should doubt whether this is so remarkable a feature of the disease as they seem to think. Most writers have described the pulse as very rapid, 120 or even 140 in the minute, but Martialis, in the "*Arch. de Méd. Nav.*" for 1874, says that he often found it not much over 80, lower, in fact, than might have been expected from the temperature. Twining long ago noticed that the countenance was flushed and of a scarlet hue, and of late it has become the fashion to speak of an exceedingly fugacious *initial rash*, consisting of bright red patches upon the face, the chest, the palms of the hands, and elsewhere, which, however, subside after the lapse of a few hours. There is anorexia; the tongue is thickly furred, with bright red edges. The stomach is extremely irritable, and may reject everything that is swallowed. The bowels are constipated.

The condition of the patient at this period might well cause considerable anxiety to a medical man unacquainted with the characters of the disease; but at the end of twenty-four or thirty-six hours the pyrexia begins definitely to subside, without any marked critical sweat, and the temperature soon falls to normal or even slightly below it. At the same time the pains in the limbs cease, and soon there is nothing for the patient to complain of except a sense of general weakness, loss of appetite, and pain in the back. At this time it is said that the glands in the neck, the axillæ, and the groins may be felt to be slightly enlarged.

The duration of the interval is about three days. At the end of it an eruption—a *second rash*, according to recent observers of the disease—appears upon the skin. It is first seen upon the palms of the hands, and it also affects the feet and the knees, and may even (though but rarely) spread all over the body. In character it is usually somewhat intermediate between the rash of scarlet fever and that of measles. It has also been compared with the erythema that sometimes accompanies rheumatic fever. Or it may resemble an urticaria, or even in some cases be attended with the formation of bullæ or of pustules. It gives rise to a distressing sensation of tingling, which presently passes into itching. Its development is sometimes associated with a return of pyrexia, but recent observations seem to have shown that, as a rule, the temperature remains normal at this period of the disease. Consequently the fact that an eruption has made its appearance is not seldom altogether overlooked; whether it is ever really absent is said to be doubtful; after a few hours, or two or three days, it subsides and disappears, and with it the fever, if any has been present. Afterwards, however, the cuticle begins to desquamate, usually as a branny powder, but sometimes in very large flakes, and this may be attended with considerable discomfort and soreness, especially of the feet.

The second eruption is often associated with a repetition of the articular pains, which are, however, less severe than at the beginning of the disease.

Or there may be a respite of three or four weeks, and at the end of that time the pains may return, compelling the patient to take to his bed again and to be fed like a child. Dr. Stedman speaks of these pains as always most severe in the morning and as wearing off in some measure toward evening. They are felt chiefly in the joints of the fingers and of the toes, in the wrists, in the ankles, and in the knees. The affected articulations may be stiff and swollen to such a degree as to produce deformity. After a few days the secondary pains in their turn begin to subside, and one joint after another becomes free from them; but months may elapse before they have entirely disappeared. It is even said that there may be a permanent partial anchylosis of some particular joint.

Prognosis.—Dengue is scarcely ever fatal, but infants sometimes die with convulsions during the primary pyrexia, and old people may succumb to exhaustion toward the end of the disease, or to bronchitis occurring as a complication. Nielly, in his "Éléments de Pathologie Exotique," mentions that pericarditis has been observed in severe cases.

Treatment.—It is recommended to give a purge or an emetic at the commencement of this disease, and afterward salines and Dover's powder. Nielly suggests the use of salicylate of soda or of colchicum, but I have not met with any recorded evidence of the value of these medicines. Liniments of chloroform, belladonna, or cajeput oil are often useful. Dr. Stedman found that the application of blisters or of sinapisms to the neck or to the loins gave great relief to the pains in these parts. When one or more joints remain stiff and painful after the subsidence of the disease, sulphur baths are said to be very efficacious.

Concluding Remarks.—The true place of dengue among infective diseases has yet to be accurately determined. In the rapidity with which it spreads over a population, it resembles influenza more than any other malady. Another remarkable point is that scarcely any one escapes, even among the inhabitants of a large city, as, for example, among the half million of residents in Calcutta in 1824. It attacks persons of all ages, including even infants a few days old, and the colored races are as liable to it as whites. When it has existed for a little time in a place and patients are recovering, one might almost think that there were none but cripples in the place, so many are seen limping about the streets on crutches, or with bodies half bent, or with arms in slings. The whole duration of an epidemic is generally short, being from two to seven months, according to Hirsch. The disease is said to prevail chiefly with sultry, cloudy weather, or at the time of heavy rains. Indeed, in temperate climates it occurs only during summer and autumn, and disappears when frosts set in; but in the West Indies viccissitudes of weather seem to have no way interfered with its course. Its diffusion from one country seems plainly to be effected by human intercourse. Whether it is contagious in a narrow sense, passing from the sick directly to the healthy, seems hardly to be known with certainty. Dr. Stedman, however, says that in 1827, it was introduced from St. Thomas into Frederickstadt, a town of Santa Cruz, by some young ladies who went to reside at the house of a Major S—; all the members of his family were attacked, and a few days later it appeared in the next house and affected every one there. Cotholendy relates that an infant, which had taken the disease while with the family of its nurse, was brought home to its mother—four days afterward she fell ill; the grandmother and the aunt did not see the child until the day after its arrival, and they were attacked a day later than the mother, each at the same hour. This writer also gives several other instances which indicate that the incubation of dengue lasts four days.

YELLOW FEVER.

INCUBATION AND COURSE—MORBID ANATOMY—DIAGNOSIS—QUESTION OF ITS CONTAGIOUS OR MIASMATIC NATURE.

I have now briefly to describe a disease which is almost limited to America, the West Indian Islands, and the West Coast of Africa. It has sometimes been imported into Europe, but never maintains itself among us. In the East it is altogether unknown.

Course.—The incubation of Yellow Fever is said to be usually from one day to fourteen or fifteen, but there are doubts whether it may not be considerably longer in certain cases. There are sometimes slight prodromal symptoms of malaise, headache, etc.; but very often it sets in quite suddenly with rigors, extreme depression, and all the other symptoms of a severe pyrexial illness. Sometimes it proves fatal in a few hours, with collapse and lividity. More often the temperature rises for two or three days, until it reaches 105°. In such cases the face becomes flushed, the conjunctivæ are red and ferrety, and the eyes bright and staring. Frequently there is pain on one side of the forehead, or an agonizing pain in the sacrum; or there may be pains in all the joints. The stomach becomes very irritable, and rejects its contents; the epigastrium is tender to pressure. The palate is reddened and œdematous; the gums may be swollen and inclined to bleed; the tongue is of a bright crimson color, and may be thickly furred; at a later stage it becomes clear, smooth, and raw looking. Constipation is more frequent than diarrhœa. The urine is very scanty, and it generally contains albumen.

About the third day, or not until the fourth or the fifth, the skin becomes jaundiced; the urine is bile-stained; but the fæces are seldom clay-colored. Hemorrhages now occur from various surfaces; epistaxis is frequent; sometimes there is a cutaneous purpura; the vomited matters, from being "white" and consisting of an acid, watery liquid, become "black," containing dark brown flocculi or masses of altered blood corpuscles. It is to be observed, however, that this symptom of "black vomit" occurs only in the more severe forms; and chiefly in such as end fatally. Thus, during an epidemic at Gibraltar, in 1828, Louis found that of the patients who died, all but one had it, and of those who recovered very few. According to Alvarenga, however, at Lisbon, in 1857, there were no fewer than forty recoveries among cases in which it was present.

Haenisch, the writer on this subject in Ziemssen's "Cyclopædia," who has himself seen the disease in the West Indies, states that there is usually on the fourth day a remission of the pyrexia, so that the temperature in twelve hours may fall nearly to normal. At the same time the patient loses his headache and experiences so much relief that he fancies himself out of all danger. The stomach, however, still remains irritable; and the urine, if not previously albuminous, now becomes so. In some cases the improvement is permanent, and goes on to complete convalescence. But more often, after a few hours, or within two days at the longest, matters change again for the worse. The temperature may now rise until it reaches

104°; but sometimes it remains normal, as, for instance, occurred in the case of a gentleman who died of yellow fever in London, in 1878, under the care of Mr. Leggatt ("*Clin. Trans.*," vol. xi).

The symptoms at this period seem mainly to depend upon disturbance of the renal secretion. The urine is often entirely suppressed for several days together; it always contains albumen and often casts. The patient now falls into an apathetic condition, or he may become violently delirious. The pulse is small and thread-like, sometimes rapid, sometimes slow. The jaundice increases until the skin has a dark mahogany color. Hemorrhages continue, blood being passed from the whole length of the intestine, from the female genitalia, from the external ear, and from the respiratory surface. Death is usually preceded by coma, but sometimes it occurs suddenly, the patient falling backward upon his bed after a state of violent excitement. But recovery may take place, even when the case has appeared very threatening; the temperature then falls quickly to normal, with profuse sweating. The mortality from the disease seems to vary widely in different epidemics, being sometimes as low as 15 per cent., sometimes as high as 75 per cent. The symptom of most value as regards prognosis at an advanced stage is said to be albuminuria; if the amount of albumen in the urine diminishes as the case goes on, the patient is likely to do well; if it increases, a fatal termination is to be apprehended. Among the phenomena which are observed during this period are suppurative parotitis, sloughing of the scrotum and penis, and the formation of boils containing blood mixed with pus. Convalescence is always slow, and several weeks elapse before the patient regains his strength.

It must be added that during an epidemic of yellow fever very mild cases occur, in which the symptoms are slightly pronounced. The pyrexia is moderate in degree; there is but slight jaundice, or it may be altogether absent, or may appear only when the attack is passing off.

Morbid Anatomy.—Rigor mortis is early and well marked; and, according to Nielly, putrefacation is rather retarded, which is certainly contrary to what one would have anticipated. The body is deeply jaundiced. Mr. Macdonald (in "Reynolds' System") says that the discoloration is sometimes more marked than it was during life. The heart is often pale and soft. The pleuræ are ecchymosed, and the lungs may contain infarctions. There is usually an acute catarrh of the whole length of the alimentary canal, and the stomach sometimes shows hemorrhagic erosions; but in Mr. Leggatt's case the stomach was pale and its mucous membrane showed no swelling.

The liver may present patches of a bright yellow color, or its tint may be that of *café au lait.* The only histological change which is usually described in it is that the hepatic cells are loaded with fat globules; but in Mr. Leggatt's case, Dr. Greenfield, who made the autopsy, found that the portal canals were crowded with leucocytes, that many of the bile ducts were filled with swollen epithelium, and that the greater part of the hepatic cells were undergoing disintegration, being swollen and fused together, and breaking up into irregular fragments. It may, however, be a question whether the infiltration of the portal canals was not an accidental cirrhotic change, due to habitual indulgence in alcohol. Dr. Greenfield's observations are of great interest, as tending to support a doctrine upheld by Liebermeister in his work on "Hepatic Diseases," that yellow fever is nothing else than a "parenchymatous hepatitis," dependent upon a specific infection. The jaundice in this disease is described as "hæmatogenous" by those who distinguish sharply between hæmatogenous and hepatogenous forms of jaundice. The biliary passages are found free from obstruction; the gall bladder contains dark green or tarry bile.

The spleen is but slightly if at all enlarged; its tissues may even be firm and healthy looking. The kidneys are enlarged and may show points of suppuration; in the case already referred to, Dr. Greenfield found that the convoluted renal tubes had their epithelium swollen and proliferating, and that the straight tubes contained hyaline casts; but it is to be observed that the kidneys were in this instance affected with chronic disease, and that, therefore, even the acute changes may have been unconnected with the yellow fever.

The *diagnosis* of yellow fever is not always easy except when it is known to be prevalent. The affection most apt to be mistaken for it is the bilious form of remittent fever. I have given elsewhere the distinctions between them (p. 261). Other diseases that must be borne in mind are relapsing fever and the various forms of local jaundice attended with pyrexia.

Ætiology.—With regard to the origin of yellow fever there have been great differences of opinion, and perhaps the time has not yet arrived when final conclusions can be arrived at. One of the most striking features of the disease is that its infectious principle is often transported by vessels from one place to another. I need only cite a few instances which have become historical. Thus, in 1823, H.M.S. "Bann" carried yellow fever from Sierra Leone to the island of Ascension. In 1845 the "Eclair" steamer brought it from the coast to Boà Vista, one of the Cape de Verde Islands. In 1861 it was conveyed by a wooden sailing ship, the "Anne Marie," from Havannah to St. Nazaire, in France; in 1865, by a similar vessel, the "Hecla," from Cuba to Swansea. Moreover, the disease frequently passes from one ship to another, not only where they have been lying side by side in the same port, but also on the high seas. The "Anne Marie," for instance, infected seven other vessels which happened to be brought near her; and during the local epidemic at Swansea, a smack, the "Elinor," which took in cargo near the "Hecla," had her crew attacked after they had left that port and had gone to Lanelly. So, in 1795, the "Hussar" frigate captured a French ship, the "Raison," on which yellow fever was prevailing; only those prisoners who were believed to be in perfect health were transferred to the English vessel, but, notwithstanding this precaution, the disease soon broke out on board her.

These facts are readily explained by the hypothesis that yellow fever, like typhus and the exanthemata, is propagated by a contagious emanation from the bodies of the sick; but it is to be observed that the infection has often been found to cling to the hull, or perhaps to the cargo, of a particular vessel, after the crew have been paid off. In the "*Med.-Chir. Rev.*" for 1848, and subsequent years, there appeared a series of very able articles, which are now known to have proceeded from the pen of the late Dr. Parkes. He professes to furnish unequivocal proof that the disease is sometimes, if not always, contagious. He relates in full detail the circumstances which attended the diffusion of it in Boà Vista from two soldiers, belonging to the fort, who were lodged while ill in the chief town of the island, Porto Sal Rey, as well as from a laborer, who brought it direct from the "Eclair" into another town, Rabil; and in each case he shows that the next persons to be attacked were those who lived close to the patients and visited them.

On the other hand, most of those who have had the largest acquaintance with yellow fever have disbelieved in its contagiousness, in the ordinary sense of that term, and many facts have been adduced in support of their view. One is that, as Griesinger has pointed out, the disease often remains localized upon the sea shore, or in close proximity to the banks of a navigable river; it may even confine itself to a small part of a seaport town in the immediate neighborhood of the harbor. Thus, when it prevailed at Lisbon, in 1857, one hundred and eighty-two persons are said to

have left the city for different places in Portugal, carrying with them the disease, and eighty-six died, but in no instance was it communicated to the other persons in the places whither they went. In 1865 Dr. Buchanan, having investigated with great care the local epidemic at Swansea, came to the conclusion that "the evidence tending to negative personal contagion was about as strong as such evidence can, by its nature, ever be."

There are, at the present time, two alternative hypotheses; one, that the disease is miasmatic-contagious; the other, that it is simply miasmatic. The first of these is held by some recent German writers, but I am not acquainted with any facts which support it; I do not know of any instance in which yellow fever has been traced to drinking water, nor does there seem to be any reason to suppose that the evacuations of the sick contain a poison which enters the soil, and then undergoes a further stage of development which fits it for re-entering the human body. But there seems to be much to be said in support of the view that the infective principle is a pure miasm. By this I do not mean that it is identical with malaria. Dr. Parkes, indeed, in the articles above referred to, maintains that yellow fever is, or may be, developed out of ague, which, in fact, undergoes conversion from a non-contagious into a contagious disease. He considers it to have been proved, for example, by Dr. Bryson, that when the "Eclair" left Sierra Leone on her way to Boà Vista, there was no yellow fever in the former place, so that the crew of that vessel must be supposed to have derived the fever from the usual "endemic sources," to which they were "exposed in a most intense degree, both in their boat services and in the unhealthy anchorages to which the nature of their duties drove them." So, again, he cites the case of a party of men detached from H.M.S. "Syren," in the Banana Islands, in the summer of 1847; among these men, according to Dr. Bryson, a series of cases of fever arose, of which "the first was of a purely endemical character, while the later ones merged into yellow fever with black vomit."

The ultimate decision of pathologists as to this question will doubtless be governed to a great extent by their judgment as to the development of contagious erysipelas, or of diphtheria from simple non-specific affections. But there is one fact which seems to point strongly in the opposite direction, namely, that yellow fever protects against itself, so that a person seldom has it a second time. I must admit, however, that this circumstance is deprived of some of its force by the further facts that mere residence in a hot climate seems also to impart a considerable degree of immunity from yellow fever, and that negroes are scarcely, if at all, more susceptible of this disease, than they are of ague.

A particular instance in which yellow fever is supposed to have been set up by the emanations from swampy ground in Barbadoes, among the men of two regiments, in 1841, is recorded by Mr. Lawson in the "*Lancet*" for 1879. And it is obvious that the facts cited by Dr. Parkes with regard to the spread of the disease at Boà Vista are quite consistent with the hypothesis that the infective principle of the disease, having once been introduced into the island from the "Eclair" upon clothing, or upon the persons of those who landed from the ship, or having been carried into Porto Sal Rey by the soldiers who were taken ill in the fort, afterwards established itself in the soil, and flourished as a pure miasm. And it seems very probable that when infection clings to the hold of a ship, the requisite nidus for the specific microzyme is afforded by the bilge water.

Prophylaxis.—If yellow fever is a miamastic disease, the precautions which should be adopted in order to prevent its transport from one country to another may be greatly simplified, in comparison with the regulations laid down until recently by the best authorities, as, for instance, by M. Mélier in

France, after the St. Nazaire epidemic, in 1861. As Mr. Simon remarks in his "Eighth Report," the segregation of persons arriving from an infected town, and their confinement in a lazaret for a definite period of time, is, on this view, superfluous; and in a trading country like England, all but impracticable. The points to which the whole energies of a port sanitary officer should be directed are the isolation and disinfection of the vessel which is known or suspected to contain the virus of the disease. It should be compelled to anchor at a distance from all other vessels; and every part of the hold should be thoroughly cleansed. It would now be advisable to use carbolic acid for this purpose, rather than the chloride of lime, which M. Mélier recommended. The cargo, and the clothes and other effects of the sailors and passengers should be disinfected at the same time.

The necessity for stringent precautionary measures against the development of yellow fever in England and in other temperate climates, is, however, very much diminished by a circumstance which I have yet to mention, namely, that a high external temperature is believed to be a very important, if not essential, factor in its ætiology. It has generally been said to be incapable of maintaining itself except in hot climates; and I may remark that this affords a further reason for supposing that its infective principle multiplies itself outside the human body and where it is freely exposed to atmospheric influences. The regions in which the disease commonly prevails are all situated near the equator; and the occurrence of a local epidemic within the temperate zone seems constantly to be associated with an exceptionally sultry state of the weather at the time. This was the case at St. Nazaire, in France, when it developed itself there, in 1861; and also at Swansea, in 1865. The latitude of that place being 51° 37″, which is beyond the geographical limit usually laid down for the disease. So, again, upon low coasts and near the mouths of rivers, the worst months are generally July, August, and September; although in small, rocky islands within the tropics the period of the year from October to February is stated to be the most dangerous. Of late, however, some exceptions have been recorded to the rule that the infective principle of yellow fever cannot resist cold. Thus Dr. Archibald Smith recorded, in the first volume of the "*Transactions of the Epidemiological Society*," the fact that in 1855 it prevailed at Cuzco, in the Peruvian Andes, where (as he was informed) the temperature of summer rarely, if ever, reaches 65° Fahr. in the shade; and also in Cerro Pasco, with a mean temperature of 44° by day. So, again, Mr. Leggatt's patient, who died in London, was taken ill on March 21st, 1878, the mean temperature of the four previous days having been 54°. He had arrived at Southampton from Rio (where the disease was epidemic) on March 17th. There had been three cases of yellow fever on board the ship on her voyage home; at that time the days are said to have been hot, but the nights cool. Afterwards the weather had been cold.

Treatment.—The treatment of yellow fever is mainly symptomatic. Quinine has no specific influence on the disease, but it doubtless acts as a tonic. A gentle laxative, or an enema, is employed at the beginning. The cold pack seems to be useful in many cases. For the relief of sacral pains dry cupping may be used, or a blister may be applied. The irritability of the stomach may be checked by a hypodermic injection of morphia, or by the administration of a few drops of chloroform or of chlorodyne. Lime water is also mentioned as serviceable in this direction. The patient's strength must, of course, be kept up as much as possible, by beef tea; but bland liquids, such as thin arrowroot, barley-water, or chicken broth, are often best retained by the stomach. Gum-water, made by

dissolving three drachms of pure gum-arabic in six ounces of cold water, has been recommended to be given, a tablespoonful every hour or two when the state of the tongue indicates that the mucous membrane below is raw and irritable. Ice is, of course, to be used, if it can be obtained; and it seems to be the usual practice to allow the patient brandy well diluted with water, or some effervescing wine, such as champagne.

ERYSIPELAS.

DEFINITION OF THE DISEASE—CONTAGION—SPECIFIC NATURE—INCLUSION OF THE PHLEGMONOUS FORM—INCUBATION, ONSET AND COURSE—RECURRENT ERYSIPELAS—DIAGNOSIS—PROGNOSIS—TREATMENT.

Erysipelas is one of those diseases of which the literature dates back to Hippocrates; mediæval synonyms for it are *ignis sacer* and "St. Anthony's fire;" in Scotland, it is called the "rose," a name which is also given to it throughout Germany. There have, however, been very great differences of opinion as to what affections should be included under these designations.

Some physicians, especially in France, have been anxious to separate from the cases which are seen in surgical practice, of erysipelas attacking the limbs and the body after injuries, a "medical erysipelas" of the face and head; in England this distinction received Sir Thomas Watson's support. Another distinction, which at one time caused much discussion, was that between erysipelas and erythema. The latter term is applied to various forms of superficial inflammation of the skin, some of which will be described elsewhere in this work, but there can be no doubt that it has sometimes been held to include cases which really were examples of mild erysipelas. But if the scope of the word erysipelas has thus been unduly narrowed in one direction, in another direction it seems to have been made far too wide. Many English surgeons regard as "erysipelatous" almost all possible forms of spreading inflammation of connective tissue, including even such a diffuse affection of the retro-peritoneal structures as that which is apt to arise after operations on the rectum or on other pelvic organs. This view is strongly objected to by German writers, among whom Volkmann, in the "Handbuch" of Pitha and Billroth, must be specially mentioned. They will not even allow that we are right in describing under the name of "phlegmonous erysipelas" cases of suppuration or gangrenous inflammation, attended with intense redness of the skin, but affecting equally the subcutaneous and even the intermuscular textures.

It is clear, I think, that the settlement of all these questions must ultimately depend upon ætiological considerations. Erysipelas is a contagious disease. Of its propagation by contagion several examples were recorded many years ago by Dr. Wells, the celebrated author of the "Essay on Dew." For a long time continental observers disputed the possibility of such an occurrence, but Volkmann cites more than a dozen instances of it, and it is now universally admitted. Perhaps the most striking series of cases that have been recorded was brought before the Paris Academy in 1864, by Dr. Blin. One of the surgeons at the Lariboisière Hospital had under his care two patients suffering from erysipelas, when he was himself seized with it. A medical friend from Guise visited him and fell ill after returning to that place, where no other case of the disease then existed. That gentleman's servant was attacked, and also a relative who came to see him, and who lived in the neighborhood. The latter gave erysipelas to his wife, and

three members of another family who were repeatedly in contact with them during their illness, suffered in their turn. From this family the disease spread to two Sisters of Mercy, and they carried it to their home and gave it also to a medical man who attended them, and from whom it passed to his daughter.

By far the simplest view of the matter is to regard erysipelas as not only contagious, but *specific*, in the sense of being always dependent upon the entrance into the body of a definite *virus* from without, and in support of such an opinion very much may be said. It is a great mistake to suppose that the disease, like pyæmia, necessarily becomes prevalent wherever surgical patients are crowded together and are placed under unfavorable conditions. In the Crimean war, in the Austro-Prussian war of 1866, and in the Franco-German war of 1870 repeated examples presented themselves of hospitals which it was impossible to keep healthy, and yet erysipelas failed to appear. On the other hand, there have been many instances in which it has prevailed epidemically, sometimes in a single ward, or in several wards of some one hospital, sometimes in various institutions of a city or town. Whether it ever spreads as an epidemic over the inhabitants of an entire district, like the exanthemata, is more doubtful. Between 1841 and 1854 this is said to have been the case in various parts of the United States, but both Volkmann and Hirsch are of opinion that the disease was really different from erysipelas and allied rather to diphtheria. In a hospital the poison of erysipelas often clings to particular wards, and even to particular beds, with extreme obstinacy. Mr. Savory, in the "*Brit. Med. Journ.*" for 1873, remarks that at St. Bartholomew's, during a small epidemic which occurred there, the disease almost always, in passing from one person to another, attacked the nearest patient who had an open wound. And Mr. de Morgan, in "Holmes' System of Surgery," cites, on the authority of Dr. Goodfellow, a most extraordinary instance in which it spread in regular order throughout a ward of thirteen beds, to almost every patient in turn, going down one side of the ward and then up the other side. Even where the poison seems to adhere most closely to a particular spot, it may be that it really comes from a little distance off. A case in point is related by Mr. de Morgan. It having been found at the Middlesex Hospital that the patients in two adjacent beds with a window between them were specially apt to be attacked, the suspicion arose that this might be due to the presence of a dust bin in the area below. This was cleaned out and there were no further cases. But two years later the disease reappeared, whereupon it was found that the dust bin had again become foul, although no unpleasant effluvium from it could be detected. The adoption of the same measures as before rendered the beds again healthy. In this instance it might perhaps be doubted whether the impure air actually conveyed a poison to the patients, or whether it merely predisposed them to be attacked. But no such doubt seems to be admissible in regard to another set of cases, recorded by Dr. König, of Rostock, in the "*Arch. d. Heilkunde*" for 1870. In the hospital of that town a small epidemic was clearly traced to infection from the cushion of the operating table, which cushion had become deeply discolored by dried blood from former patients; from the day when it was removed no fresh case of erysipelas occurred. The cushion was now soaked in water, and a brownish solution was obtained, which was inoculated upon two rabbits, with the result that one of them had an affection which closely resembled erysipelas. Dr. J. Orth, of Bonn, has since recorded in the "*Arch. f. Exp. Path.*" for 1873, a series of experiments by which he has demonstrated the possibility of infecting rabbits by the subcutaneous injection of fluid taken from an erysipelatous bulla in man; he also transmitted the disease from rabbit to rabbit by inoculating with liquid from inflamed and œdematous

parts of the skin and even with blood from animals already infected. That erysipelas can be conveyed in a similar manner from one human being to another seems to be established by an old observation made by Doepp, who vaccinated nine children with lymph from a child who on the following day fell ill with erysipelas; all were attacked by it. Orth found motionless micrococci both in the fluid with which he began his experiments on rabbits, and in that which he subsequently made use of. He even succeeded in producing erysipelas by inoculating animals with micrococci that had undergone artificial cultivation. The presence of such organisms in erysipelas had, indeed, been previously demonstrated by Hüter, not only in tissues affected with the disease and in the œdematous fluid which saturates them, but even —in small numbers—in the blood.

Thus a good case seems to be made for the view that erysipelas is a specific disease, and for my own part I am strongly of the belief that this will be the conclusion at which pathologists will ultimately arrive. At present, however, many observers hold a very different opinion with regard to it. Mr. Hutchinson, as I have remarked at p. 39, considers it to belong to the class of diseases which he terms "contagious inflammations." He is convinced that it is often set up by a draught of cold air blowing upon a wound, as when a hospital ward has its windows kept open all night.

We must now revert to the questions of the relations between the various forms of disease that have been included under the name of erysipelas. As regards the "spreading inflammations" first mentioned, Volkmann himself admits that acute diffused suppuration, severe diphtheria, or even puerperal fever, when introduced into a hospital free from erysipelas, often forms the starting point of an outbreak. He cites a case by Pujos, in which a man who had a severe gunshot wound of the foot died of erysipelas, or, as he says, "pseudo-erysipelas," which in several places had passed on to gangrene. A brother, who attended him, was attacked with erysipelas of the face, and so was a nurse; and several other persons became affected with phlegmonous inflammations. Volkmann adds that those who attend upon cases of puerperal fever often become the subjects of facial erysipelas. Such occurrences are the more striking because it is admitted on all hands that contagiousness is a far less conspicuous feature of erysipelas than it is of many specific maladies. But, after all, it is quite possible to account for them without giving up the view that erysipelas is a specific disease. It may be that there are certain cases of puerperal fever, or of cellulitis, or of diffused phlegmonous inflammation, which owe their origin to the poison of erysipelas, and that these cases alone are capable of reproducing it in their turn. Or it may be that just as in cultivation experiments there is great difficulty in keeping fluids free from accidental contamination with other bacteria besides those which one wishes to develop, so the discharges from diseased surfaces in living patients afford a favorable nidus for the growth of various kinds of microzymes which may have no relation to the complaints from which these persons are suffering.

Until recently it was thought that erysipelas of the face and head, as physicians see it, differed altogether from the surgical affection in one very important respect, namely, in appearing upon the unbroken skin, and independently of any wound or abrasion. I believe that Trousseau was the first to point out that it very often has a starting point in some slight breach of surface, as from a suppurating pimple at the angle of the eye, which the patient may have scratched, or from a trifling eczematous eruption on the nose, or from a fissure at the corner of the mouth, or even from a sore gum due to a decayed tooth, or from a herpetic affection of the fauces. This view has since been adopted both by Volkmann and by Zülzer. The most

careful record that I have met with of observations made for the purpose of testing its correctness is by König. Among twenty-nine cases of erysipelas of the face or scalp which occurred in the inmates of a prison at Ziegenhain, fifteen were traceable to previous injury of the affected parts; in the remainder no such starting point could be found, but in all of these the face was the seat of the disease, and in many of them it was so much swollen when the patient was first admitted that no complete examination could be made.

The supposition, of course, is that the contagious virus of the disease settles upon the spot which is afterwards to become the seat of the erysipelatous inflammation, and that infection of the blood is secondary. The complete want of symmetry in the distribution of the cutaneous affection may be mentioned as affording a strong argument in favor of this view.

Watson mentions cases in which the *incubation* of erysipelas appeared to last a week, and Murchison states that, in his experience, it was from one to three or four days. There is reason to believe that during this time local changes are actively going on, although there is no obvious inflammation of the skin. For it had long ago been noticed by Frank and by Chomel that the development of erysipelas of the face is often preceded by pain, tenderness, and swelling of the cervical glands; and Mr. Busk informed the late Campbell de Morgan that, after close observation of a large number of cases, he was convinced that this was an invariable occurrence. The most probable explanation of it seems to me to be that inflammatory exudation is really taking place, but that the lymphatics carry off the effused matters as fast as they appear. The possibility of such a modification of the inflammatory process is expressly admitted by Cohnheim. Doepp's vaccination cases, too, seem to show that the infective microzymes of the disease must at the same time be multiplying themselves locally, for it will be remembered that the lymph which conveyed erysipelas was taken from the arm of a child in whom the disease did not appear until the following day. Nor is there any difficulty in imagining that the blood may even at this early period be contaminated, both with microzymes and with inflammatory products, whether by direct absorption through the virus or by transmission from the lymph glands onward through the thoracic duct. And thus it is quite easy, without the hypothesis of a primary blood infection, to account for the production of constitutional symptoms, which seem to be premonitory of the local affection, although of course they are really not so. Watson says that before the outbreak of erysipelas the patient often feels ill, shivery, feeble, languid, and often drowsy; that his pulse is very frequent; that he may suffer from nausea, vomiting, and even diarrhœa. It must be added, however, that he speaks of sore throat "as an early, and almost a constant, accompaniment of the complaint." Volkmann declares that in the many cases in which he has watched the development of erysipelas in patients already in hospital, whose temperatures had been systematically observed for some time previously, he has never seen any prodromal symptoms whatever. In his opinion, whenever they seem to be present, the fact really is that the local affection has already begun, but in some deeply-seated part, so that it remains undiscovered. Now, Trousseau has maintained that erysipelas of the face often starts from the mucous membrane of the palate and fauces, and reaches the skin by passing outward through the nostrils. It is obvious that such an affection might well account for the early swelling of lymph glands, to which I have already alluded. The matter is therefore one which needs further investigation.

A case in point, which interested me very much, came under my obser-

vation in 1882. A man who had had jaundice for some time was admitted under my care, on June 24th. His temperature was then 105°. He said that his febrile symptoms had begun with a slight rigor, on the evening of the 22d. I could not make out that there was any pylephlebitis or other local affection of the liver to account for the pyrexia. On the 26th, the end of the nose was found to be red, and a blush of erysipelas soon spread over the face. It was then remembered that when he first came into the hospital he complained of sore throat, and was hoarse, and that on the 25th the clinical clerk had noticed the submaxillary glands to be swollen.

The *onset* of erysipelas is, indeed, usually sudden, and it is often marked by rigors, but in persons already suffering from a febrile illness the increase of pyrexia may be comparatively trifling. The late Dr. Woodman (in his translation of Wunderlich's work) remarks that even in adults epileptiform convulsions are not uncommon. The temperature may rise in twelve hours to 104°, and usually reaches its *fastigium*, which may be at 105° or even 106°, within the first two or three days.

Soon after the fever sets in, some part of the skin begins to burn and tingle and becomes red and swollen. The redness rapidly deepens into a crimson blush, which under pressure momentarily fades, but returns as soon as the pressure is removed. Its extent increases until in a day or two it perhaps covers the whole of the face. Where it is spreading, it has a sharply-defined, raised border, beyond which, however, projecting processes advancing into the subcutaneous tissue may be felt with the finger. On the other hand, where it is stationary, its edge fades off gradually into the healthy skin beyond. There is extreme swelling, especially where the skin is loose, as upon the eyelids; these become converted into rounded prominences, and it is impossible to separate them, so as to get a view of the eyeballs. The features are so altered that the patient would not be recognised by any one. The surface is tense and shining, though it may be made to pit by keeping up a little gentle pressure upon it. There are often a few vesicles scattered here and there, or even blebs, which may reach a large size. Moreover, Volkmann confirms a statement originally made by Sanson, that with a lens minute miliary vesicles can always be seen.

For three or four days the disease may go on spreading, until, if it began upon the face or the head, it may cover the whole surface down to the root of the neck. Volkmann, however, remarks that the chin always remains untouched by it. The conditions which determine its advance in one line rather than another, have lately been carefully studied by Pfleger, whose views are cited by Zülzer with approval. It would seem that this depends mainly upon the arrangement of the subcutaneous connective-tissue bundles; they everywhere interlace, so as to form rhomboidal meshes, but these are usually horizontal or oblique, whereas upon the chin their direction is vertical. Pfleger also maintains that wherever the skin is tied down to the deeper parts the spread of erysipelas is, retarded or arrested, as, for example, along the crest of the ilium and Poupart's ligament. Erysipelas of the face and head seldom extends far upon the chest; but when the disease begins upon the trunk or upon a limb, it may wander until it has covered the whole body. If it goes on advancing for any considerable length of time, however, it always subsides in the parts first attacked, while it is springing up elsewhere. Beyond an affected area, small islets of redness may not infrequently be seen, but these are always connected with it subcutaneously. Volkmann declares that erysipelas never gives rise to two or more patches at a distance from one another. It may, indeed, happen that in a case of double amputation, both stumps are affected; or that in a case of erysipelas of the head, the disease breaks out a few days later around a pimple on the

leg; but such should be regarded as multiple attacks of the disease. I have notes of the case of a child in whom, after a wound of the temple, redness first appeared there, and subsequently upon the chest, the right arm, and the left wrist, in turn; but I cannot say that the morbid process, might not have been continuous over the back of the body and limbs.

The morbid *histology* of erysipelas was first studied by Biesiadecki, and more recently by Volkmann and Steudener. In the dead body the disease is so little marked, the redness and swelling having almost completely disappeared, that Volkmann evidently was surprised at finding the deeper strata of the cutis, as well as the subcutaneous tissue, infiltrated with enormous numbers of granular leucocytes. He gives a drawing of the microscopical appearances, in which the cells are seen packed closely, side by side. In the more superficial strata they are, however, scattered very sparingly. The structure of the bullæ was investigated in 1868, at Vienna, by Dr. Bright, of New York. He found them to be divided into loculi by irregular septa, which are made up of cells of the rete, drawn out into long, spindle-shaped and branching processes. The fluid of the bullæ of course contains numerous leucocytes; and is often converted into pus.

While the local process is thus running its course, the pyrexia continues, the temperature rising and falling more or less irregularly, or remaining at nearly the same level. The pulse is quick, and it is generally soft and feeble; it may be dicrotic or intermittent. There is usually more or less delirium at night, and sometimes violent maniacal excitement. The patient often complains of headache, sleeplessness, and irritability of sight and hearing. He has no appetite, and is thirsty; the tongue is thickly coated; he has nausea and may vomit repeatedly; there is often diarrhœa with extremely fetid stools. The urine is scanty and commonly albuminous, and may even contain casts and blood.

The duration of erysipelas is variously stated by different writers; it is, in fact, very uncertain. Billroth says it seldom reaches fourteen days; Volkmann puts it at about six or eight days. When the disease wanders over the body and limbs it may run on for weeks or even months. The final defervescence is usually sudden, the temperature falling to the normal point in a few hours.

The subsidence of the cutaneous affection is also rapid, and then, four or five days after the commencement of the redness and swelling, the skin again becomes pale and flaccid, and shrivels. At the same time, other parts of the surface may be at the height of the disease, or may even be beginning to be attacked by it. Volkmann and Steudener have investigated the histology of this stage of the process; they find that in the subcutaneous tissue the leucocytes disappear with extraordinary rapidity, breaking down in a few hours into a granular *débris;* in the superficial layers of the cutis they remain visible a little longer, but within a day or two all signs of tissue changes may have vanished. In the meantime the vesicles or bullæ have dried up into yellowish crusts. The cuticle subsequently desquamates, and is detached either in flakes or as a branny powder. When the scalp has been the seat of the disease, the hair falls out for a time, but is soon reproduced.

Sequelæ.—But it does not always happen, even when a case of erysipelas ends in recovery, that the local affection subsides thus favorably. Delicate parts, such as the eyelids, the prepuce, or the labia sometimes slough, in consequence of the tension to which they have been subjected. In other cases when the swelling has gone down, abscesses form here and there beneath

the skin, and require to be opened with the knife. Suppuration of the swollen lymph glands is a very exceptional occurrence.

Sometimes erysipelas terminates fatally. This is especially apt to be the case where the patient has chronic disease of the kidneys, or has been intemperate, or is otherwise broken down in health. Before death the temperature usually rises to a great height, and sometimes it goes on rising for a short time afterward. When there have been severe cerebral symptoms, it has often been supposed that inflammation has extended from the scalp or the face to the membranes of the brain. Post-mortem examinations, however, have very rarely verified this suspicion. Volkmann says that he has seen the disease spread inward from the orbit, through the sphenoidal fissure, the orbital fat being found swollen and infiltrated with pus, as had been indicated during life by slight protrusion of the eyeball. It is not uncommon for the disease, when it affects the fauces, to extend onward to the larynx, and so to destroy life, unless tracheotomy succeeds in obviating the danger. The folds at the entrance of the larynx are then found intensely œdematous, or even infiltrated with pus. In other cases the immediate cause of death is pneumonia or pleurisy. In the *Guy's Hospital Reports* for 1861, Dr. Wilks recorded two cases in which erysipelas of the legs and of the surface of the abdomen appeared to have set up a fatal peritonitis. Another complication, of which two instances have been observed in France by Larcher (*Arch. Génér.*, 1864), is ulcer of the duodenum; it is of great interest, on account of the occurrence of a similar affection as the result of burns and scalds. In a third example, reported by Malherbe (*Arch. Génér*, 1865), there were ulcers in the lower part of the small intestine. Bayer has related in the *Arch. d. Heilkunde* for 1870, a case in which severe hemorrhage from the bowels preceded death; but the only lesion found at the autopsy was an intense congestion of the ileum. There has been some difference of opinion as to whether pyæmia is frequently associated with erysipelas in the same patient. Zülzer says that this is not the case, at least if the phlegmonous forms of disease and those attended with diffuse suppuration of the connective tissue be excluded from the definition of erysipelas; and our observations at Guy's Hospital seem to bear out this statement. Volkmann, indeed, declares that metastatic abscesses were present in from one-third to one-half of all his fatal cases; but he adds that every one of his patients also had a severe wound.

Erysipelas Faucium.—I have omitted to mention that during an epidemic of erysipelas in a hospital, it is no uncommon thing for cases of sore throat to occur which are evidently of the same nature, but in which the skin remains unaffected. A description of this form of the disease has been given by Cornil, based upon a study of eighteen cases. He speaks of a shining, purple-red, œdematous swelling of the faucial mucous membrane, sometimes accompanied with the formation of bullæ. The tonsils may take no part in it. The lymph glands below the jaws and in the neck are much enlarged. There is considerable pain in swallowing, and sometimes a profuse flow of saliva.

Recurrence.—Erysipelas seems to have no tendency to protect against its own recurrence in the same individual. In surgical wards it used to be no uncommon thing for a patient to have two or even three successive attacks while a large wound was healing. Women sometimes have the disease once or oftener every year; I think that in such cases it is generally directly excited by cold, as by exposure to an east wind; but Zülzer and other writers say that it almost always has a starting point in some local affection of the face, or of the nasal mucous membrane, or of the ear, or of the lachrymal passages. In course of time this "habitual" erysipelas leads to a persistent

thickening and induration of the nose and ears and eyelids which greatly deforms the countenance.

On the other hand, a remarkable effect of a single attack of the disease is that its subsidence is sometimes followed by the disappearance of long-standing cutaneous affections. This seems to have been first noticed by Cazenave in cases of chronic eczema or lupus. More recently it has been found that even sarcomatous growths of considerable size may vanish in a similar way. Volkmann gives copies of photographs taken from a woman under the care of W. Busch, who had several tumors on the face, varying in size from a hazel-nut to a pigeon's egg; a portion of one of them was excised, whereupon she was attacked with erysipelas, and this led to the absorption of all the rest. In two other patients, enormous tumors of the cervical glands underwent a great decrease of size under similar circumstances; symptoms of collapse, however, set in, which in one case proved fatal, while in the other case, as recovery took place, the growth rapidly regained its former dimensions. This patient had been intentionally exposed to erysipelatous infection, in the hope that it might act beneficially upon her disease. The tumor from the former patient, who died, was examined histologically by Rindfleisch, who found that almost the whole of it had undergone fatty degeneration, so that only in certain portions could the structure of a round-cell sarcoma still be recognized.

The *diagnosis* of erysipelas is generally very easy, if we leave out of consideration the theoretical doubts as to the relation which it bears, on the one hand, to phlegmonous affection and diffused suppuration, and, on the other hand, to certain forms of erythema. Zülzer speaks of the œdematous form of Charbon as being sometimes mistaken for it. There is, however, one disease about which I have repeatedly seen blunders committed, and that is zoster of the forehead and face. Only a few weeks before the time at which these lines are being written, I one day found lying in the hospital a youth whose face was covered with flour, through which a diffused redness was visible, while the eyelids were enormously swollen. The first glance, however, showed that the affection scarcely, if at all, passed the middle line of the forehead; and according to the account which the patient himself gave, it had begun with an eruption of vesicles. I therefore made a confident diagnosis that the case was one of zoster, and I had atropine dropped into the eye, on account of the danger of iritis. At my next visit, two or three days later, the redness and swelling had disappeared without leaving any desquamation of the cuticle; and the vesicles had dried up into characteristic dark-brown eschars, embedded in the skin. He left the hospital a week after his admission.

The *prognosis* of erysipelas is generally favorable, at least for the cases that come under the care of physicians; except, indeed, in very old people and in infants. In the latter, it not infrequently appears at the umbilicus, and proves rapidly fatal. Even in surgical practice recovery so generally takes place that it is difficult, as Dr. Wilks has remarked, to understand how it came to pass that the common form of certificate submitted to a magistrate, in cases of slight wounds, used to be that there was no danger "unless erysipelas should ensue." It may be, as he suggests, that what really was pyæmia was often set down to erysipelas. There can be no doubt, however, that erysipelas itself was once far more fatal, at least in certain institutions, than it is at present; thus Volkmann says that in the old Hôtel Dieu at Paris, it would often happen that the majority of surgical cases attacked by it ended in death. But probably much of the difference lies in the fact that patients suffering from erysipelas are now well supplied with beef tea and wine and brandy, instead of being bled and leeched and kept upon low diet.

Treatment.—In this country we believe that the tincture of steel has a marked influence in checking erysipelas. Mr. de Morgan, from his experience at the Middlesex Hospital, speaks most positively of its efficacy in reducing the duration of the disease, so that it subsides in from two to four days, instead of lasting a week or ten days. He gave at least a drachm or a drachm and a half daily, and sometimes as much as an ounce and a half or two ounces. On the Continent, it is more usual to give quinine. When a case comes under observation at the very commencement of the disease an emetic is believed by many to be useful; while Dr. Ringer believes that aconite, administered at this period, often succeeds in cutting short the attack.

Rather more than fifty years ago Mr. Higginbotham introduced the practice of applying nitrate of silver round the circumference of a patch of erysipelas, in the belief that its spread might in this way be arrested. This was termed the "ectrotic" method, and there are still some who have faith in it, including Volkmann, who prefers the silver salt to tincture of iodine, which has since been suggested with a similar object. The part must be first carefully washed with soap and water, or with a solution of soda or potash, so as to remove all fat from its surface. It may then be brushed over with a solution of the nitrate, in from eight to ten parts of distilled water, for a distance of some inches round the reddened area, on all sides of it. Sometimes the disease ceases to advance further, and on the following morning defervescence occurs, as is shown in charts given by Volkmann. The application of collodion all over the affected surface is said to be very useful as a palliative. I have, however, generally allowed the nurses to carry out the traditional plan of dusting flour upon it. When there is very severe smarting pain, or when violent delirium accompanies erysipelas of the head, an ice-bag is said to give great relief, and to be altogether harmless. If the swelling is so great that gangrene seems likely to occur, a series of minute incisions affords the most likely means of preventing it.

It is not often necessary to employ cold baths in the treatment of erysipelas; the pyrexia, although reaching a great height, usually subsides too quickly to be in itself a source of danger. But if a very high temperature is maintained for more than a week, one must carefully consider the question of adopting measures to reduce it. Volkmann gives a chart of a case in which a cold bath was given on the ninth day, the thermometer then indicating 105.8°; in the course of the following week this procedure was repeated thirteen times, and the patient recovered.

If death by collapse seems to be impending, it may be well to give turpentine, as was recommended by Copland. At least, it is difficult not to accept a case, of which he has recorded the details in his "Dictionary," and in which a woman who was already comatose, with a black tongue, and with a pulse that could not be counted, was apparently saved by this drug, administered in doses of three drachms in an electuary made with honey and also containing castor-oil.

DIPHTHERIA.

HISTORY OF THE RECOGNITION OF THE DISEASE—ITS DEFINITION—ITS PATHOLOGY: MICROCOCCI AND BACTERIA—RELATION TO "DIPHTHERITIC INFLAMMATION"—ÆTIOLOGY: CONTAGION—COURSE—VARIETIES—COMPLICATIONS—SEQUELÆ—ALBUMINURIA—PARALYSIS—PROGNOSIS—TREATMENT.

About the year 1857 medical men throughout England first had their attention forcibly drawn to the prevalence of an epidemic disease, resembling scarlet fever in being generally attended with an affection of the fauces, but differing from it in the character of that affection, and in the absence of a rash. Many accomplished physicians, including Addison, declared that they had never seen this disease before; and, although a search into medical literature brought to light several instances of its occurrence, both sporadically and in an epidemic form, yet the fact remained that, at least within the present century, it had never previously spread over any considerable part of the country at the same time. But in France it had been well known for several years, and had been carefully studied by Bretonneau, of Tours, who had given to it the name of *diphthérite* (*διφθέρα* = a skin or covering of leather).

Ever since, it has constantly prevailed with more or less severity, springing up here and there in different districts of England, as well as in other countries. Numerous investigations have been made as to its nature and mode of propagation, but in regard to many points doubts still remain, and with them a corresponding uncertainty about its relations to certain diseases which were believed to be distinct, but which some observers now suppose to be modifications of it. The designation which is now almost universally applied to it is Diphtheria. Senator, of Berlin, has indeed recently proposed to term it "Cynanche contagiosa;" but this name seems to have no chance of being generally adopted, although its use undoubtedly eliminates many sources of error and confusion, as will be found by any one who may read Senator's paper in the second series of German Clinical Lectures, published by the New Sydenham Society.

As the term diphtheria implies, the essential character of the throat affection is the presence of a membranous substance, which is spread more or less extensively over the tonsils, the uvula, or other parts. By Bretonneau, and the observers who followed him, great stress was laid upon the circumstance that this substance could be detached from the mucous membrane beneath, and that the latter was then found to be simply reddened and ecchymosed, or perhaps slightly excoriated. They pointed out that the adventitious layer, being of an ash-gray color, often simulated very closely the appearance which would have been produced by sloughing of the parts covered by it; and, indeed, that the disease had long been known under the name of gangrenous angina (malignant sore throat). But they declared that such appearances were misleading, and that no considerable loss of substance occurred, still less any extensive sloughing. Finding that

in many cases the pellicle extended down into the larynx, they took up the opinion that the complaint known as membranous croup was only a form of diphtheria.

By German writers the subject has been developed in a very different direction. Virchow, in the first volume of his well-known "*Archiv*" (published in 1847) distinguished between a "croupous" form of inflammation in general and a "diphtheritic" one. In the former, he said, the exudation lay free upon the surface of the mucous membrane; but in the latter it was seated within the superficial layer of that membrane, which generally underwent sloughing as the result of its presence. These definitions, being followed by subsequent writers, without a due comprehension of what probably was Virchow's real meaning, have led to great confusion, which is only beginning to be cleared up. The best description of the characters of "diphtheritic inflammation," in the sense attributed to it by the great leader of the Berlin school, appears to me to be that given by Rindfleisch in his "Pathological Histology." He makes it consist of an infiltration of newly-formed cells into the sub-epithelial connective tissue of the mucous membrane; and, following an idea originally suggested by Buhl, he teaches that the abundance of this infiltration compresses the blood vessels, and so arrests the circulation through the affected parts, brings their nutrition to a standstill, and deprives them of life. As examples of such an affection, he cites the putrid inflammation of the urinary bladder that is set up by decomposition of stagnant urine, the more severe forms of dysentery, and the dangerous inflammation of the uterus and vagina which may occur immediately after parturition; and he points out that a similar condition may be met with upon other free surfaces; upon the skin, as in the more distinctive kinds of variolous eruption, which lead to permanent pitting; and upon wounds, as in hospital gangrene. It must be admitted that all these affections have characters in common, and deserve to be known by a special name.

But, if this view is to be taken of the distinguishing features of "diphtheritic" inflammation, one sees at once that the disease, "diphtheria," finds no place in it. And, accordingly, Rindfleisch describes the latter under croupous inflammation, and by the name of "pharyngeal croup." He gives a very full account, which I can confirm by my own observations, of the histological characters of the pellicles which are found upon the surface of the mucous membranes in that complaint. On the palate and tonsils, he says they consist, not of fibrin, but entirely of cells, which have undergone a peculiar glassy change in their protoplasm, and have become fused together, so that a series of little fissures alone indicates the original interspaces between them. According to E. Wagner, these cells are all derived from the original epithelial elements of the affected part. In the air passages, on the other hand, Rindfleisch describes the pellicles as possessing an exquisitely laminated structure, consisting of layers of cells which alternate, at tolerably regular intervals, with layers of a homogeneous substance, which seems to be fibrin.

Some writers have attached special importance to the fact that in different affections of the throat there are great variations in the degree to which false membranes adhere to the mucous surface on which they lie. But Rindfleisch points out that these variations depend mainly upon the *seat* of the lesion. The lining membrane of the air passages possesses a smooth basement membrane which is wanting in the fauces; and it is suggested that this prevents adhesion. As between the several diseases, therefore, the distinction is valueless.

The fact that in the very same case of diphtheria the pellicles on the tonsils and those in the larynx differ essentially in their histology, is, of itself

sufficient to show that our views in regard to the disease are not to be based on its pathological anatomy alone. And, in the fauces themselves, the morbid changes present wide variations of intensity. There is, in the first place, a "*diphtheria sine diphtheriâ*"; a variety in which the tonsils and uvula are simply reddened and affected with a catarrhal inflammation, but which can be plainly recognized as a manifestation of the disease, because it occurs in members of the same family simultaneously with the more severe forms. And when membranes are present, they differ greatly in extent and in thickness in different cases. Rindfleisch, indeed, speaks of the morbid process in the pharynx as being always insular, and as consisting of circumscribed milk-white spots, on an intensely hyperæmic base, which are never raised more than half a line above the level of the mucous surface. But no physician who has had much experience of the disease can be ignorant that in many instances the whole fauces, tonsils and uvula are covered with a thick, tough layer, which looks like wash-leather, and may ultimately be detached *en masse*. In 1858, Dr. Fuller exhibited to the Pathological Society a cylindrical cast of the pharynx, four inches long, which had been ejected by a girl aged eleven. Indeed, if such false membranes are removed, or become detached, they may be renewed again and again. This is, I think, well known to all who have observed severe epidemics of the disease; and Dr. Sanderson vouches for it as the result of his own observation in 1859. "At Crowle," he says, "I had an opportunity of seeing an example of extreme rapidity of reproduction of faucial and pharyngeal concretion; and I have notes of a case, in a robust adult at Hertingfordbury, watched with the greatest care and attention, in which on some of the mucous surfaces the pellicle must have been renewed from twenty to thirty times in the course of three or four weeks." I quote this statement because Rindfleisch expressly denies that false membranes on the palate or tonsils ever recur in the same place, unless they have been stripped off before their spontaneous maturation; and Oertel (in his article in Ziemssen's 'Cyclopædia') likewise limits the possibility of their reproduction by conditions which I cannot fully comprehend. Another point on which Sanderson speaks very positively is the frequent occurrence of ulceration, which, in two cases that he observed, led to perforation of the palate; and he mentions one instance in which the tonsil and the arch of the velum were destroyed, so that a continuous sloughing surface extended to the pharynx. Bretonneau observed that in cases which presented exactly the appearance of sphacelus—the uvula and tonsils seeming to be in a state of "putrid dissolution," almost entirely detached—the fauces were found after all to be intact, when the false membranes at length came away. But all recent writers, both French and German, admit that Bretonneau went too far in denying the possibility of gangrene.

Within the last few years some observers have endeavored to cast the whole subject of diphtheria into a new shape, in consequence of the discovery that the structures concerned in the disease are permeated by living organisms. This discovery, which seems to have been made by Buhl in 1867, and by Hueter and Oertel independently in 1868, is one of which the real value is still doubtful; but I must briefly recapitulate the main facts which have been brought forward. The organisms in question are among the most minute that are known to exist. They are usually spherical in form, in which case Oertel and others call them *micrococci;* but mixed with these are some which are rod-shaped, and which are known by the name of *Bacterium termo*. To distinguish the former from mere granules of albumen or fat is very difficult, if not impossible; except when they perform active movements, which is not always the case; some

observers think that the way in which they are massed together shows that they have been developed by a rapid process of germination. The latter present a more characteristic appearance, especially as they are often arranged in long chains; but then precisely similar bodies exist in abundance in all putrid fluids.

According to the writers who lay stress upon the existence of these parasites in diphtheria, they are found not only in the false membranes (both within the epithelial and other cells, and outside them), but also in the subjacent structures. They are described as being present from the very commencement of the morbid process, and as extending beyond the limits which appear to the naked eye to separate the affected from the healthy tissues. They are said to block up the lymphatic channels, to penetrate into the interior of muscles and cartilages and bones, and to be carried by the blood to distant organs, such as the kidneys, when they germinate afresh in their glomeruli, and in their secretory tubes.

Now, there is, I believe, no question that micrococci are really present in the tissues primarily affected by the diphtheritic process. But many authorities doubt the validity of the observations in which they have been supposed to be detected in the blood, since granules, even in health, exist in it. And the investigations of Dr. Creighton have shown how great caution is required in the interpretation of such appearances as those which have been held to indicate the occurrence of organic forms in parenchymatous organs, such as the kidneys.

But an important distinction must now be drawn. The first observations of Hueter were made, not upon cases of "diphtheria," as we define the disease, but upon those of hospital gangrene. More recently several writers have described a form of endocarditis, in which vegetations on the valves of the heart have been found full of micrococci. And Virchow met with similar organisms in the kidneys of women who had died of puerperal fever. Now, no clinical observer will maintain that these affections are identical with diphtheria. In other words, the parasitic theory is applicable rather to "diphtheritic inflammation" in the wider sense of that term than to "diphtheria."

The conclusion just stated appears to me to be also fairly deducible from a large number of experiments, made by different observers, in which animals have been inoculated with "diphtheritic" products. Portions of false membrane have been inserted beneath the skin, or introduced into the trachea of the dog or the rabbit; or the cornea of the eye has been punctured with a poisoned needle. The result has been a disease which proved fatal in two or three dogs. The tissues in the neigborhood of the spot to which the infective material was applied have been found full of masses of micrococci; and Oertel says that he detected these organisms even in distant parts. But Recklinghausen succeeded in generating a "diphtheritic keratitis" in a rabbit, by inoculating the cornea with matters from a case of hospital gangrene. In Oertel's series of experiments, he passed on the infection from one animal to another, choosing sometimes the trachea, and sometimes the muscles of the neck or chest, as the seat of the induced disease; and after six transmissions he obtained a product capable of giving rise to the formation of a false membrane in the air passages of the last animal experimented on. One may reasonably suppose that this result was due to some specific property of the material used for inoculation, other than its mere power of infecting the tissues with micrococci.

Indeed, were it not for the experiments just referred to, we should be strongly disposed to think that the constitutional disturbance caused by this so-called "diphtheritic" inflammation, excited by inoculation in the lower animals, is perhaps, after all, nothing but a septicæmia; and that the

substances employed act merely as other decomposing substances might act. In taking such a view of the matter we might suppose that the presence of micrococci in the affected tissues in cases of diphtheria simply indicates the unhealthy character of the inflammatory change in them. This notion accords well with the statement of some recent writers that the false membranes in the larynx and trachea contain scarcely any living organisms; for, as we shall presently see, those cases of diphtheria in which the air passages are affected commonly end fatally before a septic process begins.*

One very important question in regard to the pathology of the disease has still to be considered, namely, whether it is from the first constitutional, or whether Oertel is right in maintaining that it is originally local and infects the system secondarily. His chief argument is based upon a contrast between the results obtained in the experimental transmission of diphtheria to the lower animals, and those which follow the inoculation of horses with glanders. In the former case, he says, the affection always develops itself at the spot to which the poison is applied; in the latter it invariably affects the nasal membrane, even though the contagious principle should have been inserted beneath the skin. But it is to be observed that even this view of the matter leaves diphtheria in the same position as syphilis and variola. I think there can be no doubt that the disease has really a special tendency to attack the fauces, and that the frequency of throat diphtheria does not merely mean that the poison comes into contact with this mucous surface more frequently than with any other. At any rate, it is certain that when the affection has begun elsewhere the throat often suffers secondarily, and apart from any direct extension of the morbid process. Thus we have had in Guy's Hospital a man who was admitted with an affection of the prepuce, of a doubtful nature, but in whom before his death the palate and tonsils became covered with a false membrane, so as to clear up the nature of the case. And in epidemics of conjunctival diphtheria, of which several have occurred in Berlin, the fauces have occasionally been attacked.

In the *ætiology* of diphtheria the first point to be insisted on is its contagiousness. This, indeed, is not always a very obvious feature of the disease as it is seen in private practice; for if several cases occur in succession in the same house, one cannot say whether they all depend upon a common cause, or whether one has given rise to another. But when a patient suffering from diphtheria is admitted into a hospital, it often happens that persons occupying beds in the same wards are subsequently attacked. And Sir William Jenner relates several instances in which patients sent into the country infected those with whom they came into contact. The epidemic in East Kent, in 1856, was ushered in by a striking instance. "No case of diphtheria had ever been seen in Folkestone," says Mr. Eastes, of that town, "during my time, until Isabella W—, aged 4¾, arrived from Boulogne, on the evening of July 2d, being then in an advanced stage of the disease. She died on the following day. On the 6th, her sister, aged ten, was attacked, who had always resided on the East Cliff; another case occurred in the same house three days after, and they all terminated fatally." But the most conclusive of all, perhaps, are the instances in which a medical man has fallen sick with diphtheria, immediately after having had phlegm coughed into his mouth or nose by a patient whose fauces he was examining; or after having used his lips to inflate his patient's lungs, or to blow through a tracheotomy

* On this difficult subject, compare Cohn's statements as to the distinction between *Micrococcus septicus* and *M. diphthericus*. ("Beitrage zur Phys. der Pflanzen," 2te Heft, p. 164, *et seq.*)

tube. Oertel mentions by name five physicians whose lives were thus sacrificed, among whom was Valleix, the writer on neuralgia. A case belonging to the same category is that of Dr. Wiessbauer's child, who was attacked shortly after having put into his mouth a cannula that had just been removed from the throat of a patient suffering from diphtheria. Oertel, indeed, is inclined to assume that in ordinary cases in which infection is traced from one person to another the act of kissing has afforded a direct means of transference of the poison.

But even when the conditions seem to be altogether favorable to the spread of diphtheria, it sometimes fails to propagate itself. Those in attendance upon patients suffering from diphtheria have been known to blow through tracheotomy tubes for the purpose of cleaning them, over and over again, without suffering any ill effects, and Trousseau and Peter actually inoculated themselves on the palate and tonsils with diphtheritic matter, and were none the worse. There is not, indeed, anything very surprising in these facts, for persons exposed to contagion from other diseases not infrequently escape. But many writers seem to think that diphtheria is infectious in a much lower degree than the exanthemata. Thus Oertel says that it diffuses itself over a district much more slowly than they do. However, I have long thought that the doctrine of the comparatively innocuous character of this complaint is in great part based upon the fact that so many physicians include under diphtheria what I regard as a non-infectious malady, namely, croup. The question is one of great difficulty, and it can more conveniently be discussed hereafter. But I may state in this place that unless one allows that many cases in which the air passages become lined with false membranes, even when the fauces are likewise affected, are distinct from diphtheria, one must recognize and account for the fact that such cases can much less often be traced into communication with other cases of diphtheria (whether as having arisen from them, or as giving origin to them) than can cases belonging to the other forms of the disease. Thus in the "*Guy's Hospital Reports*" for 1877, I have recorded a series of fifty cases of what would be regarded as diphtheria in the common acceptation of the term; and whereas eleven of fifteen cases in which the larynx was free could be brought into connection with other cases (by either ascent or descent) only eight out of thirty-five cases in which the air passages were involved could be so brought; moreover, all the eight cases just referred to were cases in which the fauces were severely affected; of ten cases in which there were only very slight patches of false membrane upon the tonsils or palate there was not one in which any evidence of infectiousness was obtained. I shall hereafter have to discuss whether these facts must be taken as showing that diphtheria attacking the air passages is actually less contagious than usual, or whether they indicate that a large proportion of the cases in question really belong to a different disease, a simple non-specific inflammation, attended with the formation of false membranes.

In support of the opinion that diphtheria is highly infectious, must be mentioned the fact that its contagious principle sometimes adheres with great obstinacy to particular houses or apartments. Squire mentions that in a country house in Scotland a visitor was attacked while occupying a chamber in which a case had occurred eleven months before. The infection from a mild case may generate a severe one, or *vice versa*. How soon a patient ceases to be infectious is not yet known. Bristowe says that the disease has sometimes seemed to be communicated by a child who had apparently been well for two or three weeks.

Doubts have been expressed as to whether the fact that a person has had diphtheria implies a subsequent immunity from the disease. In Simon's "Report" for 1859, several cases are recorded of individuals who suffered

twice, at intervals of two or three months or longer. But it is to be noted that the second attacks are always more severe than the first ones.

The period of *incubation* in diphtheria is said by Trousseau to be from two to seven days. Oertel puts it at from two to five days; but he quotes cases in which it was longer. One such instance is related by Jenner; that of a young lady who sickened eight days after having been sent into the country, from a house where her brother and her sister had before been attacked. Senator even says that the interval may be as much as three or four weeks. Where there has been a direct transference of the poison from one person's fauces to another's, the period seems to be much shorter. Valleix had a pellicular deposit in the tonsil the day after he became infected with the disease, and died in forty-eight hours. Oertel supposes that the incubation is likely to be less prolonged during the prevalence of an epidemic, especially where the type is malignant.

In speaking of measles and of scarlet fever, I have mentioned that each of those complaints is now and then accompanied by an affection of the fauces resembling that of diphtheria. A fact related to Dr. George Johnson by Dr. Dewes, of Coventry ("*Lancet*," 1875), tends to show that both the specific contagia are present in some cases of this kind. Two brothers had been in succession attacked by scarlet fever, and had been separated from one another throughout the course of the disease. The elder had remained at his school, to be nursed; the younger had been sent to a cottage in the country. The former passed through a mild form of the disease; the latter had it severely, and also had his fauces covered with diphtheritic exudation. After a time, the man at the college sickened with scarlet fever; and he, too, had a diphtheritic throat. When both brothers had been convalescent for a fortnight, they were allowed to be together, and even to lie in the same bed. Presently the elder one fell ill of diphtheria with laryngeal complications, and died on the third day. A point to which Senator draws attention is, that when scarlet fever is accompanied with the formation of false membranes upon the fauces it scarcely ever happens that the morbid process extends to the air passages, although the throat affection may be apparently identical with that of diphtheria.

Reverting to the question of the causes of diphtheria, one must admit that the disease often springs up in isolated houses, and under circumstances which accord ill with the theory that it must be due to a specific contagion. In this there is not, indeed, anything to be wondered at; for the same thing is likewise true of all the exanthemata. But Dr. George Johnson has advocated the opinion that cases not directly traceable to infection are often due to contaminated water, or to the effluvium from foul sewers or cesspools. He relates the cases of four children, who were all attacked on the same day; three days previously, a cesspool distant about twenty yards from the house had been emptied; and the wind was blowing toward the house at the time, and conveyed to it a very offensive smell. Soon afterward two of the servants fell ill, and at length the mother. The only members of the household who escaped were the father (who was away from home all day) and one servant. I must add that there is reason to believe that, even when diphtheria is epidemic, and when the specific contagion has perhaps already been introduced into the system, exposure to cold may act as an exciting cause in bringing out the manifestation of the disease upon the fauces. At least, Dr Yeats ("*Ed. Med. Jour.*," 1876) states that this was the case in an epidemic which occurred in Auchtergaven, Perthshire, between March and June, 1875. Several persons, he says, were attacked who had been working late in their gardens, or

playing croquet on a damp lawn, or driving after sunset. Others, who had been confined to bed for a considerable time, from other causes, were infected on their first appearance in the open air. It is obvious that these cases have an important bearing on the question of the existence of a membranous croup distinct from diphtheria, since such an affection is supposed to be due merely to exposure to cold.

Whether cold, altogether independently of epidemic influences, can ever set up a simple, non-specific inflammation of the fauces, attended with the formation of false membranes, it is very difficult to say; I shall have to revert to that question when discussing the relations of croup to diphtheria. It is, at any rate, certain that such a morbid process may result from the action of more powerful irritants. I have related in the "*Guy's Hospital Reports*," for 1877, the cases of two children, in whom the palate and tonsils presented appearances exactly like those of diphtheria; but in one of them the affection was caused by swallowing a piece of hot potato, in the other by a burning stick, which had been thrust into the little patient's mouth by another child. A similar condition has been met with in three or four cases of scald of the throat, in which boiling water or steam had been sucked out of a teapot or kettle. Lately I found a well-marked separable false membrane on the lingual surface of the epiglottis, as well as on the base of the tongue, in a woman who committed suicide with white precipitate, but who lived six or seven days after swallowing the poison. There was extensive "diphtheritic inflammation" of the œsophagus, the stomach, and the lower part of the intestines.

It is well known that, instead of attacking the throat, diphtheria sometimes shows itself on the mucous membrane of the genital organs, or of the eyes, or upon denuded parts of the cutaneous surface. The study of such forms of the disease ought to throw great light on its ætiology. Dr. Braxton Hicks has recorded in "*Guy's Hospital Reports*," for 1871, an epidemic of diphtheria in the obstetric wards, in which two women were, in turn, affected with diphtheria of the labia, while two others had the ordinary affection of the fauces. Moreover, in Berlin, and some of the other cities of North Germany, where conjunctival diphtheria has prevailed to a remarkable extent, I believe that in each epidemic there have been some cases in which the disease has spread either downward through the lachrymal passages to the palate and fauces, or in the reverse direction upward, or, in which the throat and the eyes have been attacked simultaneously and independently. Moreover, the conjunctival affection, as I myself have observed, sometimes occurs as a sequela of measles, just as is the case with diphtheria of the fauces. The existence of so close a relation between these different forms of the disease affords a strong argument for its specific character, and against the supposition that it arises spontaneously, as the result of defective hygienic conditions. But it must be added that both Jacobson (of Königsberg) and Hirschberg admit a sporadic variety of conjunctival diphtheria; stating, for instance, that gonorrhœal ophthalmia often assumes a diphtheritic character. And it may be that the affection of wounds and raw surfaces known as hospital gangrene is a sporadic representative of cutaneous diphtheria. Jacobson thinks that the conjunctival disease, even when it is of purely local origin, may spread by contagion. I should hesitate to admit this; although it would certainly be somewhat analogous to the case of a contagious porrigo of the skin, which often seems to develop itself out of a simple eczema.

Distribution.—Whether the poor are proportionately more liable to diphtheria than the rich is perhaps a question. Sir William Jenner remarks that only a small number of his cases occurred in his hospital practice; and it is notorious that the disease often carries off very healthy and robust-looking

children. All writers say that it is far more apt to attack those who are under ten years of age than older persons; and there appears to be no doubt that this is the case, although I myself think that many observers have made the proportion of children appear unduly large by including membranous croup under diphtheria, since the former affection never occurs in adults.

There seems to be no proof that one season of the year rather than another is favorable to the spread of diphtheria. The very cold winter of 1860 did not hinder its epidemic diffusion in London; and it has often prevailed during the summer. Oertel says that it is of much less frequent occurrence toward the tropics than in the temperate zone and the adjacent part of the frigid zone. Geological conditions of soil seem to have no part in its causation.

Course.—The *symptoms* of diphtheria differ widely in its different cases. Sometimes the disease begins with marked constitutional disturbance; anorexia, headache, lassitude, and loss of energy; nausea or vomiting, acceleration of pulse, and shivering, followed by fever. There may from the first be difficulty of swallowing, the sensation varying from a slight pricking up to a severe darting or shooting pain, which may quite prevent the patient from taking food, but this pain is rarely so extreme as in acute tonsillitis. On looking at the fauces one finds that the uvula, the palate, and the posterior wall of the pharynx are more or less swollen, and of a red or violet color. After a few hours, or at latest two days, one or more whitish-gray spots begin to appear on some of those parts; they are at first small, and they may remain of the same size for several days. From an early period the lymphatic glands of the neck, especially those near the angles of the jaws, become swollen and painful.

In some instances the febrile disturbance is very slight, and in others, when it is more considerable, it is of very short duration. The patient often seems to have entirely recovered within two or three days, by which time the pellicles are sometimes detached and cast off.

But in many cases the disease takes a less favorable course. The fever continues, the temperature remaining at 102° or 103°, or mounting higher; or, if it should have fallen, it rises again on the fourth, fifth, or sixth day. The diphtheritic spots on the fauces rapidly run together, and extend in every direction; so that the tonsils, the uvula, and all the visible parts of the throat may in a few hours be covered with a yellowish or grayish membrane. The cervical and submaxillary lymph-glands swell still more, and the tissues about them may be so infiltrated with inflammatory products that the hollow of the neck is entirely obliterated, the whole space from the chin to the sternum being occupied by a uniform brawny mass, the skin over which is shining and red, as in erysipelas. Presently the diphtheritic membranes begin to soften and decompose, acquiring a brownish color, and separating here and there in shreds. The patient's breath then becomes horribly fetid. An ichorous discharge may run from the corners of the mouth, which excoriates the surface and gives rise to ulcers, which in their turn become covered with a grayish-white layer. At this time it is not uncommon for sore spots—behind the ears, for instance, or in the creases of the skin, even of remote parts of the body—to become distinctly diphtheritic. If a blister should unfortunately have been applied, the raw surface may be coated with a membrane; and leech-bites are apt to pass into unhealthy, spreading ulcers.

From the first commencement of these more serious local changes, the constitutional symptoms begin to present features of depression which characterize the whole further progress of the case. The pulse may still be quick, its beats rising to 120 or more in the minute, but it becomes daily smaller and weaker. The countenance acquires a waxy pallor, which per-

haps contrasts strongly with the ruddy aspect of the patient a few days before; the muscular power is greatly enfeebled. Purpuric spots often appear upon the skin. Delirium sometimes sets in, as in a case recorded by Sir William Jenner; the tongue may become dry and brown, sordes may collect on the teeth and lips, and, in fact, a "typhoid" state may be developed. Such cases are generally fatal, and on post-mortem examination the serous membranes, and even the endocardium and the substance of the lungs are commonly found to be ecchymosed, as in septicæmia. Pneumonia or œdema of the lungs is, in many instances, the immediate cause of death.

Most frequently the mind remains perfectly clear. At length the temperature falls below normal, to 97° or 96° F., and the pulse may become irregular and intermittent, its beats scarcely amounting to 50 or even 40 in the minute. Death may either occur very gradually, by asthenia, or more often it takes place suddenly, while the patient is in the act of sitting up in bed, or making some other slight movement. Such cases generally terminate between the tenth and the fourteenth day.

Fatal syncope, however, is not limited to those cases of diphtheria in which the disease has been severe throughout its whole course; it sometimes happens when all the symptoms have been of the slightest. Jenner relates such an instance; a boy, aged ten, who was convalescent from a very mild attack, was attacked with vomiting. His medical attendant, found on visiting him, as usual, that the pulse (which had been becoming less frequent for two days) was only 36 in the minute. There was nothing in the patient's appearance which would have suggested that he was in imminent danger of death, but notwithstanding the free use of stimulants, the pulse continued to fall; by the next afternoon its beats were only 24, and soon afterwards they ceased altogether. I have seen two similar cases in children. One was that of a little girl in whom there were all along well-marked diphtheritic patches in the fauces, but who seemed to have so little the matter with her, that she was allowed to play with other children in the garden in front of the house. Early one morning she was being brought down stairs from the nursery, as usual, before being dressed, when it was noticed that she looked very pale, and I was hastily sent for. I found her pulseless, with her extremities perfectly cold, and in spite of all that could be done she died about eighteen hours afterwards, without having rallied in the least from her collapsed condition. I also remember a third case, which I believe to have been of the same kind, but in which there was no direct proof that the disease was diphtheria. It was as follows: A man, aged fifty, a sailor, was admitted into Guy's Hospital under the late Dr. Barlow, on June 24th, 1866. His pulse at the wrist was imperceptible, and he was unable to swallow. When he took any liquid into his mouth he made a gurgling noise and spat it out again. He was perfectly conscious, but his speech was thick, and he dragged his legs after him and was unable to walk without support. He said that he had been perfectly well until the morning of the 19th (five days before he was brought to the hospital), when "he found himself unable to speak; his throat afterwards felt sore, and seemed full of phlegm, and his breath was short." On the day after his admission he had a sort of fit, and threw himself out of bed. This was followed by a paralytic condition of all the limbs, and he seemed as if he would die at once; but he rallied a little and lived until 1.40 A.M. of the 26th. A post-mortem examination was made; no disease could be found in the body, and the only possible conclusion seemed to be that the case was one of diphtheria; but neither during life nor after death were any definite morbid appearances discoverable in the fauces.

Even when characteristic diphtheritic patches exist on the mucous membrane of the fauces, it sometimes happens that the real nature of the disease

may be overlooked from the patient failing to complain of difficulty of swallowing or pain in the throat. I have had children brought to me in the out-patient room at the hospital, suffering from febrile symptoms for which I could discover no cause until, as a matter of routine, I examined the fauces. And some years ago a chlorotic girl, who had been an inmate of one of the wards for a few days, died without any one suspecting the real nature of her disease. The chief symptom was a weakness so extreme that she could not sit up even when supported. A post-mortem examination showed that the fauces were covered with false membrane.

But perhaps the most obscure of all the forms of diphtheria is one which is limited to the *nasal mucous membrane*, or which, at least, may be unattended with the presence of any obvious pellicles upon the pharyngeal surface. The chief local symptom is, then, the escape of a thin, sanguineous or muco-purulent fluid from the nostrils, the orifices of which become more or less reddened and excoriated. Epistaxis, however, is not infrequent, and it may be so profuse that one would have been satisfied to attribute to it the bloodless appearance of the patient if it had not been for the fact that anæmia is so constantly present in all severe cases of diphtheria. With a speculum we may sometimes perceive that the turbinated bones are covered with a membranous layer, or casts of them may be discharged from the nostrils. After the first day or two the secretion from the affected parts is commonly of a brownish color and very offensive. The disease not infrequently extends along the nasal duct to the conjunctiva, which then becomes coated with a perfect diphtheritic membrane. Or it may pass through the Eustachian tube to the tympanum, causing a singing or buzzing noise in the ears and deafness; perforation may then take place, and matter be discharged through the external meatus.

Another direction in which diphtheria sometimes spreads, is from the pharynx down the *œsophagus*. In one fatal case I found a number of small ulcers in the stomach close to the cardiac orifice, some of which were coated with a distinct layer of false membrane; and instances have been recorded in which the whole œsophageal and gastric mucous membranes have taken part in the morbid process. The condition seems to have given rise to no special symptoms.

Of all the secondary forms of diphtheria, however, arising by extension of the disease from the fauces to adjacent parts, the most important by far is that which affects the *air passages*. The continuity of the false membranes is often traceable directly over the epiglottis and the aryteno-epiglottidean folds; but sometimes this is not the case, and certain observers have, in consequence, been led to regard the spread of the disease to the larynx and trachea as the result of what they have termed "auto-infection," an inoculation of the mucous membrane by particles of secretion drawn downward with the air inspired through the mouth. Upon the epiglottis and the vocal cords the false membrane is firmly adherent; it may pass straight over the space between the true and the false cords. Below the glottis it is very loosely attached to the mucous surface. It becomes thinner as it descends, and in the trachea, at a variable distance down, it commonly ceases, and becomes continuous with a muco-purulent layer which lines the rest of the air passages. But in some cases, even the bronchial tubes within the lungs present a delicate tubular diphtheritic lining. Oertel quotes Bartels as having pointed out that this is always confined to those tubes which run upward or downward in the back part of the lungs, and never occurs in those which course forward toward their anterior edges. These writers also maintain that the existence of old pleuritic adhesions over any portion of a lung favors the penetration of the fibrinous exudation into the corresponding bronchial tubes.

It is seldom practicable to employ the laryngoscope to determine the presence of false membranes in the air passages in a case of diphtheria, and only a very practiced observer would be able to obtain a satisfactory view of the interior of the larynx, at least in a child. The diagnosis of laryngeal diphtheria rests mainly upon the fact that the entrance of air into the lungs is impeded. This is shown, not only by the rapidity and gasping character of the respiration, but also by the way in which at each breath the soft parts above the clavicles and sternum are sucked in, as well as the lower intercostal spaces, and in young children even the sternum and ribs, which at an early age are soft and yielding. Every time that the patient inspires, a loud, crowing or croupy noise may be audible, and the cough is often hard and brassy. In other words, the symptoms are precisely the same as those of croup, and as I have already stated more than once, some observers believe that croup, when attended with the development of membrane, is always an effect of the diphtheritic poison. The dyspnœa is apt to become greatly aggravated from time to time, a circumstance that must be due either to the supervention of spasm in the muscular walls of the affected parts, or to the accidental impaction of portions of membranous or other secretion in the narrowed chink of the glottis.

When diphtheria affects the larynx, extension of the disease to that part commonly occurs within from three to six days after the commencement of the disease. Jenner, in 1861, had never known it delayed beyond the end of the first week; but Oertel says that it is not infrequent on the eighth or tenth day, and may be as late as the thirteenth. In Jenner's cases death always occurred in five days from the setting in of laryngeal symptoms; and he says that out of twenty-six fatal cases of Bretonneau's there were only five in which life was prolonged after the third day, and that there was but one in which it was prolonged after the sixth day, except as the result of operative interferences.

Were it not for the circumstance that the presence of false membranes in the air passages is in itself dangerous to life, from mechanical interference with respiration, it is probable that many cases in which the disease assumes this form would be of a mild type. In other words, one need not wonder that the laryngeal variety of diphtheria, even when it terminates fatally, is often unattended with those symptoms of bodily prostration, anæmia, and depression of the heart's action, which play so prominent a part in many uncomplicated cases of the disease.

It is said that sub-pleural emphysema is of frequent occurrence from distention and rupture of the pulmonary alveoli, and that the escaped air may diffuse itself into the mediastinum and even into the subcutaneous textures of the neck and of the body generally. Bartels, in one case, heard a loud sound on auscultation, which he supposed to be due to pericarditis, but which proved to depend upon the presence of a quantity of air in the areolar tissue outside the sero-sacs.

In certain cases, diphtheria seems to commence in the air passages, there being no primary affection of the fauces. In the epidemic form of the disease, however, this is of rare occurrence. Bretonneau states that he met with but two instances of it; of one of these he gives details (Case 45 in his fourth "Memoir"). But I think that there is no more than a presumption against its being a case of simple membranous croup—unless, indeed, we absolutely deny the existence of that disease. The patient was an infant, a year old, in charge of a nurse at Tours, where no case of diphtheria had been seen for months. The nurse was a native of a hamlet some miles distant, where the disease was prevailing, and a nephew of hers had died of it a few days before the infant fell ill. Bretonneau puts the relative frequency of a primary laryngeal diphtheria at one in thirty

cases; Guersant at one in twenty cases. In an epidemic which occurred at Auchtergaven, in Perthshire, Dr. Yeats ("*Ed. Med. Journ.*," 1876) observed, among one hundred and eighty-three cases, fifteen in which laryngeal symptoms were present from the commencement, but in which there was no visible affection of the fauces when they were first brought under notice; and in six of these the pharynx remained free throughout the whole progress of the disease.

When we come to discuss the question of croup, we shall find that it is important to know what is the proportion of cases of epidemic diphtheria in which the morbid process spreads to the larynx; but with regard to this I have been able to find no satisfactory information. In the reports made in 1859, by Greenhow and J. B. Sanderson, to the Privy Council, in reference to the epidemic which had been prevailing for three or four years in various counties of England, it is generally spoken of as exceptional. Among a large number of cases occurring at Spalding, in Lincolnshire, and recorded by Dr. Capron, there are only two in which it is mentioned. Out of twelve fatal cases that were seen by Mr. Schofield, of Highgate, nine terminated by "asthenia," three only by "croup." At Birmingham, it is reported that croupy symptoms were comparatively rare. Mr. West had never seen any marked affection of the larynx, and Dr. Heslop did not think that such complications occurred in more than five per cent. of the cases. Only at Stalham and at Smallburgh is it said that the majority of patients died with croupy symptoms or that such symptoms were very common. So, again, at Crowle, Sanderson reported that he had "no means of determining in how many cases the symptoms of laryngeal complication existed; it is certain, however, that they bore a small proportion to the fatal cases. At Launceston, on the other hand, out of twenty deaths, it appears that eighteen were preceded by the symptoms of croup." Now, it is true that in a schedule of questions, previously sent round to practitioners in the districts where diphtheria prevailed, it had been asked whether or not "the diphtheritic inflammation . . . *sometimes* descends along the mucous membrane of the air passages;" so that the medical men in question must have seen that the disease was regarded as distinct from the well-known membranous croup, and may to some extent have been influenced in excluding from their returns cases in which the main symptoms were laryngeal. But I think that no one can read their reports without coming to the conclusion that an extension of the disease to the larynx and trachea was comparatively infrequent.

On the other hand, the assertions of French physicians as to the extreme frequency of such extension are deprived of most of their value by the very fact that these physicians recognize no membranous croup apart from diphtheria. Again, Bretonneau's "Memoirs" contain forty-five cases, related in detail, and in the immense majority of them the air passages were involved. But one must remember that the main object of this writer was to "establish the identity of croup with malignant angina," under the common name of diphtheria; and in one place, after recording an ordinary instance of "pharyngeal diphtheritis," he says, "a larger number of special cases of this kind would present no interest."

It seems to me that Trousseau's statement that diphtheria extending to the air passages is the most common form of the disease requires allowance for the same fundamental belief on his part. For he proceeds to say that this is the form which diphtheria takes when sporadic, and also that which it exclusively assumes in severe epidemics. Now, the former assertion involves the very question at issue, namely, whether sporadic membranous croup is a manifestation of diphtheria. As I have already stated, I found in, 1877, in putting together a series of cases of diphtheria and croup which had

occurred at Guy's Hospital, that among fifty cases, such as would commonly be regarded as examples of the former disease, there were thirty-five in which the air passages were affected. But I shall hereafter endeavor to show that in many of them the disease must have been a non-specific inflammation.

The *urine* in diphtheria is not only scanty and high colored, as in other acute diseases, but it very commonly contains a considerable quantity of albumen. This fact, which was first pointed out by Dr. Wade, of Birmingham, in 1858, is of great value, as sometimes aiding in the diagnosis of cases that would otherwise be obscure. There may also be epithelial and hyaline casts, and, much more rarely, blood. Eberth is quoted as having found that in his experience albuminuria was present in two cases out of three; but the proportion seems to vary in different epidemics. In some patients it is observed within a day or two from the commencement of the disease; in others not until convalescence already seems to be established. It is sometimes very transitory, and may be detected only once or twice, even in cases in which the urine is repeatedly examined; but it more often lasts for a week or two. It is generally supposed to be devoid of prognostic significance; but Oertel, although he admits that the urine of some patients who die rapidly contains very little albumen, yet says that he has been able to make out a close relation between the quantity of this substance excreted in the twenty-four hours and the general intensity of the disease. From one to three drachms was the amount usually passed in cases so severe as to threaten life or to terminate fatally; and the albuminuria persisted, in those who recovered, for six or eight weeks after the subsidence of the throat affection.

When an opportunity is afforded of observing the state of the kidneys, they are said to be found large and congested, the epithelial cells being swollen, opaque, and granular, and filling the tubes. Minute extravasations of blood are also described as present in many cases; and, according to Oertel, masses of lymph corpuscles often surround the capsules of the Malpighian tufts. The renal changes thus appear to be undistinguishable in kind from those which occur in scarlet fever; for I have already expressed doubts as to the real existence of the micrococci in the uriniferous tubes and Malpighian bodies and interstitial stroma, to which Oertel attaches so much importance. The observations which have been made at Guy's Hospital would, however, suggest the belief that the affection of the kidneys in the two diseases differs widely in degree; that observed in cases of diphtheria being comparatively of a slight character. And this accords with the well-known fact that dropsy very rarely occurs after diphtheria. A few instances of general anasarca have, indeed, been recorded by different observers; one, for example, by Oertel, in the "*Deutsches Archiv*" for 1871. But when this writer speaks (in Ziemssen's "*Handbuch*") of fifty fatal cases, mostly attended with suppression of urine and dropsy, as having occurred in Kiel and the neighboring villages, one is apt to doubt whether the epidemic was not really one of scarlet fever with diphtheritic complications. Sauné asserts that the rarity of dropsy in diphtheria depends on the fact that only one of the kidneys becomes diseased, and not both, as after scarlet fever; but no other observer (so far as I am aware) has found that this is the case.

It has been recently maintained by MM. Bouchut and Labadie-Lagrave that *endocarditis* is of frequent occurrence in diphtheria. I have repeatedly searched for such an affection in making autopsies in children who had died of the disease; but the valves have always appeared to me to be perfectly healthy. And I find Sauné (who, being also a member of the School of Paris, has had good opportunities of testing the value of the statements in

question) declaring that the slight irregularities that are commonly found under normal conditions on the upper margins of the mitral and tricuspid valves have been mistaken for vegetations.

Sequelæ.—Convalescence from diphtheria is sometimes attended with the development of a series of sequelæ, which are, perhaps, the most remarkable that have hitherto been traced to any acute disease; I refer to the so-called diphtheritic paralyses. These commonly begin during the second or third week after the subsidence of the throat affection, but they are said to be sometimes postponed until the lapse of a month or six weeks. They appear in a somewhat regular order. First, the soft palate is affected; it hangs flaccid, the uvula cannot be drawn up; its sensibility is lost, so that neither pricking it nor applying the solid nitrate of silver causes any pain. One consequence is that the patient speaks indistinctly, "or through his nose;" another, that when he attempts to swallow any liquid, part of it passes upward and escapes through his nostrils. The pharyngeal muscles are occasionally involved; deglutition is then difficult, and some of the food is apt to find its way into the air passages. Next, generally after an interval of a few days, one or more of the ocular muscles may be attacked; the patient then sees double and squints; or paralysis of accommodation sets in, affecting both eyes, so that he is not able to distinguish near objects properly, and cannot read small print with comfort. The limbs are commonly affected somewhat later still; but sometimes they are the parts in which a loss of power is first observed. Sensations of numbness or pain in the feet are complained of; and presently the legs grow weak and tremble, the gait is shuffling and uncertain, or the patient may be unable to stand without support. The arms are much more rarely involved but sometimes their strength fails so that he cannot dress and undress himself, or hold anything in his fingers, or even raise his hands in the air. According to Oertel, the electric sensibility and contractility of the affected muscles are greatly impaired or entirely abolished, and their substance undergoes a very rapid wasting. In some instances the bladder and rectum take part in the paralysis, and the genital organs, with complete loss of sexual desire in adults. Again, the muscles of the larynx may be affected, in which case the vocal cords may be seen, in the laryngoscopic mirror, to lie motionless, in a position midway between that of respiration and that of phonation—the position which they always occupy in the dead body, but which is never seen under normal conditions during life. Wilks has insisted on this affection as a frequent cause of suffocative dyspnœa, when a tracheal tube is removed, after having been worn for a few days. The inspired current of air draws the paralyzed cords inward, until they meet and close the glottis. Lastly, the muscles of the neck and of the trunk may be affected, the patient being unable to keep his head supported, or to raise his body from the recumbent position, or to turn over in bed. Or the diaphragm and the intercostal muscles may be paralyzed, in which case he is very likely to die of suffocation. Dr. Priestley, of King's College, was, in 1863, attacked with diphtheritic paralysis in a severe form; a very complete account of his case is to be found in the fifth edition of Watson's "Lectures."

As to the cause of these sequelæ of diphtheria there is still much difference of opinion. In one extreme case Oertel said that he found extensive changes in the spinal cord and its membranes; hemorrhages surrounding the roots of the nerves, proliferation of nuclei in the gray matter, fibrinous exudation upon the surface of the ciliated epithelium lining the central canal. In another instance Buhl observed hemorrhages in the membranes and substance of the brain, and a red, swollen, softened state of the roots of the spinal nerves, with accumulations of cells and nuclei in

their sheaths. The muscles have also been found atrophied and in a state of fatty degeneration. But it is difficult to believe that similar conditions are present in those cases which terminate quickly in recovery. And the fact that the palate is so commonly first affected is obviously favorable to the theory of a "*neuritis migrans*," a morbid process starting in the nerves of the part originally attacked, and spreading along the fibres until it reaches the centres. Such a view is quite consistent with the observation of Trousseau, that paralysis may follow cutaneous diphtheria; for, in the case related by him, the limbs seem to have been affected as early as the fauces. Moreover, Senator declares that an abscess of the tonsil may give rise to paralysis; and in speaking of mumps, I shall relate a case of that disease, attended with a remarkable depression of the vital functions, very like that which occurs after diphtheria. On the other hand, some writers maintain that the power of producing such nervous symptoms belongs to diphtheria only as a member of the large group of acute diseases, and that paralysis not unfrequently arises after typhoid fever, typhus or smallpox. But I think that this must be of infinitely rare occurrence. The only case in point that has ever come under my observation is that of a boy, aged five, who was admitted into Guy's Hospital, under Dr. Wilks, in the autumn of 1877, for a paralytic affection, which had commenced four months before, after a very mild attack of measles. He was unable to speak, and had difficulty in protruding his tongue, and in swallowing. He could not stand, in consequence of weakness in both legs, and his hands and forearms were rigidly flexed.

The *prognosis* in diphtheria is always doubtful; no case, however mild, is to be regarded as free from peril. The average mortality varies greatly in different epidemics, but, according to Oertel, it generally ranges between 30 and 40 per cent. The larger the proportion of young children among those who are attacked, the more fatal the disease; for in adults it comparatively seldom assumes the laryngeal form, in which from 90 to 95 per cent. die. However, according to Trousseau, the nasal variety is almost equally dangerous.

The main indications in *treatment* are to maintain the patient's strength, and to control the various tendencies that might lead to a fatal termination. From the very first, he should be kept strictly confined to bed. If the skin be hot, and the pulse quiet and of fair power, stimulants may for a time be withheld. But in many cases the heart soon begins to flag; brandy or port wine should then be prescribed freely, and nourishing soups (especially turtle soup) should be given in small quantities at very short intervals, night and day. Jenner mentions the case of a child, three years old, who had from three to five ounces of brandy given to him in twenty-four hours, with apparent advantage. At the same time tonic medicines are usually prescribed, of which the tincture of perchloride of iron is supposed to be the best. Dr. Squire speaks of administering as much as half an ounce of this preparation in the course of the day, even to very young patients, and still larger quantities have been ordered for adults. For some cases quinine, or bark with ammonia is preferred. Dr. Hermann Weber has drawn attention to the fact that the practice, universal in England, of giving abundance of nourishment in diphtheria, is far from having obviated the liability to the occurrence of the sudden collapse, which we have seen to be one of the principal modes in which the disease proves fatal; but whatever may be said with regard to this complication, I do not think there can be any doubt that the general tendency of such treatment must be good.

As to the *local treatment* of the throat affection, there has been uncertainty of opinion. All observers are now agreed that membranes upon the

tonsils and palate should never be forcibly removed. Bretonneau recommended that the diseased surface should be brushed over every day with strong hydrochloric acid, or a mixture of one part of the acid to two or three of honey. And, in 1861, Jenner expressed the opinion that this powerfully corrosive agent, if once efficiently applied, would frequently stay the spread of the inflammation; but he advised that it should not be repeated. One must be alive to the fact that mucous membranes with which the acid is brought into contact show whitish patches for at least twenty-four hours afterward; for I have myself seen Bretonneau's practice continued day after day when no disease of the fauces was left, except that which was produced by the application. The white marks produced by nitrate of silver—either the solid caustic or solutions in distilled water up to the strength of gr. xx to ʒj—do not last so long. But of late years the practice of applying these powerful agents to the throat in diphtheria has been almost abandoned in all parts of Europe. The fact has become recognized that the local affection (or such of it as is within reach) never constitutes the really dangerous part of the disease. At the present time disinfectants are much employed—for the purpose of destroying the micrococci, by those who hold the parasitic theory of the disease, to lessen putrefaction, and purify the surface of the affected parts, by those who reject that theory. Diluted chlorine water is said to be the most useful of such agents; and this accords with the results obtained experimentally by Oertel, who added various disinfectants to liquids in which diphtheritic membranes had been repeatedly washed, and afterward tested their powers of setting up a putrefactive process in "Pasteur's fluid." The other substances which he found most effective were alcohol, solution of permanganate of potash (gr. iss–gr. iiss ad ʒj), and solution of carbolic acid (gr. iiss ad ʒj). He recommends that one or other of them should be used as a gargle, once or twice, at least, in every hour.

The local application on which Dr. Squire lays great stress is the weaker solution of perchloride of iron; he adds a little glycerin to it, and paints the fauces over with a camel's hair brush two or three times a day. He also recommends for a gargle lime water, which, I believe, was first suggested by Küchenmeister, on account of its remarkable power of dissolving diphtheritic membranes. If the patient is very young, the fauces may be syringed out with equal parts of lime water and milk. On theoretical grounds, Oertel makes a great point of setting up a suppurative action on the surface of the affected parts as early as possible; and he therefore advises that the vapor of boiling water should be inhaled for a quarter of an hour at a time, twice every hour, and would even reduce the patient's time for sleep to three or four hours, in order to keep up this treatment as fully as possible. He also speaks highly of solutions of common salt and of chlorate of potass, for inhalation; but for this a spray apparatus is requisite. Most English physicians, however, allow patients suffering from diphtheria to suck small pieces of ice, which often give great relief.

When the disease has its seat in the nasal cavities, these parts should be frequently cleansed with disinfectants (especially the dilute solutions of the permanganate of potash or of carbolic acid), which may be injected with a syringe every two hours, or even oftener. If the patient is old enough "Weber's douche" should be used. This consists of a flexible tube, with a nozzle fitting tightly into one nostril, through which a stream of fluid is made to run by syphon action from a vessel at a higher level. The patient is directed to incline his head forward, and to breathe quickly with his mouth widely opened. The soft palate is thus drawn up against the wall of the pharynx, so that the fluid, instead of escaping into the fauces, passes round and runs out of the opposite nostril into a basin placed under the chin.

A solution of common salt may be employed, or lime water, or a weak lukewarm infusion of camomile, as advised by Oertel.

When diphtheria attacks the larynx the treatment must be exactly such as would be employed in the more advanced stage of croup. An emetic of ipecacuanha or of sulphate of copper should be given, and if a good result is obtained it may be repeated after an interval of some hours. Oertel recommends that a camel's-hair pencil attached to a suitably curved handle should be passed down into the larynx, and moved about so as to entangle and bring away any bits of false membrane that may be loose. This practice, he says, proved successful in three out of eight cases in which he employed it, the patients being respectively five, six, and eleven years old. The membranous pieces did not always come out with the instrument, they were sometimes ejected afterward, by means of a short, choking cough. He also advocates the inhalation of lime water from a spray apparatus, but (as he remarks) very little can be expected from such a procedure unless the patient spends nearly half his time at it, and most physicians will be of opinion that to advise this is to lay too heavy a tax upon his strength. Sooner or later the question of tracheotomy has to be taken into consideration; many circumstances render it a desperate resource in diphtheria. Even if the air passages can be freed from the obstruction which immediately threatens the patient's life, there is always the fear that the general symptoms may increase in intensity and prove fatal; and, moreover, the edges of the incision are very likely to become coated with a diphtheritic layer. I may here remark that I do not take the mere fact that the wound looks unhealthy, and becomes coated with an ash-colored layer, to be proof that it has been exposed to the action of the specific poison of diphtheria.

For diphtheria affecting the cutaneous surface the local application of calomel is said by Trousseau to be useful; it should be dusted over the affected parts. I may take this opportunity of insisting on the rule that, on account of the liability to the subsequent development of false membranes at spots to which blisters are applied, they should never be ordered for a patient suffering under any form of the disease.

Diphtheritic paralysis generally subsides within three or four months, and sometimes still earlier; a case under Donders is mentioned as rare, in which recovery did not take place until ten months had elapsed. Some years ago, however, a boy came to me as an out-patient who had been in the hospital four years previously for this affection; he was still unable to swallow perfectly, and fluids still sometimes returned through his nose if he tried to drink rapidly. This paralysis is very rarely fatal. In 1876, a little girl died unexpectedly a day or two after her admission into Guy's Hospital for partial paraplegia of obscure origin. I only saw the child once; the house physician drew my attention to the odd way in which she would roll about in walking across from one bed to another, so that she fell unless supported by the nurse. After her death we learned that she had recently had diphtheria, a cause for her paralysis which had not occurred to my mind. I then regretted that she had not been kept strictly at rest and in bed.

Iron and other tonic medicines are indicated in cases of this kind, and often seem to be very useful. Oertel objects to the administration of nux vomica or strychnia, and he does not recommend that galvanism or faradization should be employed, except at a late period. During convalescence sea-water baths and sulphur baths are said to be serviceable. Sometimes the patient is left permanently with slightly impaired power of certain muscles.

CHOLERA.

HISTORY AND NAME—ONSET AND EARLY SYMPTOMS—STAGE OF COLLAPSE—STAGE OF REACTION: THE URINE AT THIS STAGE—SO-CALLED "CHOLERA TYPHOID"—CHOLERINE—COMPLICATIONS—POST-MORTEM CONDITIONS—PATHOLOGY—ÆTIOLOGY—THEORY OF CONTAGION—PROGNOSIS—TREATMENT.

In the winter of 1817–18 there appeared in the camp of the Marquis of Hastings, then engaged in the Mahratta war on the banks of the Sind, a very fatal malady, attended with vomiting and purging. It is now believed to have prevailed in India, from time to time, during the previous century, and, indeed, as far back as history goes; but it was then taken for a new disease, and created the utmost terror. During the next few years it spread over a large part of Asia, through many of the countries of Europe, and even to America. It had, of course, to be named, but the designation which was chosen for it, Cholera, was extremely inappropriate, having been used since the days of Hippocrates for a complaint attended with a flux of bile (χολή), whereas the Indian disease was marked by an absence of bile in the matters vomited or discharged from the bowels. For a time, therefore, there was much confusion, and the epithets "Asiatic," "epidemic," "malignant," were commonly applied to the new malady, by way of distinction from the old one. But of late it has become more and more usual to speak of the former, which is certainly a specific disease, as the only *cholera*, and to classify as diarrhœa or gastro-intestinal catarrh cases which are now and then seen, and which appear to be merely exaggerated forms of these affections, although their symptoms are more or less "choleraic" in character.

Course.—The onset of Cholera may be either gradual or sudden. After exposure to the exciting cause of the disease there is a period of incubation, which is believed to be generally two or three days, but sometimes not more than twelve or twenty-four hours. Dr. Goodeve, in "Reynolds' System of Medicine," cites an instance, recorded by Dr. Barry, in which a detachment of sepoys, on their march from one place free from cholera to another, passed through a village where it was raging; one of the sepoys was attacked after forty hours, and fresh cases appeared subsequently. When the disease sets in gradually the earliest symptom is generally diarrhœa, which is often called "premonitory," and which may be attended with griping pain and with a sense of exhaustion. In some cases malaise, depression of spirits, headache, vertigo, noises in the ears, oppression at the epigastrium, are present at this period. At the London Hospital, in 1866, a nurse was doing her work, as usual, when she was attacked with "singing in her ears" and nausea, so that she lay down hoping that her symptoms would pass off, but they were quickly succeeded by those of cholera. Another patient there complained not only of noise in the head, but of being pained by the sound of his own voice; and another could not rest, and kept shifting his head from the top to the bottom of his bed every few minutes, trying to escape

from the ever-pursuing noise. The countenance of a patient during the *premonitory stage* is often pallid, anxious, and sorrowful. Annesley cites a case in which the approach of cholera was suspected mainly from the aspect of the patient nine hours before the characteristic symptoms appeared. The duration of the premonitory stage varies from a few hours to two or three days. In many instances it is altogether absent, and then the disease develops itself with the most startling suddenness. It very often—according to Lebert, who writes on the disease in Ziemssen's "Handbuch,"—in more than half the cases, begins in the early morning, perhaps waking the patient up from sleep. It then sets in with violent purging, the contents of the bowels are rapidly swept out, in a fluid form, and the discharges soon become almost colorless, like whey, or like water in which rice has been boiled, so that they are commonly spoken of as "rice-water evacuations." On standing, this fluid deposits a loose, whitish-gray material, which consists of mucous flocculi, containing numerous leucocytes, and of immense numbers of granules, including many bacteria. At one time it was thought that columnar epithelial cells were present in abundance, but this is now known not to be the case in rice-water fluid discharges during life, although that which is found in the intestines after death is full of them. The specific gravity of the liquid is from 1.006 to 1.013; it has a neutral or slightly alkaline reaction, and chiefly contains chloride of sodium, with a very small quantity of albumen. So profuse is the flow that Dr. Goodeve speaks of a patient as almost filling the pan of a night stool in two or three hours; he sometimes voids several pints, or even quarts. Dr. Goodeve remarks that when all that is voided is collected in the same vessel, the bile contained in what was first passed may give it a yellowish color. Sometimes the tint is pinkish, from the admixture of blood. There is often no pain whatever in the bowels, but some patients complain of more or less griping. After an interval, or even at the very commencement of the attack, vomiting sets in; the fluid rejected from the stomach (unless mixed with food) is pale and watery, being, in fact, identical with the rice-water liquid; nay, it may even have a still lower specific gravity of 1.002 to 1.005, in which case there is probably a large admixture of water ingested during the attack. It is often poured out of the mouth with great force, and quite suddenly. Another early symptom is severe cramping pain in the muscles of the feet, in the calves of the legs, and sometimes in the thighs, hands, chest, or abdomen. This usually comes on at intervals, and lasts for a few minutes at a time. It is of a most agonizing character, causing the patient to shriek out and to start up from the bed on which he is lying. Lebert says that in a third of the cases observed in the epidemic at Zürich, in 1855, cramps were absent.

The Stage of Collapse.—These symptoms are followed, more or less rapidly, by the development of a very remarkable condition, which is known as "cholera collapse." By Goodeve and others it is described under the name of the "algide stage" of the disease. It commonly appears within six or seven hours of the commencement of the purging, and often earlier still. Indeed, in some exceptional cases the patient actually dies collapsed before there has been any evacuation, the rice water fluid being found accumulated in the bowels after death. The essential sign of this state is a failure of the circulation, beginning at the periphery, but afterward affecting parts less distant from the heart. The pulse at the wrist becomes more and more feeble and thread-like, until it is altogether imperceptible; even the brachial artery may no longer be felt beating; on auscultation, according to Lebert, the second sound of the heart may be inaudible, while the first sound is still heard. One result is that the surface of the body becomes cold, livid, and shrunken. The hands feel like ice, and look

shriveled, as though they had been long soaked in water. The features have a leaden hue, the eyeballs are deeply buried in their sockets, the nose is pointed, the tongue feels cold, even the breath may be entirely devoid of warmth. The skin is often covered with a profuse sweat, which must still further deprive it of heat. According to Goodeve a thermometer placed in the mouth indicates from 79° to 88°. In the axilla, however, the temperature is seldom below 93° or 94°, as was shown by careful observations made at the London Hospital, in 1866, by Mr. F. M. Mackenzie. And in the rectum, or in the vagina, the temperature rises through the period of collapse, reaching 100°, or 102°, or even 104.4°. Mr. Simon, in discussing these facts in his "Ninth Report," infers that the choleraic affection of the bowels is a "heat-making" process. But I see no reason for doubting that the heat is generated in the muscles and in the glandular organs, just as it is under ordinary circumstances; Mr. Mackenzie, indeed, observed that a severe attack of cramp raised the axillary temperature as much as 2° F. The only question seems to be whether the elevated temperature in the interior of the body is due to a mere accumulation of heat from deficient loss, or whether there is a positive shifting upward of the point to which heat regulation is set, as in true pyrexia (see p. 56). It is a strong argument against the latter view, that during the reaction, as we shall presently see, when the peripheral circulation becomes restored, the internal temperature falls and is actually below normal. And I think that cholera collapse must be regarded as essentially different from the initial rigors of acute specific or inflammatory diseases. One distinction, on which Hutchinson has insisted ("*London Hosp. Rep.*," vol. iii), and which is of much interest, is, that in collapse the pupils are of natural size, whereas in rigor they are widely dilated; but I am not sure that his conclusion is necessarily justified—that there is no arterial or vasomotor spasm in the one condition, as there doubtless is in the other.

The breathing during collapse is increased in frequency, being at the rate of 24, 30, or even 40 in the minute; and there is often a sensation of dyspnœa. The heart's action, however, is but little accelerated, remaining at about 100. There is great muscular weakness. The voice becomes feeble and whispering, or it may be so completely extinguished that nothing but a movement of the lips occurs when an attempt is made to speak. Lebert thinks that this is due to dryness of the vocal cords, as well as to relaxation of their muscles. As a rule, the patient soon becomes unable to rise from the recumbent position; but sometimes he can sit up, or even walk across the room, very shortly before his death. There is usually great restlessness, the limbs being abruptly tossed and jerked about. The mental state is generally, from the first, one of indifference rather than of anxiety; and in some cases it is only when the cramps cause the sufferer to cry out that he seems to be conscious of his condition. But he can easily be roused to understand what is said to him, and may answer quite rationally. Coma sets in only toward the very last, if at all. It is to be noted that when collapse has developed itself in a marked form, the purging ceases altogether, or becomes greatly diminished. Probably what is now passed had been poured out into the bowel at an earlier period; Goodeve describes it as often containing gelatinous, mucus-like masses. The stomach, however, still remains very irritable. There is the most intense thirst; the patient craves for water, and drinks it with eagerness, only to vomit immediately afterward. Another symptom which may cause much distress is a sense of burning heat at the epigastrium, often accompanied by great tenderness, and sometimes by hiccup. The abdomen is commonly retracted. Lastly, one of the most marked phenomena of collapse is suppression of urine. This seems often to be present from the earliest period of the disease; but

Lebert says that patients may for a time micturate when the bowels act. There is no doubt that, as was first pointed out by Griesinger, the arrest of the renal secretion in cholera is a result of the defective flow of blood through the kidneys. Hermann and Cohnheim have each shown that precisely the same thing occurs in animals when the renal arteries are compressed or ligatured.

Fatal Event.—Cholera collapse often leads directly to a fatal termination, which usually takes place between twelve and twenty-four hours after the commencement of the attack, but sometimes earlier, and sometimes during the second day. Lebert mentions that before death the eyes may become dry and the corneæ slightly opaque. On the other hand the internal temperature has often been found to rise after life has become extinct. Another phenomenon sometimes observed in the dead body is the occurrence of spasmodic twitchings and quiverings of the muscles within the first half hour after death. The limbs may actually move; in a case at the London Hospital, in 1866, the elbow became raised about three inches above the level of the chest, across which it was lying.

Reaction.—Not infrequently, even when collapse has been present in an extreme form, it is nevertheless recovered from. In that case *reaction* is said to occur. This usually begins to develop itself at the end of twenty-four or forty-eight hours. Goodeve remarks that the subsidence of restlessness and jactitation often constitutes a sign of very favorable import. The patient dozes quietly, with easy respiration; then a flickering pulse at the wrist is detected, which gradually becomes more distinct, the superficial veins on the back of the hands begin to fill, the surface is felt to be slightly less cold; the features look less sunken and acquire a better color. As a rule, the improvement occurs slowly, and step by step. But the older Indian physicians spoke of cases in which it was very rapid, as, for example, that of a man "standing at his door on Wednesday, who on Monday was in perfect collapse."

According to Mr. F. M. Mackenzie, the temperature during reaction, when the pulse has fairly recovered, is usually about 97° in the rectum or the vagina, and 96° in the axilla. But in some cases at the London Hospital, in 1866, it was observed that certain parts of the surface conveyed to the hand a sensation of burning, pungent heat, the degree of which, however, was not determined by the thermometer. Another point noticed at the same time was that the pulse often fell to 54, or even to 45, in patients who were doing perfectly well. Lebert, on the other hand, speaks of the pulse as remaining at about 100. It often becomes full and bounding, and may be dicrotic. The patient's aspect is peculiar. The cheeks usually present sharply defined patches of dusky redness; the conjunctivæ are deeply injected, the eyes half closed with the corneæ turned upward, the expression heavy and vacant.

In some cases slight pyrexia occurs at this stage, and it is attended with the development of a bright crimson or scarlet *rash*, which is commonly spoken of as a roseola, though it may rather have the character of an urticaria. Sometimes it resembles a scarlatinal eruption pretty closely. Mr. Mackenzie found, in 1866, that it was always accompanied by a rise of temperature, both external and internal. In one instance the former was 101.6°; the latter 102.4°. The backs of the hands and the forearms are favorite seats of it, but it may also cover the trunk, and may even be seen on the face. In the museum of Guy's Hospital we have excellent models of it, made during the epidemic of 1854. It seldom comes out until a week or ten days after the commencement of the attack, and in one case recorded by Wilks it did not appear until the seventeenth day. It lasts two or three days, and may be followed by desquamation. It is more often observed in

young patients than in old, and is particularly well marked in children. As a rule the cases in which it occurs end in recovery.

The period of reaction, however, is by no means free from danger. Sometimes a relapse occurs; purging, vomiting, and exhaustion set in again, and end fatally. In other cases the reaction is said to be *imperfect*. The pulse, after improving up to a certain point, remains weak, and the surface of the body continues to be colder than natural. The bowels are still relaxed, and the evacuations are watery, although more or less colored with bile. There is no appetite, and the patient is depressed and drowsy. Sometimes the most prominent symptom is irritability of stomach, attended with thirst and burning sensations along the œsophagus and at the epigastrium. Goodeve also mentions sleeplessness, lasting for two or three days or more, and causing great discomfort to the patient.

Urine.—But at this stage of cholera the most important condition is that of the kidneys. We have seen that during collapse there is generally complete suppression of urine; if any is secreted it is apt to be, even at that period, albuminous. When reaction sets in, the suppression often continues for several hours, or even for two, three, or four days. According to Buhl, if it lasts up to the sixth day the case is hopeless. Sometimes, however, when the renal function has really been re-established, the patient, nevertheless, fails to void urine, which is retained in the bladder. The hypogastric region must, therefore, always be carefully examined, and, if necessary, a catheter may be passed. The instrument should be lubricated with carbolic oil, for cystitis and pyelitis have often been observed in cases fatal at an advanced stage. In a female patient who died in Guy's Hospital during the epidemic of 1866 there was suppurative nephritis, which appeared to have arisen by extension from the vesical mucous membrane.

The urine which is first secreted during the reaction stage of cholera is described by Goodeve as high colored, acid, and possessed of a strong animal smell. As to its specific gravity there are some discrepancies in the recorded observations. At the London Hospital, in 1866, it was found to range from 1.005 to 1.017, the average being 1.006. Wyss, however (*Arch. d. Heilk.*, 1868), found that the density of the first urine passed was from 1.012 to 1.033, the average being 1.020. It was only at a later period, when the quantity was more abundant, that it fell to 1.010, or even lower. The amount of urea and of salts is much diminished, but albumen is generally present, and sometimes blood, and there are usually hyaline casts in immense numbers. Lebert says that the occurrence of albuminuria at this stage is constant, but I do not think that such a statement accords with the experience of English observers. At the London Hospital, in 1866, albumen was found "in about half the cases examined." As a rule, the urine continues to be coagulable during from two to seven days. Until recently the renal affection attended with albuminuria in cholera was commonly regarded as a nephritis, and as comparable with the acute renal affection that follows scarlet fever or diphtheria. But Bartels, in Ziemssen's "Handbuch," pointed out that it might be fairly regarded as the result of the ischæmia which exists during the stage of collapse, and this view has since been upheld by Cohnheim. In cases fatal before reaction has set in, the kidneys are found small, tough, and of a dark brown or slightly livid color. At a later stage they are much enlarged—Dr. Moxon speaks of them as weighing up to fifteen or sixteen ounces—soft, grayish-white or yellowish. The epithelium very early becomes cloudy, opaque, or fatty; it has lately been suggested that this change in it is analogous to the "coagulation-necrosis" which Litten has found to occur in the renal epithelium of rabbits after arrest of the arterial blood current through the kidneys. The Malpighian tufts and their capsules are said to present no morbid appearances

in cases of cholera. Sometimes infarcts are found in the kidneys, being, doubtless, results of the ischæmia of the stage of collapse. The renal affection of cholera appears never to form the starting point of chronic Bright's disease. This fact accords with the modern opinion that it is theoretically to be distinguished from the various forms of nephritis, but it is to be observed that the same may probably be also said of the renal affection of diphtheria, the inflammatory nature of which is indisputable.

Cholera Typhoid.—The reaction stage of cholera often presents a grave complication, which is known as "cholera-typhoid." This sets in about five or six days after the commencement of the attack. It is attended with great prostration, headache, giddiness, and stupor. The face is at first flushed, but afterward becomes pale. The temperature does not rise more than 2° or 3°. The tongue becomes dry. The pulse, which is sometimes at first very slow, becomes weak and small. There is often a remarkable rigidity of the muscles, so that the patient strongly resists any attempt to open his mouth, to separate his eyelids, or to straighten his elbow. Convulsions are seldom observed. Death by coma usually occurs in about forty-eight hours, but sometimes the typhoid state continues for several days, and sometimes ends in recovery.

Following Frerichs, most writers are of opinion that this complication of cholera is due to uræmia. Urea has, in fact, been found in the blood in cases of cholera-typhoid, by several observers. Buhl estimated the quantity of it in one instance at .2 per cent. In some instances, too, there has been observed upon the face and neck an efflorescence of urea in a crystalline form, this having been excreted by the sweat-glands. But Goodeve has pointed out that in some cases a consecutive fever, attended with typhoid symptoms, occurs independently of uræmia, or at least while urine free from albumen is being secreted. Both Bartels and E. Wagner drew attention to the same fact, and attributed the pyrexia in many cases to local inflammation of the intestines or other internal organs.

Slighter Forms.—I have still to mention certain minor forms of cholera, cases of which are certainly not uncommon, but of which the frequency in relation to that of the typical disease cannot be positively stated, because they are often allowed to run their course without being brought under medical observation. One of them is commonly called "choleraic diarrhœa." It is identical with the "premonitory diarrhœa" already described, except that it subsides after a time and does not pass on into cholera. It often begins suddenly, after a chill, or after the patient has eaten unwholesome food. There are three or four or more evacuations of yellow fecal matter daily, with some pain, and perhaps with slight cramps. A more severe form is sometimes termed by foreign physicians "cholerine;" it is attended with vomiting, with cramps, with a transitory disappearance of bile from the stools, and even with some degree of coldness of the limbs. Lebert says that he has seen it followed by pronounced typhoid symptoms.

Complications.—The convalescence from cholera may be accompanied by certain complications, which require brief mention. One of them is an affection of the lower segment of the cornea, generally of each eye. This doubtless results from the part having been exposed and irritated during the stage of collapse. It now, four five days after reaction, becomes hazy, or covered with a layer of opaque lymph; and ulceration presently occurs in it. Sometimes perforation takes place, but generally—if the patient should recover, which seldom happens—the eye is preserved, with little or no disfigurement. In other cases, during the second or the third week the parotid glands swell or suppurate. This seems to indicate weakness, and it also interferes with swallowing food, so that death often follows. Sometimes bronchitis, pneumonia, or pleurisy sets in; or diphtheritic inflammation

of the fauces, or the bladder, or of the sexual organs, or gangrene of the scrotum and of the penis, or even of the nose. Bed sores, too, are often a source of danger at this period of the disease.

Morbid Anatomy.—This throws but little light on the pathology of cholera. The bowels commonly contain more or less of the rice-water fluid; this microscopically is found to contain an immense quantity of cylinder epithelium, not only loose cells, but coherent masses of considerable size, forming complete casts of the villi. If there is no liquid, the cast-off epithelium forms a soft, creamy pulp. At one time the opinion was held that shedding of the intestinal epithelium was the fundamental lesion of cholera; but it is now known to be of post-mortem occurrence. For, not only are the cells absent from the evacuations passed during life, but (as Cohnheim observes) denudation of the mucous membrane, if it took place before death, could not but be followed by severe inflammatory changes in it. All that is found is a slightly injected, swollen, and œdematous condition of the intestinal coats, with perhaps some ecchymosis of the valvulæ conniventes. The lymph-follicles in the mucous membrane are also, in many cases, enlarged, and Lebert says that there may be slight ulceration of Peyer's glands. In a case that occurred in Guy's Hospital, in 1854, the lining of the rectum showed a patch which was œdematous and sloughing, with suppuration in the submucous tissue. The peritoneal coat may be reddened and sticky, and in 1866 Dr. Moxon found, in one case, in which the collapse had been unusually protracted, that a tenacious viscid material like spider web lay between the coils of intestine, and could be drawn out into long filaments by separating one coil from another. Other points on which he insists are the want of fecal odor and of the blackening produced by sulphuretted hydrogen, and the absence of gas in the intestine, so that the coils lie in a flabby, compact mass in front of the spine. The mesenteric glands are sometimes slightly swollen.

The blood which is found in the heart and in the great vessels is remarkably viscid and tarry; and, whenever venesection has been practiced during life, the same peculiarities have been seen. This state of the blood, however, is not special to cholera; it was well marked in a patient who died in Guy's Hospital in 1871, of acute ulceration of the intestine. It is, no doubt, a consequence of the drain of fluid from the body; and another result, which has been noted by more than one observer, is the rapid disappearance of dropsical exudation in the course of a few hours when a patient suffering under dropsy happens to become attacked with cholera. In all probability the blood change is the cause of the formation during life of ecchymoses, which at the autopsy are often found scattered over the outer surface of the heart, upon the pulmonary pleura, in the pia mater, and elsewhere. It also renders the tissues peculiarly dry. Cohnheim remarks that the serous cavities in the dead body of a cholera patient have a soapy feel, which is not likely to be forgotten by any one who has ever placed his hand in them; and the spleen, the lungs, the liver, and the other organs are tough and leathery. The peculiarly shrunken appearance of the lungs is attributed by Dr. Moxon, no doubt correctly, to the dry, empty state of the bronchia, which allows the elasticity of the organs to drive the air out of them more completely than usual after the chest is opened. On the other hand, when death has occurred during reaction, the finer tubes are often found full of pus, and parts of the lungs may be œdematous, or even in a state of broncho-pneumonia.

Pathology.—Hitherto there has been the greatest uncertainty as to the origin of the various symptoms of cholera, and as to their relations to one another. The characters of the rice-water liquid are so unlike those of any inflammatory exudation which is known to occur, in whatever part of the body, that we cannot possibly suppose it to be of such a nature. An

experiment, however, originally made by Moreau has, perhaps, suggested the clue to its real significance. Physiologists had previously learned from Thiry how to isolate a portion of the intestine, so as to obtain from it an unmixed succus entericus in small quantity. Now Moreau has found that when an isolated loop of intestine has all the nerves in its mesentery cut through, it becomes filled, within a few hours at latest, with an abundant secretion of a thin, alkaline, yellowish fluid, which is of very low specific gravity, which contains numerous mucous flocculi, but in which there is only a very small quantity of albumen, while the chief salts in it are those of soda.* There seems, in fact, to be a paralytic hypersecretion of the succus entericus; and, according to Masloff, it possesses the property of converting starch into sugar. Now Kühne has shown that the rice-water liquid of cholera likewise contains a ferment having this property, besides corresponding so closely with the other liquid in all other respects. The inference, therefore, seems justifiable, as Cohnheim points out, that the cholera liquid itself is nothing else than the secretion of the intestinal glands, enormously increased in amount. Whether its formation should be regarded as a sign of paralysis of intestinal nerves is left, by Cohnheim, an open question. In proof that the muscular coats of the bowel are not always paralyzed in cholera, he cites the fact that invaginations are often found, of the kind which are known to be of post-mortem origin. But he insists that the so-called "cholera sicca," in which rice-water liquid is found in the intestine after death, though none had been voided during life, owes its peculiarities to an early exhaustion of the excitability of the intestinal walls; and he refers to the same cause the frequent cessation of vomiting and purging as collapse becomes developed.

It cannot be doubted that the acceptance of this view with regard to the nature of the rice-water liquid will tend powerfully to support the doctrine, already held by many observers, that the primary action of the cholera virus is upon the solar plexus, rather than upon the bowel itself. Cohnheim, indeed, agrees with those who think that the phenomena of collapse are adequately explained by ascribing them to the drain of fluid from the body, and to the viscid, inspissated condition of the blood which we have seen to result from it. This opinion was opposed several years ago by Dr. George Johnson, one of whose arguments was that no relation could be traced between the degree of severity of the collapse in a given case, and the amount of fluid discharged from the stomach and from the intestine. But I think that a much stronger argument has since been advanced by Mr. Sedgwick, namely, that cholera collapse closely resembles that which follows cases of perforation of the stomach, of intestinal obstruction, and of other abdominal diseases in which no purging occurs. At Guy's Hospital, during one of the London epidemics, a man actually died of internal strangulation of the bowel, who was supposed during life to be suffering from the so-called "cholera sicca." I imagine, however, that this mistake would have been avoided if the duration of the symptoms had been taken into account. It must, therefore, be supposed that collapse is the result of an action on the part of the great ganglia of the abdominal sympathetic. This action, however, is by no means identical with that which causes syncope. Cholera patients have no sensation of faintness; and even when they have no pulse at the wrist, they may be able to sit up, or to walk about, without sudden failure of the heart's action. On the other

*[Moreau's results have since been confirmed and extended. See the Reports of a Committee appointed by the British Association, published in the "*Transactions*" for 1874, 1875, and 1876; also papers by Dr. M. Hay, in the "*Journal of Anatomy and Physiology*" (vol. xvi, p. 243 to vol. xvii, p. 441), and by Dr. Brunton, in the "*Practitioner*" for November, 1884, *et seq.*—Ed.]

hand, the collapse of cholera is essentially different from rigor. May it not be that, whereas in syncope an influence from the solar plexus is thrown solely upon the heart, and in rigor solely upon the arterial system, in collapse it is exerted evenly upon them both, there being spasm of the peripheral arteries, while the force of the heart is lowered just to the point at which it can effectually propel the blood, diminished as it is in volume, through the deeper arteries? The suppression of urine which occurs in collapse is a part of the general state, since it is observed likewise in cases of acute intestinal obstruction, of perforating ulcer of the stomach, and of arsenical poisoning. Thus, I think that there can be no doubt that Dr. Johnson was in error when he endeavored to trace all the phenomena of cholera collapse to obstruction of the flow of blood through the pulmonary capillaries. And although the high authority of Parkes can be cited in proof of the fact that the lungs weigh much less than usual in cases of cholera, this may be sufficiently accounted for by the diminished volume of the blood generally, since the organs in question contain so little solid material that their weight, so long as they retain their spongy character, must be mainly that of fluid in their vessels or in their interstices. During the epidemic of 1866 I, like my colleague, Dr. Moxon, failed altogether to observe that the branches of the pulmonary artery and the right side of the heart were distended or gorged with blood when death had occurred even at an early stage. It is true that cholera is attended with a lividity which is wanting when collapse is due to other causes. But the altered constitution of the blood may be fairly supposed to interfere with its due aëration, and the amount of carbonic acid exhaled from the lungs has been shown to be really much reduced. So, again, absorption from the alimentary canal is retarded, although not entirely annulled. Magendie is said to have found that when camphor was injected into the rectum, five minutes elapsed before its odor could be detected in the breath, instead of only one minute, as in health; and Lebert states that belladonna fails to dilate the pupils when administered by the mouth, though not when injected into the blood. It is, however, a very curious circumstance that in women who are suckling children the secretion of milk continues during collapse, even to an extent which is troublesome. The menstrual flow also goes on unchecked; it may actually set in before reaction is established, if the proper time has come around for its appearance. A sanguineous muco-purulent discharge from the vagina also frequently appears, independently of the catamenia. And it must, I think, be admitted that the mental and the bodily vigor are often maintained throughout the whole of an attack of cholera in such a way as to show that the circulation still remains very active in the brain and also in the muscles. When, however, the disease seizes upon a pregnant woman, the fœtus seems always to die; if the period of gestation is early, abortion takes place; if late, the mother often dies undelivered. In this case it seems to be useless to perform the operation of Cæsarean section, even immediately after her death.

The facts which have been already stated as to the course of the external and the internal temperature, respectively, during the reactive stage, seem to show, as I have already remarked, that there is no true fever, such as might be supposed to have for its aim the destruction of specific organisms in the blood. The reaction seems to be what its name implies, and analogous to the hot, swollen condition observed in a part when its vessels are allowed to fill with blood, after having been kept empty; as, for example, in Cohnheim's experiments on the ears of animals.

Ætiology.—With regard to the causes of cholera much has been learned in the course of the last half century, although many points still remain obscure.

In the first place, we may take it as established that the diffusion of the disease over other parts of the world from India is the result of human intercourse.

When, in 1823, and the following years, it *first* entered Russia and spread through North Europe in a northwesterly direction, its resemblance in this respect to influenza led many physicians to think that, like that disease, it was caused by some mysterious atmospheric or telluric agent. But its progress was, in reality, far too slow and halting to render such a view probable. Prussia was reached by cholera in 1831; in October of that year it passed from Hamburg to Sunderland, and entered London in January, 1832. Shortly afterward it invaded France, *viâ* Calais, and it also spread across the Atlantic to Canada and the United States. In 1833 it appeared in Portugal, passed in an easterly direction through South Europe, and became for the time extinct, in 1837. In fact, one can hardly doubt that its diffusion from India to the west rather than to the east depended simply upon the circumstance that communication and intercourse from town to town and from country to country are so much more free in the one direction than in the other. The *second* epidemic, which had begun in India in 1840, extended to Europe in a manner very like the first, reaching London direct from Hamburg, in 1848, and continuing to prevail in England during the following summer. The *third* affected this country in 1853–4. The *fourth*, which occurred among us, in 1866, differed from the others in having entered Europe from Alexandria; it had been carried from India to Arabia and thence to Egypt, partly by coast trading vessels, but in great part also by Mohammedan pilgrims to and from Mecca. A detailed account of all the circumstances connected with this epidemic, written by Mr. Netten Radcliffe, may be found in a supplement to Mr. Simon's "Report" for 1874.

One of the most remarkable circumstances with regard to cholera is that, although it has spread to almost every part of the world, and has sometimes prevailed under widely different thermometric and other conditions, it seems to be capable of establishing itself permanently in no country except India, and there only in a particular region. This of itself would suggest the probability that, in what may be termed the home of the disease, its mode of entrance into the human body may not be generally the same as in districts in which it is merely an occasional visitor. So that we need not, perhaps, be surprised that Indian physicians have been unable to trace the spread of cholera in certain ways, of which the occurrence has been clearly established in Europe; even if we do not regard their inability to confirm our observations as an example of the fact that far greater obscurity often hangs over the ætiology of a disease where it commonly prevails than where it is rarely seen.

However this may be, there can be no doubt whatever about the value of the European discoveries with regard to the mode of diffusion of cholera, which, in fact, place the disease by the side of enteric fever, as *miasmatic contagious* in nature. Like enteric fever, it but seldom passes directly from a sick person to those who nurse or visit him. A few instances have, indeed, been recorded, which look as though direct infection did sometimes occur. Thus, in a paper in the "*Edinburgh Medical Journal*," for 1838, the late Sir James Simpson related how certain mendicants brought cholera with them to Bathgate, on May 27th, 1832, and how one of the nurses who attended upon them was attacked and died on the 30th. He also cites the case of a woman who, having caught the disease at some iron works where it raged, and being afraid of being sent to the hospital, went, on May 12th, to a cottage where her mother lived, four or five miles off. On the 14th the mother fell ill, and died in a few hours. Dr. John Snow relates that a man who had been working at Chelsea, died at Streatham, of a bowel complaint which lasted

only a day or two. At that time no other deaths from cholera had been registered within two or three miles of Streatham, but the man's mother, who attended him, was taken ill on the very next day and quickly succumbed to the disease. But marked instances of this kind are very rare, and one may almost say that, as a rule, persons who have come very closely into contact with patients are scarcely, if at all, more likely than others to fall victims to cholera.

The explanation is believed to be that the contagion of cholera escapes from the body in the rice-water evacuations, but that it is not then in an active state, and becomes so only after an interval of from two to four or five days; in other words, the evacuations are supposed to be infective only at a certain stage in their decomposition, and not when they are fresh. The evidence in support of this view consists mainly of the results of experiments made by Thiersch in 1854, and repeated by Dr. Sanderson in 1866. These observers took liquids passed by patients during life, or taken from the intestines after death, and left them exposed to the air, in glass vessels. Every day pieces of filter-paper were carefully soaked, one with each liquid, and dried. The paper was then given to mice, a square inch to every animal. The consequence was that a considerable number of the mice died with an extremely low temperature and with disturbance of the functions of the intestinal canal, their excrement being soft and altered in color. Of the mice employed by Dr. Sanderson 11 per cent. suffered when the rice-water liquid had been one day old, 36 per cent. when it had been two days old, 100 per cent. when three days old, 71 per cent. when four days old, 40 per cent. when five days old; by the sixth day it became innocuous again. The morbid appearances found in the mice after death appeared to be consistent with the view that they were affected with cholera, and Mr. Simon afterward expressed the opinion that the value of the experiments in explanation of the facts of human infection was conclusive. No equally satisfactory results have hitherto been obtained with other kinds of animals, although some instances have been recorded of dogs being attacked by a fatal disease resembling cholera after having devoured matters vomited by cholera patients, and in 1853 Dr. Lindsay set up a similar disease in dogs by confining them in a small room, the floor of which was strewed with the excreta of such patients.

It may be thought that if a living organism, possessing specific properties, is present, in however immature a form, in rice-water discharges, it ought to be easily demonstrated with the aid of the microscope. But the alvine evacuations, even in health, contain various organisms in large numbers, and it is in reality by no means surprising that the search for special cholera microzymes has not yet met with success. Dr. William Budd, indeed, published figures of what he regarded as a cholera fungus, as far back as 1849; and many observers have since made statements more or less similar. In the "*Journal of Microscopical Science*" for 1881 Dr. Cunningham states that "monads," belonging either to *Cercomonas* or to *Trichomonas*, are found in excreta of patients suffering from cholera, in unusual numbers and in a peculiarly active state, but he adds that they occur to some extent under other conditions of disease, and even in health. He also finds amœbæ to be very abundant in certain cases of cholera; bacteria, he says, make up a very large part even of the normal fæces, at least in India.*

Passing on now to consider how the contagious principle of cholera gains access to the human body, we find the fact best ascertained to be its frequent entrance by means of drinking water. The late Dr. John Snow deserves special commemoration, not only as having been the first to uphold this

* [The discovery of the so-called "comma-bacillus" in the intestinal secretion of cholera patients, by Dr. Koch, was made after the above lines were written.—ED.]

view with regard to the ætiology of the disease, but also as having devoted infinite labor and pains to establish it. He collected instances occurring as far back as 1849, in which local outbreaks were traced more or less conclusively to the contamination of surface wells by sewage; one such occurred in Thomas Street, Horsleydown, another in Albion Terrace, Wandsworth, a third at Salford, a fourth at Ilford. A dramatic incident was the following: A gentleman who lived at Bath was the owner of some houses at Locksbrook, near that town. Cholera, which did not prevail at Bath, appeared at Locksbrook and became very fatal. The people complained of the water of the well attached to their houses, drainage from the cesspools having entered it. The owner went to the place, said he could smell nothing wrong with the water, was asked to taste it, and drank a glass of it. This was on a Wednesday; he returned home, was taken ill with the disease, and died on the Saturday.

But it was not until 1854 that the evidence of the communication of cholera by drinking water became irrefragable. Then occurred the celebrated outbreak in and around Broad Street, Golden Square, which is said to have destroyed in ten days more than five hundred persons living within a radius of 250 yards. This was traced by Dr. Snow to the water of a surface well, with a pump situated at the corner, between Cambridge Street and Broad Street. At least sixty-eight of the first eighty-three deaths occurred in persons who were actually known to have drunk the water in question; and it may be supposed that many took it without being aware that they did so, mixed with spirit in public houses, or with effervescing powder, under the name of sherbet. One very striking case was that of a lady at Hampstead, who was in the habit of sending every day for the Broad Street water; she was attacked by cholera and died. Her niece, too, being on a visit to her, drank some of the water, returned afterward to her own house at Islington, and died there. On the other hand, scarcely any of the inmates of the workhouse in Poland Street, where there was a separate well, were affected; and the disease also spared the men employed at a brewery in Broad Street, close to the pump. The water from the pump had a reputation for being very pure, and when analyzed, three days after the commencement of the outbreak, it was found to contain but little organic matter; but it had long been noticed to become offensive to taste and to smell when kept for two days; and Mr. Gould, the well-known ornithologist, who sent for some on the second day of the epidemic, did not drink it, because he observed that, although fresh from the pump, it had an offensive smell.

Observations on a still larger scale were also made, in 1854, in the south of London, over a district containing a population of at least 300,000 persons, and supplied with water partly by the Lambeth Water Company, conveyed from the Thames at Ditton, partly by the Southwark and Vauxhall Company, whose intake was from the same river at Battersea. What rendered these observations peculiarly conclusive was the fact that over a large area the mains of these two companies ran side by side, each feeding some houses and not others, according to the arbitrary fancies of owners or occupiers at a former time, when there had been an active competition for custom. Dr. Snow went, in detail, throughout the area in question, from street to street and from house to house, and he found that, during the first four weeks of the epidemic, out of three hundred and thirty-four fatal cases of cholera, there were two hundred and eighty-six in houses supplied by the Southwark and Vauxhall Company, twenty-two among persons who obtained water directly by dipping a pail into the Thames, but only fourteen in houses receiving a supply from the Lambeth Company. It is to be noted, however, that the number of houses

supplied by the Southwark and Vauxhall Company was greater than that of the houses supplied by the Lambeth Company, in the proportion of forty to twenty-six. As the epidemic continued, the difference in the cholera death-rate between the customers of the two companies became less, although it still remained very striking. Bethlem Hospital, the Queen's Bench Prison, and other institutions on the south side of the river which had deep wells, scarcely suffered at all from the disease.

In 1865 and in 1866 further evidence was collected bearing in the same direction. In 1866 the parts of the metropolis mainly affected were the eastern districts, and Mr. Netten Radcliffe, who investigated the matter for the Privy Council, found that there was a great preponderance of cases among persons whose water, supplied by the East London Water Company, had passed through the reservoirs at Old Ford, whereas comparatively few cases occurred among those who received the water of the same Company pumped directly from the filtering beds at Lea Bridge into the mains. The difference was far from being as conspicuous as in South London during 1854, but one must bear in mind that among the poorer classes very many eat and drink and pass their days at a distance from the houses in which they are said to reside. Moreover, at an early period of the epidemic a notice was issued, warning people not to drink any water which should not previously have been boiled, and it is worthy of notice that from the very week in which this appeared the epidemic began to decline. A point of particular interest is that, assuming the reservoirs at Old Ford to have been in some way concerned in causing the disease, one can plausibly account for the entrance of the cholera poison into their water; for, although this water was intended to have been all filtered at Lea Bridge, some of it was sometimes drawn directly from two other reservoirs which differed from the rest in being uncovered, and which communicated freely with the River Lea, by soakage. But this part of the river was, in fact, a canal with locks, and received a large quantity of sewage; so that it was little better than a cesspool. Now, shortly before the epidemic in East London began, a man and his wife, living in Priory Street, Bromley, near the banks of the Lea, had died of cholera, and their evacuations had entered the river about 600 yards below one of the open reservoirs.

In the previous year, 1865, there had occurred in an Essex village, Theydon Bois, a most remarkable local outbreak of cholera, which illustrates in a very striking manner how insidious its propagation may be. Between July and November the disease seems to have been several times introduced into Southampton by persons who arrived from the Mediterranean ports, where it was then prevailing. Whether it was carried from this town during August or September to Weymouth or Portland or Dorchester, or whether it reached either of these places in some other way, was never ascertained. But this is certain, that a gentleman and his wife, visiting these places (but not Southampton) in September, contracted, one or other of them, a diarrhœa which, in the case of the lady, developed into fatal cholera after her return to her home, at Theydon Bois, on September 25th. On the 30th, while she was still collapsed, one of her daughters was attacked and died in a few hours. The same night a serving-lad in the house was seized and barely escaped with his life. Altogether in a fortnight, eleven persons within a narrow circle, father, mother, grandmother, two daughters, son, doctor, foot-boy, maid, laborer, and countrywoman, fell ill, and only three of them recovered. Now, the drinking water of the house all came from a well beneath the scullery, and into that well there was habitual soakage from the water closet.

Such cases as those of Theydon Bois are of especial importance, because they are inconsistent with a view which at one time found many supporters,

that drinking impure water, instead of being the means of introducing the specific contagion of cholera into the body, acts merely as a predisposing cause of the disease, by rendering a person unduly susceptible to the action of the true exciting cause. Dr. Snow long ago pointed out that some of those who, in 1866, died of cholera in houses supplied by the Southwark and Vauxhall Water Company were young men and maid-servants who had come from the country only a few days before.

I must now briefly mention a view with regard to the mode of diffusion of cholera which has been put forward by certain German observers in rivalry with the "drinking-water theory." This is the so-called "soil-water" or "ground-water theory" of Pettenkofer. The observations on which it is based are such as cannot be described in detail within the limits of this work, but I believe that his fundamental proposition may be said to be that cholera never prevails epidemically where the soil is impermeable to water, or where the level of the soil water is not liable to fluctuations. He brings forward several instances in which districts seated upon hard rock escaped, while adjacent towns, built upon alluvium, suffered. Some apparent exceptions to the rule he accounts for more or less satisfactorily. Thus Gibraltar, as is well known, has shown no immunity, but, in visiting it, he found that the town really lies on a slope of red earth, containing more than 200 surface wells, So, again, Malta, in which the disease has prevailed severely, is built on a solid rock; but this, a sandstone, is so soft and so permeable by water that the Government Comptroller told Professor Pettenkofer that it was like a sponge saturated with all kinds of filth. On the other hand, Lyons has, during each of the European epidemics, remained free, or nearly so, from cholera, even when both Paris and Marseilles were ravaged by it; in 1865, twenty thousand persons are said to have flocked thither from Marseilles for safety. But the greater part of Lyons lies on a river alluvium, so that on Pettenkofer's theory one would have expected its inhabitants to be severely attacked. The explanation which he gives is that in this town, unlike most other towns, the level of the soil water is diminished by that of the rivers which flow through it, the banks of the Rhone and the Saone being, in fact, so porous that their streams may almost be said to run in part subterraneously beneath Lyons. In other words, the supposition is that Lyons escapes cholera either because there is too much surface water, or because its height is subject to scarcely any variation. But ingenious as these several statements are, one cannot help wondering whether it might not be possible, by a similar reasoning, to deprive of all force the very instances on which Pettenkofer himself relies.

In this country the ground-water theory of cholera has met with no acceptance, and I believe that observers have failed to find evidence of its applicability to those parts of India where the disease is endemic, and where such a doctrine appears *primâ facie* to have much to recommend it. But it is very curious that in the East London epidemic of 1866 one fact was elicited which is strongly in favor of Pettenkofer's views. In a school at Limehouse there were four hundred pauper children, not one of whom was attacked with cholera or with diarrhœa. Now, the house had its sole water supply from the Old Ford reservoirs, by which, as has been already stated, the disease was being spread, and the children at all times made free use of the water. A special investigation of the soil beneath this school brought to light the fact that it stood upon a thick layer of fine brick-earth, and not of gravel, as appeared to be the case with the streets immediately adjacent.

It must be left to the future to show whether this observation, and others like it, can be attributed to accident. For if the question were merely whether there is a greater liability to cholera on the part of

those who reside on porous soils, as compared with those who reside on non-porous soils, independently of any proof of actual exposure to the contagion of the disease—it would be sufficient to suppose that a permeable soil facilitates, in one way or another, the diffusion of the contagion, and favors its entrance into the human body. According to Pettenkofer, the condition of soil under which cholera is most apt to prevail is that which occurs when the level of the ground water has begun to fall after being high. This might, perhaps, cause noxious matters to be washed into wells which would at other times be free from them. But a still more likely cause for the development of an epidemic under such circumstances is that which has been alluded to at p. 46, namely, the desiccation of contagious microzymes within the upper layers of the soil, and their subsequent diffusion in the air. I may remark that, in 1866, Parkes attributed an outbreak of cholera in Southampton mainly to an atmospheric effluvium disengaged from sewage which was constantly being pumped up by a steam engine, and sent frothing and churning down an open channel for some eight or nine feet.

Pettenkofer, however, considers that a complicated theory is needed to explain his facts. He supposes that when human intercourse conveys cholera from one place to another, that which is given off by a patient, or carried by an individual not himself susceptible, is, *not* the cholera-contagion, but what may be termed a "cholera germ," which is capable of generating the contagion when it meets with a certain "material substratum." An analogy which he suggests will make his notion readily intelligible: It is, he says, conceivable that if the yeast fungus were absent from certain countries, the inhabitants might prepare beverages from grapes or apples, or malt, and drink them without any liability to intoxication. But, if some one were to come from a country where the yeast fungus was found, and to bring its spores with him upon his clothes or in any other way, the hitherto harmless liquids might speedily produce an "epidemic of drunkenness." Yet the cause of the symptoms would not be the yeast fungus, but the alcohol of the fermentation set up by it. So Pettenkofer imagines that the "cholera-germ," which he symbolizes as x, acting under certain conditions upon the soil, which he terms y, generates a "cholera poison," which he designates z. He does not limit himself to the view that this poison is actually a chemical product, like alcohol, but suggests that it may probably consist of "secondary germs" developed during the putrefaction of organic matters contained in the soil.

Nägeli, who is a staunch supporter of Pettenkofer's observations, has more recently suggested what he terms a "diblastic theory," according to which the union of x and y is supposed to take place within the human body. He imagines that the soil gives off certain microzymes, which must be present in the body of every one who is to afford favorable conditions for the development of another set of microzymes derived from a pre-existing case of cholera. This, in other words, is to reduce the "soil" element in the ætiology of cholera to the rank of a "predisposing" cause. Nägeli appeals to cultivation experiments as affording frequent instances in which the growth of one kind of fungus in a medium induces in it changes that enable it subsequently to afford pabulum to another kind of fungus. Or, as he points out, it is possible that the contagious microzymes of cholera which enter the body are commonly too few to be able to overcome the resistance of the living structures, unless the latter has first been weakened by the previous operation of soil microzymes.

The advantage of this form of the hypothesis is that it admits of the direct contagiousness of cholera in certain cases, if the cholera microzymes happen to be introduced in very large numbers. For it is

undeniable that cholera sometimes seems to be propagated from individual to individual in succession in such a way as to render very improbable the hypothesis that any predisposing cause is *essential.* Take, for example, the following case recorded by Dr. Simpson, of New York, and cited by Dr. Snow. An agricultural laborer was attacked at Monkton Moor on December 28th, 1832, at a time when the disease was not known to be prevailing within thirty miles. His wife and some other persons who visited him were seized on the following day. Presently it was found that his sister had died of cholera a fortnight previously at Leeds, and that her wearing apparel had been sent to Monkton in a box which had been opened by him the evening before he fell ill. During the illness his mother, who lived in a healthy village five miles off, came to attend him, washed his linen, and after two days set off to return home, but was attacked on the road and had to be conveyed to her cottage. Not only did she die, but her husband and her daughter likewise. Only one other case occurred in the village. Pettenkofer himself, however, explains such facts as these by supposing that the "*z*"—the actual cholera poison—is sometimes carried from place to place on clothes or in some other way. A few instances have been recorded in which the disease has apparently been conveyed by articles of food. Dr. Snow mentions that several persons were attacked in the village of Carisbrook, in the Isle of Wight, after eating some stale cowheels which had belonged to a man at Newport, who had died a day or two before. In future epidemics one may be sure that observers will be on the lookout to trace the spread of cholera to milk supply.

There are still to be mentioned certain conditions which seem undoubtedly to influence the diffusion of cholera, however indirect their operation may be. Dr. Farr laid down a "law of altitude," at least for London, showing that the incidence of the disease upon the population varied inversely as the height above the River Thames. This, however, is but an expression of the fact that on an average persons living at a low level are affected by the causes of cholera in larger numbers than persons living at a high level. Sometimes the case is reversed; thus Lebert says that throughout the epidemic at Zürich, in 1855, the upper parts of the town suffered more than the rest. The circumstance that in Europe cholera has never been known to prevail at an elevation of more than 600 or 800 mètres above the sea may be set down to the absence of the conditions required for its development; in other quarters of the world it has raged at altitudes of 2000 to 2500 mètres. In temperate climates the summer and the autumn are the seasons most favorable to it. It often dies out as winter approaches, to reappear in the following year. But in Russia, in 1830, it withstood extreme degrees of cold.

The *diagnosis* of cholera in its well-marked forms is seldom difficult. Almost the only morbid state which can be mistaken for it is that produced by the poisonous administration of arsenic. Lebert relates how Louis actually committed this blunder in regard to the Duke of Choiseul, who killed himself with arsenic the day after his arrest for the murder of his duchess. At that time, in the summer of 1847, the disease was not prevailing in Paris. There is, therefore, good reason for bearing in mind the possibility that a crafty person might take advantage of an epidemic to give poison with but little risk of being suspected.

To distinguish from an ordinary diarrhœa, the slighter effects of the virus of cholera is often quite impossible. Under the name *cholera nostras* or *cholera Europæa* writers describe an affection of which the symptoms are identical with those of the specific disease (called by them *cholera Asiatica*), but which differs from it in being scarcely ever fatal and in generally occurring sporadically. A typical instance occurred at Guy's Hospital, in 1865, and

may be found recorded by Wilks in the "*Medical Times and Gazette*" for that year. A blacksmith's hammerer, aged thirty-three, was at work as usual at 7 A.M. on May 25th, when he was suddenly seized with profuse vomiting and purging. He was admitted collapsed, with cold breath; he had cramps in the flanks and in the legs; the evacuations and the matters vomited had the appearance of rice-water, consisting of an alkaline liquid, with flocculi floating in it. He recovered in about twenty-four hours. Dr. Wilks is reported to have said that every year he saw one or two such cases, but seldom so early in the summer. It is to be observed, however, that at the very time when this man was attacked, an epidemic of cholera was approaching England. And utterly improbable as it may seem that an isolated case should spring up in London several weeks before the commencement of the local epidemics at Southampton and at Theydon Bois, in the autumn of the same year, I do not think that the possibility of such an occurrence can be denied. At any rate, so far as I am aware, no case of so-called "English cholera" (attended with collapse, rice-water stools, and cramps) has been admitted into Guy's Hospital since 1870; and it is certain that when the first epidemic arose in 1831, Sir Thomas Watson, and the elder Dr. Babington, and many medical men of experience declared that they had never "met with the same complaint before." I am therefore disposed to doubt whether it is necessary to call any disease cholera except that which I am now describing; and I am sure that on every ground it is very undesirable to designate attacks of diarrhœa, in infants or even in adults, "choleraic," whenever they happen to be attended with symptoms approaching those of collapse.

The *prognosis* is less favorable in children and in old people suffering from cholera than in adults or in middle-aged persons. The mortality, which over a whole population generally averages about 50 per cent. or a little higher, rises among the former classes to 70 or 80 per cent., while among the latter it may fall to about 40 per cent. The disease is more than usually fatal when it affects drunkards or those who are already sick or weakly. In an individual case the degree of collapse is of far more prognostic importance than the amount of purging.

Treatment.—The *preventive measures* which should be adopted when a locality is threatened or actually attacked with an epidemic of cholera were carefully elaborated by Mr. Simon and his colleagues in 1866, and will be found detailed in his ninth "Report." They are generally such as might be anticipated from what has been already stated with regard to the ætiology of the disease. Mr. Simon also attached great importance to the avoidance of all such food as is apt to set up diarrhœa—half-fermented beer or wine, meat or game no longer fresh or not completely cooked, stale fish or shellfish, vegetables or fruit long gathered or badly kept, and the like. He deemed it unwise to take purgative medicines, except of the mildest kind. And, in accordance with almost every other observer, he insisted on the necessity at such seasons of checking looseness of the bowels, however painless and trivial. Dr. Bristowe, I believe, stands alone in maintaining that the diarrhœa which so commonly prevails when cholera is epidemic is neither more nor less likely to pass on into that disease whether it is left alone or encouraged by purgatives; nor will he allow that, if it is really premonitory of cholera, it can be arrested by any medicine whatever. Most writers recommend that it should be treated with opium in considerable doses, and with astringents, such as acetate of lead, nitrate of silver, catechu, or chalk. I think, however, that it is doubtful whether the physiological action of these drugs upon the hepatic and the other secretions may not be injurious. In India Dr. Goodeve says that it is usual to give from one to five grains of calomel with the first two-grain doses of opium; in my limited experience I have found a simple alkali with a little ammonia and peppermint effectual in checking

much of the diarrhœa occurring during a cholera outbreak; but whether this treatment would suffice for an attack really premonitory, I would not venture to say. It is, of course, admitted on all hands that some cases come under medical observation at a very early period, and yet run on into cholera in spite of everything that is done to control them.

When *collapse* has actually developed itself, the administration of opium or astringents is believed to be useless, or even worse than useless. The state of the circulation is now such that absorption is nearly arrested; and drugs introduced into the stomach in successive doses may, if not vomited, accumulate there so as to produce inconvenient effects at a later stage, should reaction happily occur. For the same reason, alcohol must be used with great caution, if at all. Dr. Goodeve says that its admissibility depends upon its effect on the pulse. If a little weak brandy and water causes the pulse to revive ever so little, whereas it falls back or disappears when the brandy is withheld, there is no harm in continuing to give the stimulant in small quantity. In any case ether or ammonia may be used, properly diluted, unless sickness is produced. But Dr. Goodeve finishes his remarks upon this point by saying that many cases in which there is extreme collapse come round by themselves, as well as, if not better than, with stimulants. Ice-cold water may be freely allowed to patients with cholera, not indeed in such large quantities as their morbid thirst might lead them to swallow, but by tablespoonfuls or by wineglassfuls at a time. Lebert would also give effervescing draughts made with carbonate of soda and with lemon juice; and at the London Hospital in 1866, a "saline lemonade" was employed with apparent advantage.

There seems to be no objection to administering a dose of morphia subcutaneously if the cramps are very severe. Or an occasional whiff of chloroform may be given by inhalation. It may also give relief to rub pure chloroform or turpentine into the painful parts. The burning sensation at the epigastrium may be alleviated by the local application of cold, or of a sinapism. The limbs should be kept wrapped in warm flannels, and hot bottles should be placed in the bed, but not in contact with the patient's skin. At the London Hospital, in 1866, baths at a temperature of from 98° to 104° were employed in about one hundred and thirty of the worst cases, and with most marked effects. For a few seconds after immersion there was commonly difficulty of breathing, and sometimes an unpleasant sensation of heat. But in less than a minute a favorable action generally became manifest; the pulse returned, or (if it had been perceptible before), became fuller and quicker; the patient became less distressed, ceased to moan or to shout with pain, and even fell into a quiet slumber. In many cases recovery appeared to be the direct consequence of the bath; but in many more the symptoms afterwards returned.

The experience at the London Hospital seems also to have been rather favorable to the practice of injecting a warm saline solution into a vein, when patients are in an apparently hopeless condition from cholera collapse; among fifteen cases so treated by Mr. Little there were four recoveries. Most writers, on the other hand, speak of this procedure as incapable of producing any permanent good result, and therefore as deserving to be abandoned. The immediate effect of an injection is admitted on all hands to be often most wonderful; a person speechless, and almost dead, may regain consciousness, sit up, talk, and even joke; and the pulse may become distinct and full. But in most cases the improvement has been only temporary; the symptoms soon return and end fatally even though the operation may have been repeated. Still, as Sir Thomas Watson observes, even such a transitory amendment may sometimes be of great importance, enabling, for example, a will to be executed. The fluid used by Mr. Little con-

tained sixty grains of chloride of sodium, six grains of chloride of potassium, three grains of phosphate of soda, twenty grains of carbonate of soda, and two drachms of pure alcohol, to twenty ounces of distilled water. Eighty ounces were introduced at a time, the temperature being about 110°, and from twenty to thirty minutes being occupied in the operation. There were three cases in which the injection was repeated; one of them ended successfully.

When *reaction* begins, the management of a case of cholera continues to require great care and caution. A little beef tea or chicken broth may now be given in small quantities at intervals, or some light farinaceous food, such as gruel or arrow root. Should vomiting continue, it may often be checked by a dose of opium, or by a blister applied to the epigastrium. But sometimes it is necessary, for a time, to have recourse to nutrient enemata. To make up for the loss of salts from the blood, carbonate of soda and chloride of sodium should be given in moderate quantities. If the tongue becomes dry and brown, a powder containing two grains of hydrargyrum cum cretâ, half a grain of ipecacuanha, and two and a half grains of Dover's powder, is said to be very useful, according to the experience at the London Hospital. When suppression of urine continues during reaction, the patient should be encouraged to drink freely; he may have a drachm of liquor ammoniæ acetatis, with five or ten drops of tincture of digitalis, every two or three hours; and mustard plasters or cupping glasses may be applied to the loins.

THE PLAGUE.

HISTORY—INCUBATION, ONSET AND SYMPTOMS—DIAGNOSIS—FATALITY—TREATMENT—ANATOMY—CONDITIONS OF CONTAGION AND CAUSES OF ITS DECLINE.

Throughout the middle ages, the English word Plague was applied indiscriminately to every fatal epidemic disease; and when the malady now to be described appeared, in the fourteenth century, it became known as the "Black Death." But for the last 200 years the only epidemics comparable with those of former times have been distinguished as epidemics of fever. Consequently we can now speak of "the Plague," without ambiguity, although some writers still prefer the term "Oriental Plague," or "Levantine Plague." Its prevalence has for very many years been gradually declining. In the early part of this century there were outbreaks of it in Malta, at Noja in South Italy, upon the Lower Danube, and in the Balkah Peninsula. Up to 1844 they were of frequent occurrence in Egypt, but that country has since been free from them. The disease also vanished from Syria, and hopes seem to have been entertained that it had become entirely extinct. But in 1858 it appeared again in Tripoli, on the south shore of the Mediterranean; and it is now known to have visited the highlands of Western Arabia in 1853. There is also reason to believe that it had never been absent for many years together from parts of India; in 1834 an epidemic which occurred in Rajpootana became known as the "Pali Plague," and as recently as 1877 it prevaled at Kumaon, on the southern slopes of the Himalayas. Between 1863 and 1876 there were six or seven outbreaks of it in Western Asia; some in Persian Kurdistan, others upon the Lower Euphrates. In 1877 it showed itself at Resht, near the southwest corner of the Caspian Sea. Many observers believe that from this place it made its way to the Lower Volga, where it prevailed, in the district between Astrakhan and Tsaritzin, from October, 1878, to February or March, 1879, and excited great alarm throughout Russia, and in Europe generally.

Course.—I need not give a detailed account of the general symptoms of the plague, which resemble those of any other severe febrile disease. Its incubation is believed to be from two to seven days. This is followed by great malaise and depression, so that the patient sometimes totters like a drunken man. Intense pyrexia then sets in, which may be accompanied by violent delirium, and may rapidly assume a typhoid form. Death not uncommonly occurs at a very early period, before there have been any symptoms characteristic of the malady; it may be preceded by the development of petechiæ and large vibices in and beneath the skin; there may also be hæmaturia, and the vomited matters may be blackened by altered blood. But if life is prolonged to the second or the third day, there commonly appear one or more *buboes,* attended with severe pain and tenderness. The seat of this affection may either be the groin (in which case

glands belonging to the vertical rather than to the horizontal chain are affected), the armpit, or the neck. According to Liebermeister (in Ziemssen's "Handbuch"), a plague bubo is sometimes so small as to be detected only on careful examination, but it may attain the size of a hen's egg or more. Its formation is commonly attended with the subsidence of the delirium and of the fever, the skin becoming covered with a profuse sweat, the pulse falling to 90 or 100. It may ultimately subside without discharging, but usually it breaks, or is opened by the surgeon, and gives exit to a mixture of blood and pus, and this may be followed by a tedious process of suppuration, which greatly prolongs the patient's illness. Another affection which may be associated with the buboes, but which is less often seen, is of a carbuncular character. It appears most often upon one of the lower limbs, upon the buttock, or upon the neck; sometimes there are no fewer than a dozen carbuncles in the same case. Bullæ and pustules may also appear upon the surface of the skin.

Diagnosis.—It will readily be understood that to distinguish the plague from other malignant fevers is not always easy, unless the character of a prevailing epidemic has already been determined. Dr. Milroy, in "Reynolds' System," cites the remark of Heberden that "on first breaking out, the disease has never been known to be the plague;" and he says that in Constantinople or in Cairo, no physician ever ventures to give a name to an epidemic until a case occurs in which a bubo or a carbuncle is seen. Moreover, as he goes on to point out, similar local affections are sometimes, though very rarely, present in other forms of pernicious fever, whether malarial or contagious. But it is certain that the diagnosis would not have been left open so long as it often has been, were it not for the reluctance to admit unpleasant truths which has always contributed so largely to the spread of every infectious disease. According to Dr. Milroy, a special difficulty has often arisen, during the prevalence of the plague, from the occurrence of glandular pains and swellings, or of carbuncles, in persons who remain well enough to follow their occupations, and who speedily get well under any simple treatment. He himself expresses no opinion as to whether such cases should be regarded as examples of the plague, and submitted to an enforced segregation. On the other hand, Liebermeister speaks of instances, especially when an epidemic is declining, in which the general symptoms are mild, and which run a favorable course without any local lesion appearing, so that they may be termed abortive.

Prognosis.—The fatality of the plague exceeds that of all other diseases. It usually destroys from 70 to 90 per cent. of those whom it attacks; very seldom less than 60 per cent. Moreover, it often carries off half the population of a town, or of a district, in which it prevails, and it may completely root out whole families, so that no survivor remains. The "Riley Graves," near Eyam, in Derbyshire, still tell of the tragedy that befell two families in that place, during the epidemic of 1666; one, that of the Talbots, consisting of seven persons, was utterly eradicated within twenty-five days; the other, that of the Hancocks, lost seven out of eight members, the only one left being the mother. It is, however, probable that no small part of the excessive mortality from this disease was due to the fact that instead of isolating each case at the earliest possible moment, the ancient practice was to close up every infected house, preventing both ingress and egress, and confining the sick and the healthy together. Those who were attacked were often left without any nursing, to take their chance of death or recovery.

Treatment.—There can be no doubt that it is of the first importance to maintain the patient's strength with plenty of food. He should also be given alcoholic stimulants, ammonia, ether, and the like. Liebermeister suggests that quinine in large doses, as an antipyretic, should have a good

trial. He also recommends the use of the cold bath, and says that cold affusion has already been found useful. The buboes should be opened early.

Morbid Anatomy.—Post-mortem examinations throw little or no light upon the plague. The viscera are soft and blood stained, and the spleen is more or less enlarged. The serous membranes are ecchymosed Some of the internal lymph glands are enlarged; perhaps a chain of swollen glands extending up from the pelvis along the front of the spine, or a mass lying in the mediastinum; their tissue is of a bright red color, or full of points of suppuration; and blood may be diffused and extravasated into the surrounding structures.

Contagion.—That the plague is propagated by an infective principle derived from the bodies of the sick is probably now doubted by no one, although in former times there were "non-contagionists" with regard to this, as to every other disease. Liebermeister, indeed, advocates the opinion that its mode of diffusion is "indirect," and that it is *miasmatic-contagious*, rather than simply *contagious;* but I fail to discover any sufficient basis for this suggestion. The statement commonly made that it is inoculable might be supposed to settle the question; but, according to Liebermeister, the results of the experiments which have been made in this direction are in reality far from conclusive. It protects against itself; second attacks are rare, and when they occur they are generally of diminished severity.

The virus seems very often to be conveyed by articles of clothing, wool, silk, hair, paper, books, etc. In 1665, the local epidemic at Eyam, in Derbyshire, so well known by the heroism which it called forth from the vicar, Mr. Mompesson, began with the case of a tailor who had just received a box of clothes from London (150 miles off), and who was watching them hanging before the fire when he was taken ill; at that very time the disease in the metropolis was at its worst. The bedding and the linen of those who have had the plague are particularly apt to be carriers of infection to other persons, sometimes after a considerable interval of time. When epidemics used to occur in Western Europe, the notion was that it reached Holland direct from Turkey, in bales of cotton or silk, and that it was brought over to England from Holland in a similar way.

Many collateral circumstances, however, greatly influence the spread of the disease. It is said to have been especially apt to prevail where the soil is alluvial and marshy, as for example, along parts of the Mediterranean coast, and in the basins of great rivers. Warm and damp weather are favorable to it; thus in European Turkey it occurred chiefly in the spring and in the early summer, whereas in Egypt the winter was the chief season for it. In Nubia, and in other countries with a hot, dry climate, it has hitherto been unknown. Overcrowding, dirt, and poverty seem greatly to encourage its development. It affects especially the poor; and at Malta, in 1813, it was noticed, according to Dr. Milroy, to be far less common among the inhabitants of the upper stories of the lofty and more airy houses of Valetta than among those who lived in the basements. The higher parts of a district sometimes escape when the lower are infected with the plague; thus it is said that the citadel of Cairo and the village of Alem Dag, near Constantinople, used to remain free, although communications with the cities below were not interrupted. Whatever weakens the health, including the dread of the disease, is believed to favor its invasion. Lastly, it seems less apt to occur in persons over fifty years old than in those who are younger.

It is greatly to be hoped that the improvement in the hygienic condition of the inhabitants of Western Europe, which has been going on during the last two centuries, has been the chief reason why the plague no longer appears among us, and that Hirsch is wrong in attributing this result

mainly to the system of quarantine. For it is certain that at the present day, if the disease were again to travel toward our shores, there would be extreme difficulty in maintaining that system effectually. Liebermeister cites in detail the proceedings that were adopted to prevent the extension of the plague from Noja, in Italy, in 1815. The town was surrounded by two deep ditches, a triple military cordon was drawn round it, the soldiers had orders to shoot down any one who attempted to pass, and no articles were allowed to be sent out except letters which had been first dipped in vinegar. The advantage to be derived from such measures is well illustrated by instances in which large bodies of persons isolated in the middle of an infected city have escaped. Thus Dr. Aitken says that during an epidemic at Marseilles a large nunnery was "shut up," with the result that all the inmates remained free, although there was an infirmary on one side for those ill of the disease, and a burying-ground on the other side for those who died of it. This writer, however, speaks of quarantine as a barbarism and as an unwarrantable nuisance; and it is well known that many of the best English authorities hold the same opinions. Indeed, the chief reason why our Government from time to time enforces quarantine regulations in our possessions in the East and elsewhere, is that if we did not do so, other countries would at once refuse intercourse with our more exposed ports on the mere chance of our becoming a source of danger to them.

EPIZOÖTIC DISEASES.

(*GLANDERS, ANTHRAX, FOOT AND MOUTH DISEASE.*)

GLANDERS: ITS HISTORY AND NOMENCLATURE—ORIGIN—ACUTE FORM—ERUPTION, OZÆNA, ETC.—PYREXIA—FATALITY—CHRONIC FORM.
ANTHRAX: NOMENCLATURE—THE BACILLUS—ÆTIOLOGY—MODES OF INFECTION—VARIETIES: CHARBON, INTESTINAL AND THORACIC ANTHRAX.
FOOT AND MOUTH DISEASE: IN CATTLE—IN MAN.

There are three epizoötic maladies, Glanders, Anthrax, and the Foot and Mouth Disease, which sometimes pass from animals to man. These may conveniently be described in succession.

GLANDERS.—From an early period in the present century it has been known that those whose occupation brings them into contact with horses sometimes become affected with a disease to which these animals are liable. The disease in question was described as far back as the fourth century A. D., under the name of μᾶλις or *malleus*. In England, writers on veterinary medicine have been accustomed to break it up into two affections, "glanders" and "farcy," the former of which is characterized by a morbid state of the nasal mucous membrane, the latter by the formation of nodules and abscesses in and beneath the skin, and by indolent inflammation of the lymph glands and lympathics, attended with swellings (or, as they are termed, "farcy buds") at the valves. But it is now acknowledged that these are only varieties of a single malady, and it is to be hoped they will hereafter receive a single name. The first case in the human subject of which the nature was correctly interpreted in this country, occurred in 1821, to Mr. Muscroft, of Pontefract. In 1830 Dr. Elliotson drew the attention of the Royal Medical and Chirurgical Society to the disease, and at a later period he proposed to call it *Equinia*. But the term glanders seems to be preferable; according to Virchow its derivation is from the Latin *morbus glandulosus*, the equivalent German word *Druse* being, in fact, still in use for one of the varied forms of the malady.

Contagion.—As a rule, the virus of glanders is directly introduced into a wound or sore, or fissure in the skin, especially of the face or of one of the hands. A horse slaughterer who died in Guy's Hospital in 1866 appeared to have poisoned a slight cut on the lip which had been made by a barber in shaving him. Injuries to the hand in skinning dead horses, or in dissecting them, have sometimes been the starting point of the disease. It has also been transmitted by a bite from a glandered animal, probably through the saliva. Or a diseased horse, in sneezing, may propel a drop of muco-purulent secretion from its nose directly into the eye, or the nose, or the open mouth of any one standing near it. The contagion remains active in a dried state for a long time, for horses in a particular stable have been attacked many months after the occurrence of a case. Thus, nothing is more likely than that it should sometimes be deposited upon the straw in a stall, and that a groom, or any one else sleeping upon the straw afterward,

should disturb the dried particles and cause them to float off into the air so as to infect him with the disease. However, the Messrs. Gamgee, in Dr. Reynolds' "System," express doubts on this point.

According to Bollinger, who describes glanders in Ziemssen's "Handbuch," it may be communicated by eating the flesh of a glandered animal, at least in a raw state; he says that lions in menageries often become diseased in this way. Decroix, indeed, is stated to have repeatedly fed upon such flesh uncooked, without injury. But it must be borne in mind that in all probability comparatively few persons are susceptible of the virus of glanders. At any rate, the disease in man has always been exceedingly rare, even in countries in which (as in France during the first half of this century) it has committed the most terrible ravages among horses. When it affects human beings, it is capable of spreading from one individual to another. Dr. Elliotson mentions in the "*Lancet*" for 1838 that a laundress who washed the clothes of one of his patients contracted it. In at least one case infection has taken place through a cut received in examining the body of a person who had died of it. It is also inoculable from man to the lower animals, and goats and rabbits are said by Bollinger to be the most suitable subjects for such experiments. Oxen are stated not to be susceptible of it. It may occur in all other kinds of domesticated animals, but next to horses, it is especially apt to affect asses and mules. It is seen in hot as well as in cold climates. A point of some importance is that grooms and other persons who have to do with horses are sometimes attacked with glanders when they are not aware that any of their animals have been diseased. Bollinger seems to have satisfactorily explained this, by showing that in the horse glanders sometimes fails to present its more characteristic symptoms and attacks the lungs and the air passages only.

In man the disease occurs in two forms, of which one is termed acute and the other chronic.

1. In *acute glanders* the patient is first attacked with malaise, headache, and pains in the limbs. There is often no initial rigor, and for a time there may be no marked pyrexia. However, at this stage the disease is generally mistaken for acute rheumatism or for enteric fever. But if a wound or scratch has been infected with the virus, the part commonly becomes red, swollen, and very painful; and an erysipelatoid inflammation may diffuse itself over a wide area, affecting, for example, the whole side of the face, or the hand, and part of the forearm. In such cases the diagnosis generally inclines toward septicæmia or pyæmia from a poisoned wound. In other instances, as in that of a man who died in Guy's Hospital in 1863, the first complaint is of pain in the side, with dyspnœa, so that acute pleuropneumonia is suspected.

Presently symptoms appear which to an experienced eye reveal the nature of the disease, and which, indeed, correspond fairly well with those which characterize it in horses.

One of these is a cutaneous *eruption*. Upon the surface of the limbs and of the body there arise papules, which rapidly pass into flat vesicles and then into bullæ or pustules, attaining the size of peas or even of sixpenny pieces. They become depressed in the centre, and rupture, allowing a thin, purulent fluid to escape, which is often blood stained. With regard to their histology, von Wyss has ascertained that the papulation begins as a local inflammatory change in the papillary layer of the cutis. When an incision is made through a pustule after the patient's death, its floor is found to consist of an ashen-gray, infiltrated layer. The eruption sometimes appears within twenty-four or forty-eight hours after the patient is first taken ill, sometimes not for a week, or even longer. It is, I believe, quite characteristic

of glanders, although it might be mistaken at an early stage for smallpox or chicken-pox, and perhaps later on for pemphigus. Generally there is mixed with it an affection which may be taken as the representative of "farcy" in horses. This consists in the formation of hard, painful lumps or swellings in the subcutaneous tissues and muscles; they more or less rapidly suppurate, and if they are incised they may ulcerate, so as to expose tendons or bones beneath. It is curious, however, that in most cases the lymph glands fail to become enlarged. The most marked instance to the contrary that I have met with is one recorded by Travers in his work on "Constitutional Irritation." Not only was there suppuration of the glands of the arm originally affected, but those at each angle of the lower jaw and in the groin are also said to have been swollen. The joints in glanders not infrequently become inflamed, and pus is effused into them.

Ozæna.—The other characteristic symptom is an affection of the mucous membrane of the nose and of adjacent parts, representing what in horses is termed "glanders" in the narrower meaning of the term. First a thin, whitish mucus runs from one or both nostrils; afterward it becomes purulent, blood stained, and fetid. The nose itself now becomes swollen, red, and very painful, and inflammation may spread from it toward the forehead or over the cheeks. If an opportunity arises of examining the parts after death, the lining of the nasal passages is found to be ulcerated, and the septum may even be necrosed. It is to be observed that in the human subject a discharge from the nose is by no means always present in glanders; according to Hauff it was observed in only thirty out of seventy cases which he collected. It is often not an early symptom, but appears in the second or the third week. Thus, during the first few days of a doubtful case, the fact that the nose is healthy must never be supposed to be of diagnostic significance.

Other mucous membranes are also affected. The conjunctivæ may be severely inflamed, and the eyelids may become greatly swollen. Virchow relates a case which came under von Graefe as one of acute exophthalmos, and its real nature was not suspected until after the autopsy. Sores may form in the mouth, and the gums may become spongy. The pharynx and the palate may ulcerate or become covered with a kind of false membrane. Sometimes ulcers form in the larynx, producing hoarseness, and an œdematous laryngitis may set in, so that tracheotomy may be required. Symptoms of bronchial or of intestinal catarrh may be present. Pustules and sores, which might easily be supposed to be chancrous, are said by Virchow to occur on the glans penis.

The general condition of the patient becomes worse from day to day. The pulse is generally much accelerated, but sometimes it remains slow. The temperature rises irregularly until it may reach 104°. The tongue becomes dry and brown. Albumen appears in the urine, and sometimes leucin and tyrosin. Delirium and sleeplessness pass into stupor and coma. Finally there are symptoms of collapse, with involuntary escape of the urine and fæces. Death usually occurs toward the end of the second or in the third week. Sometimes the disease has ended fatally within a week, or even in three or four days, sometimes not for four weeks. Bollinger gives one case of recovery among thirty-eight which he collected. All that can be done in the way of treatment is to sustain the patient's strength with quinine, tincture of iron, nourishing food, and stimulants.

2. *Chronic glanders* is much more insidious. It is often characterized by the formation of intractable ulcers with thick, livid edges. In the museum of Guy's Hospital we have two models of such ulcers, the part affected being in one case the back of the hand, while in the other case there are separate sores upon the forehead, the lobule of the ear, and the side of

the face. In other instances, abscesses form about the joints and give rise to fistulous sores; or inflammatory swellings appear on the limbs, beneath the skin, or in the muscles. Or, again, there may be an eruption of pustules like those which are seen in acute glanders, but more slowly developed. The nose often remains free, but sometimes there is an erysipelatoid redness of the skin or a fetid, purulent discharge from the mucous surface, beginning, perhaps, after two or three months have passed. Bollinger says that the root of the nose may even become gangrenous. The nostrils are often blocked with dark-colored crusts. Such an affection is very apt to be regarded as syphilitic, or to be classed under the vague head of ozæna. It is of great importance to remember that the diagnosis may be cleared up by inoculating a goat or a rabbit with some of the discharge. This practice was adopted in some of the earliest cases of glanders that are known to have been observed in this country, those which were recorded by Travers. Bollinger gives details of two such experiments which he performed with matter taken from horses; the inoculated animals, which lived two or three months, showed characteristic symptoms and lesions. In a case that occurred at Guy's Hospital in 1866, Poland introduced pus from the patient's face beneath the skin of the shoulder and set up pustules there; but I do not suppose that this result was of any diagnostic significance. Virchow mentions that at Würzburg a man was for six months in the hospital on account of refractory sores upon the limbs. At the autopsy, lesions characteristic of glanders were discovered. It was not then known that the disease existed in the town or in the neighborhood, but inquiries were set on foot and led to the discovery of an epizoötic prevailing over a wide area among the horses used for towing barges on the Main. In some cases the chief symptoms of chronic glanders are cough, with sanguineous expectoration, hoarseness, pyrexia, and emaciation, so that phthisis may be suspected.

The average duration of chronic glanders was found by Bollinger to be four months; sometimes it was not more than two or three months. It may end by passing into the acute form of the disease. Among thirty-four cases which Bollinger collected from various sources, including many of slight severity, there were seventeen recoveries.

As might have been anticipated, the convalescence is generally very slow, and is sometimes incomplete. He was himself acquainted with a veterinary surgeon who, at the end of eleven years, was still cachectic and was troubled with cough and other symptoms due to the presence of cicatrices in the larynx and in the nose. But he says that some patients, after a severe illness lasting for months, get quickly well and regain the flesh that they have lost.

He thinks that carbolic acid and iodide of potassium are the internal medicines most likely to be useful; the Messrs. Gamgee speak of arsenic in combination with strychnia as having sometimes exerted a remarkable influence. Ulceration of the interior of the nose should be treated with injections of creasote water, or a solution of carbolic acid, and the application of nitrate of silver, or tincture of iodine may be of service.

Pathology.—It does not appear that the contagion of glanders has yet been traced satisfactorily to any form of microzyme. Some observers have spoken of the presence of bacteria, but Bollinger says that he failed to find such organisms in very careful examinations of the fresh blood of glandered animals and of fresh nodules from their bodies. He cites Gerlach as having stated that the virus is not destroyed by putrefaction.

Many of the *internal lesions* which are found in cases of glanders in man appear to be the result of pyæmia or septicæmia, which often arises secondarily, just as it does in erysipelas; but nodules and more or less extensive patches of catarrhal pneumonia, which have no pyæmic characters, are

also seen. Virchow placed glanders, in association with syphilis and lupus, among the "granulation tumors." But he himself states that in the human subject the process concerned in the formation of the pustules and abscesses has little to distinguish it from a simple inflammation. In animals, both he and Bollinger describe the affection of the nasal mucous membrane as beginning with the development of minute miliary papules, and in the lungs they speak of nodules of various sizes, some almost exactly like tubercles, others as large as peas or even as walnuts. In man, Virchow says that on careful examination characteristic small yellow bodies may be seen in the mucous membrane of the nose, of the frontal sinuses, and even of the larynx and of the trachea. In von Graefe's case he detected them in the choroid of the eye. Von Wyss has since observed them in the gastric mucous membrane. It is interesting to find in one of Elliotson's cases, recorded in the "*Med.-Chir. Trans.*" for 1833, a description of similar appearances. "On cutting into the various tumefactions on the head, trunk, and extremities, they were found to be full of pus, underneath which, in many, a number of small white granules were seen, and others, in several instances, were closely attached to the periosteum and perichondrium. The frontal sinuses contained a jelly-like mucus, and a number of similar granules, and on the septum narium was one ulcer exactly like those I have seen in the nostrils of glandered horses, and upon it lay a cluster of granules. Two of these large, white, circular elevations were found immediately below the sacculi laryngis; Mr. Youatt, who was present, called them true glanderous chancres. About an inch below the valve of the colon, for three inches in extent, upon the whole of the surface were white granules exactly like those in other parts."

In a case which occurred at the Middlesex Hospital in 1872, and which is recorded in the "*Med. Times and Gaz.*" for that year, Dr. Coupland discovered an acute inflammation of the spinal cord. He mentions that in another case large parts of the brain were found to be softened.

Anthrax.—It seems probable that the name of Anthrax, which has hitherto been used as a synonym for carbuncle, will in the future be reserved for a very different disease which human beings derive by infection from one that is epidemic in cattle, horses, sheep, and other herbivorous animals. This disease has until lately been very imperfectly understood in England, and has generally been termed *Malignant Pustule* or *Charbon*, the latter being the French designation for the epizoötic malady. The Germans call it, whether in animals or in man, *Milzbrand*. But certain forms of it have also, in all European countries, been commonly known under the name of *Anthrax*, and others have been described as *Anthracoid*.

In different kinds of animals, the symptoms of infection by the specific virus of anthrax differ considerably. But, according to Bollinger, three principal varieties may be recognized:—

1. Sometimes the animal (generally an ox or a sheep) becomes convulsed and insensible, with rapid breathing, and dies at the end of some hours, or even in a few minutes.

2. Other cases are characterized mainly by pyrexia, which is often remarkably remittent in type. Clonic spasms of the limbs are also observed. The fæces may contain a large quantity of blood.

3. In yet other cases the most marked feature is the formation of brawny inflammatory swellings in and beneath the skin of the neck, chest, abdomen, or any other part, which may lead to extensive sloughing and ulceration.

Obviously, only the last of these varieties would naturally be termed anthrax. But it is on every ground very desirable that a single name should

be given to them all. Indeed, although pathologists until lately regarded the more rapidly fatal cases as examples of a specific fever, without localization, it is now known that there always is in reality a local lesion in some part of the body. Very often this consists of an infiltration of gelatinous exudation and blood into the connective tissue of the abdomen and of the chest. Or there may be an acute inflammatory swelling of the tongue, or of the submucous tissue of the pharynx, or of the lining membrane of some part of the intestine. We shall presently see that all these forms of the disease are also met with in men. It is to be observed that anthrax is by no means always fatal to animals; the average mortality in horned cattle and in horses is said to be 70 per cent. In cattle a very characteristic lesion is an enlargement of the spleen, which is from two to five times its normal size, and which is also softened and of a black color.

The proof of the identity of the various forms of anthrax lies in the fact that they are all caused by a single microzyme, which is now commonly known as the *Bacillus anthracis*. This organism was discovered independently by two German observers—by Pollender, in 1849, by Bräuell, in 1857. But Davaine was the first who, in 1863, maintained that the contagion of the disease lay in these bodies, which, on account of their being motionless, he distinguished under the name of *bacteridia*, from the common mobile bacteria of putrefaction. They consist of straight or slightly-bent rods, measuring 0.007–0.012 mm. inlength. Frisch and Dr. Cossar Ewart have, under certain circumstances, observed independent movements in them. By Cohn, they have been classified as belonging to a form of bacillus, which he terms *B. anthracis*, though he observes that it is almost exactly like the *B. subtilis* which constitutes the butyric acid ferment.

The rods sometimes cohere together at their extremities, and Koch and Ewart have described them, when cultivated in aqueous humor, as growing into long filaments, in the interior of which bright granules appear. To these granules great importance has been attached, it being supposed that they are spores of the bacillus, far more capable of resisting heat and desiccation than the adult microzyme, and retaining for an indefinite length of time the power of development into it. Such a view is, however, rejected by Nägeli, who maintains that bacilli, like bacteria, multiply only by fission.

The *ætiology* of anthrax, as it occurs in cattle and horses and sheep, is of great interest, and seems to have important bearings upon the class of human diseases which are known as *miasmatic-contagious*, and of which it seems to be a representative.

In the first place, it is readily inoculable from one animal to another. Inoculations are frequently practiced by veterinarians and by pathologists, for diagnostic purposes. There is reason to believe that the virus is not infrequently introduced by the stings of insects. Bollinger took gad-flies from the body of an ox that had died of anthrax, and found by direct experiment that the contents of the stomach and intestines of these flies were capable of conveying the disease to rabbits. He even thinks that droppings from flies upon the skin of an animal may suffice to communicate it. Another way in which it is sometimes given to sheep is by the bite of a dog that has been just feeding upon infected flesh.

On the other hand, there seems to be no proof that emanations from the bodies of living animals affected with anthrax pass into the air and infect other animals through the lungs. Like enteric fever and cholera in man, it appears not to be directly contagious. Yet the virus of the disease is said by Bollinger sometimes to cling to stable utensils, harness, straw, or hay. He mentions a local outbreak which he carefully investigated at Weriken, and in which for four years the cattle in two sheds of the same proprietor were decimated by anthrax, while it did not affect those in other buildings,

some of which actually lay between the two that yielded so many cases. An analogy for this occurrence may be found in those local house epidemics of enteric fever in which the virus seems to go on multiplying itself in or near a particular water closet, so as to affect a number of persons in succession, sometimes at long intervals of time.

Anthrax also resembles the miasmatic-contagious diseases of man in being often spread in ways still more indirect. It prevails especially among animals pastured upon damp soils containing much humus, as for instance, upon peat-bogs, and near the borders of lakes or rivers that have overflowed. And it is most frequent during the hot months of the year, particularly in August and September, There often seems to be a connection between it and ague; but this is only a coincidence, for in other districts the two affections occur quite independently of one another.

Buhl has accordingly included anthrax among the diseases to which he applies his "ground-water" theory; and he states that among the horses belonging to a large stud kept near Donauwörth the disease, which had been raging for a long time, ceased as soon as a system of drainage was carried out. But Bollinger argues that the reason why its prevalence is affected by dampness of soil is, in reality, that ground containing moisture affords conditions favorable for the multiplication of the *Bacillus anthracis*, which he supposes to be capable of maintaining an independent existence under such circumstances. Some observers have even thought that this organism may sometimes appear without having been derived from a previous case of anthrax. Buchner has recently asserted that, by a very arduous series of experiments, he converted a bacillus which is found in infusion of hay into an organism capable of producing in animals an infective disease identical with anthrax, and he asserts that to turn the anthrax bacillus into a hay bacillus is comparatively easy.* This is equivalent to saying that the malady may sometimes be purely *miasmatic*. But Bollinger maintains that this is never the case. He believes that the specific bacilli of anthrax exist in the soil, or in water, only when they have been introduced from a previous case. They may be either derived from the excreta of diseased animals, or from their dead bodies, buried as they often are in the fields, or even left to decay among the brambles and nettles. The same view has been strongly upheld by Nicolai, to whose work I have not been able to refer; and Oemler and Leinhardt are said to have recorded instances in which 'enzoötics" of anthrax have ceased when stringent rules for the disposal of all dead animals were enforced. Probably some of the bacilli undergo desiccation, are suspended in the air, and inhaled into the lungs; others pass into drinking water, and thus reach the stomach.

With regard to the *geographical distribution* of anthrax, the parts of Europe in which it is most common are said to be Poland, Hungary, the countries on the Lower Danube, Prussian Saxony, and certain departments of France. It is very prevalent in Siberia and in the western part of Asia. It occurs not only in domesticated animals, but also in deer, reindeer, buffaloes, and even elephants. It also affects swine. It is easily inoculable upon rabbits and guinea pigs. Carnivorous animals are much less susceptible of it; but cats are more so than dogs.

Infection.—Shepherds, farm laborers, and other persons who come into contact with living animals affected with anthrax, seem seldom, or never, to take the disease by mere *contagion*. But veterinary surgeons may be infected in performing venesection, and slaughterers in killing or in skinning; the

* [There is good reason to disbelieve this alleged transformation of the bacillus of hay into a septic bacillus. See Prof. Klein's Report to the Local Government Board, "On the Relation of Pathogenic to Septic Bacteria, as illustrated by Anthrax Cultivations." (Blue Book for 1882.)—Ed.]

probability is that a slight abrasion or scratch upon the skin then becomes directly inoculated with the virus.

Another way in which anthrax may arise in man is as the result of *eating the flesh* of an infected animal. Leube, of Jena, has recorded an instance in which the imperfectly-cooked liver of a diseased goat conveyed it. Thus the human gastric juice seems not necessarily to destroy the virus, as has been found to be the case with the gastric juice of carnivorous animals, such as the dog. Thorough cooking probably removes all danger, but there still remains the risk of injury to the butcher, and to those who are concerned in preparing such meat for the table; and this is sufficient reason why the use of it, as food, should be prohibited. According to Hensinger, the disease may even be conveyed by milk or by butter.

But by far the most important cause of anthrax in man is infection *from the dried skins or hair* of diseased animals. In this way it is brought straight from Asia, or, perhaps, from South America, to English workmen.

At Guy's Hospital cases are of not infrequent occurrence among the men engaged in the Bermondsey leather trade, and especially among those employed at a particular wharf on the river side where foreign hides are unshipped. Broca, years ago, remarked how often anthrax was set up by carrying skins upon the shoulders. It is remarkable that the danger appears to be greater when the hides are first brought ashore than afterward.

The same thing has been observed at Bradford, where a peculiar *Wool Sorters' Disease* has for many years prevailed, and this has recently been shown to be identical with anthrax. The source of infection in that case is chiefly "Van mohair," a material which contains much putrid matter and many "fallen fleeces," torn off the bodies of dead animals. Dr. Bell, who gave an excellent account of the affection in the "*Lancet*" for 1880, remarked that the men who are attacked are chiefly the "bag men,"—that is, those who open the bags in which the hair is packed, shake it out, and sort it into different qualities. The reason is, no doubt, that the virus is in a dried state, and that as soon as it is disturbed it diffuses itself in the air and is inhaled into the lungs. Perhaps it may actually have multiplied itself during the transmission of the bags from Asia Minor; for, according to Dr. Bell, the hairs are not known to communicate disease to persons who handle them in the country where they are grown. Indeed, no form of anthrax so severe as the Wool Sorters' Disease had hitherto been described as occurring in man. The work of sorting is said to be free from danger, if the contents of the bags are first washed, or even moistened with water.*

Other occupations which sometimes lead to infection with anthrax are the picking of Russian horse hair (as reported by Dr. Russell, of Glasgow, in 1879), paper making, and the manufacture of coarse woolen hats.

Varieties of Anthrax.—There are several forms of this disease in man.

1. The most common form is that which has long been known as *Malignant Pustule* or *Charbon*. This consists in the formation of a vesicle, seated upon a more or less brawny base. Being usually produced by the inoculation of a scratch or slight sore, it occurs chiefly on the face, the neck, the arms, or some other exposed surface. There is first a period of incubation, lasting generally some days, but sometimes only a few hours. A slight pricking or burning sensation is then felt, which often leads the patient to think that he has just been stung by an insect. A papule quickly appears, and soon passes into a transparent vesicle, which may reach a considerable size. This ruptures and dries up into a

* [See Reports to the Local Government Board by Mr. John Spear; "On Wool Sorters' Disease," 1880, and "On Anthrax among Persons engaged in the London Hide and Skin Trades," 1883.—Ed.]

dark-colored scab. Round it there sometimes arise smaller vesicles arranged in a ring. Meanwhile the base of the vesicle becomes indurated, and a red or purple areola is developed round it. A brawny œdema quickly spreads over the adjacent parts, affecting, perhaps, the whole of one arm or of one side of the neck. Sometimes inflammation of lymphatic vessels occurs, and the corresponding glands become swollen.

During the early part of this morbid process the health remains unaffected. The patient may continue at his work, and at the most complains of slight malaise and of febrile disturbance. But after about forty-eight hours severe pyrexia often sets in, with delirium, prostration, diarrhœa, sweating, and acute pains in the limbs; and death, preceded by a state of collapse, may occur between the fifth and the eighth days. In one case which occurred at Guy's Hospital, the original vesicle had been situated an inch and a half behind the left angle of the lower jaw, and respiration became so difficult toward the last that recourse was had to tracheotomy. I made an autopsy, and found that brawny infiltration had extended deeply to the fauces, so that the entrance of the larynx and its interior were greatly swollen. In other instances the immediate cause of death is septicæmia or pyæmia. Or there may be coexistent anthrax of the intestine, or of the lungs.

So far as I know, the only disease liable to be mistaken for anthrax affecting the skin is that which, occurring on the face, has been especially noticed by Sir James Paget under the name of *facial carbuncle*. It presents no definite vesicle or central scab; and in the instances which I have seen, the veins of the face have been found obviously plugged with puriform thrombi, which has not been the case in anthrax.

It must, however, be added that Bollinger and other writers described, under the name of *anthrax œdema*, a modification of the affection, in which there is neither vesicle nor eschar, but only a pale yellowish or greenish swelling of the subcutaneous tissues. The eyelids are the most frequent seat of this variety.

The recognition of the ordinary external form of anthrax is of extreme importance, because surgical *treatment* at an early stage is capable of arresting its progress. The disease is not, indeed, necessarily fatal, even when left to itself. Sometimes the central part of the swelling sloughs out, the surrounding induration subsides, and an ulcer is left, which more or less quickly heals. I do not know that any statistics exist as to the proportion of cases in which such a spontaneous cure has resulted; but it is, perhaps, worthy of mention that, among nine instances recorded, in 1863, by Dr. William Budd, of Clifton, eight ended fatally. On the Continent, it has long been the practice to destroy the local lesion as soon as it is brought under the eye of a surgeon. Bollinger cites the experience of two observers who lost only thirteen cases out of one hundred and forty-two; another had still greater success, since among two hundred and nine patients all but eleven recovered. In 1878, my colleague, Mr. Davies-Colley, recorded in the "*British Medical Journal*" two cases, in each of which recovery took place after excision of the entire mass of indurated tissue, with the application of chloride of zinc paste to the wound. Contrasting with these is a case which was left alone, and which ended fatally. Several other successful cases have occurred at Guy's Hospital since that time. These have been recorded, together with an analysis of the Guy's Hospital cases, by the same writer, in the "*Med.-Chir. Trans.*," vol. lxv, 1882. The rapidity with which the surrounding œdema subsides after the operation is often very striking. It may be effectual even when the case is at an advanced stage, and when severe general symptoms are present. But under such circumstances quinine should be given in large doses, and the patient's strength must be

kept up with nourishing food and with stimulants. Carbolic acid should also be administered internally, for Bollinger says its therapeutical value in the lower animals when affected with anthrax has been proved beyond doubt.

2. Another form of anthrax affects the *gastro-intestinal mucous membrane.* This has been recognized only within the last few years, and chiefly by German pathologists and physicians. It is sometimes associated with an external malignant pustule; a case in point, in which Dr. Goodhart made an autopsy, occurred at Guy's Hospital, in 1877. Otherwise its symptoms seem to be obscure, so that a correct diagnosis is not likely to be made during life, unless the patient is known to have been exposed in some way to the virus of the disease. However, Bollinger, in Ziemssen's "Handbuch," gives the following account of the symptoms: The patient first complains of malaise, loss of appetite, pains in the limbs, giddiness, and headache. Then vomiting may set in, and a more or less severe diarrhœa, the evacuations often containing blood. There may be pain in the abdomen, which becomes somewhat tumid. Dyspnœa and lividity appear, with restlessness, and with excitement or stupor. Epileptiform convulsions may occur, the upper limbs may be affected with tetanic spasms, there may be opisthotonos, and the pupils may be widely dilated. The pyrexia is but slight, and death is preceded by extreme collapse. The duration of the disease is usually from two to seven days, but sometimes it is scarcely twenty-four hours.

On *post-mortem* examination the abdominal cavity is found to contain a moderate quantity of serous fluid which is often blood stained. The lining membrane of the stomach and intestines shows patches of swelling, generally of the size of lentils or coffee beans, but sometimes one or two inches in diameter. These, on section, are seen to consist of a pink, fleshy infiltration of the mucous and submucous tissues, so that the valvulæ conniventes and other ridges appear firm and prominent; the surface of the affected parts is more or less excoriated and discolored or covered with an adherent layer of extravasated blood. There are also spots of ecchymosis on both the serous and the mucous aspect of the gastro-intestinal tract. The mesenteric and the lumbar lymph glands are often greatly enlarged, and of a dark red color. The mesentery may form a large, brawny swelling, and the connective tissue in front of the spine may be infiltrated in a similar manner. The spleen is softened to a pulp, but it is not generally much enlarged.

Hitherto, so far as I am aware, no instance of recovery from intestinal anthrax has been recorded. Leube, however, has related a case in which the lower lip, the inside of the cheek, and the hard palate presented indurated patches of a bluish-red color; epistaxis and hæmaturia appeared, but the malady ended favorably in about a fortnight. The patient, whose name was Schmidt, took fifteen grains of carbolic acid and thirty grains of quinine each day, and the patches in the mouth were cauterized three times daily with carbolic acid. But I must confess to a doubt whether the diagnosis of anthrax was established beyond question. Schmidt had been admitted five weeks before with pyrexia, which proved to be due to latent tuberculosis; and for one night he was placed upon a mattress on which a patient with anthrax, named Werner, had been lying for twenty-four hours about three days previously. As so long an incubation as five weeks could hardly be supposed to have occurred, Leube suggests that the virus was perhaps taken up by, and remained for a time attached to, some article of property which Schmidt is not unlikely to have hidden in his bed during that first night, and which may thus have come directly into contact with dried stains of the bloody fluid that had been expectorated by Werner. It is said, indeed, that the microscopical examination of the blood in

Schmidt's case proved that he really was suffering from anthrax; but to this question I shall allude further on.

3. A third form of anthrax affects mainly the *thoracic viscera*. It has hitherto been recognized chiefly among the wool sorters of Bradford. After Dr. Bell, of that town, had drawn attention to it, Mr. Spear and Dr. Greenfield investigated it for the Local Government Board. Dr. Greenfield's description of it is briefly as follows: The earliest symptoms are great prostration and a sense of oppression of breathing. Shivering seldom occurs. The respiration is not much accelerated, but it is labored and difficult, with a feeling of pressure or constriction. There may be more or less abundant bloody expectoration, or none at all. Auscultation seldom reveals anything more than slight rhonchus. The face is sometimes congested, sometimes pale, with a slight cyanotic tint. The extremities are cold and bluish; even in the axilla the temperature may be subnormal; but in the rectum the thermometer may rise to 102° or 103°. The pulse is rapid and weak and sometimes irregular. There may be nausea and vomiting, but not generally diarrhœa. In many cases the appetite remains good and digestion seems to be unimpaired. There is sometimes a sort of hysterical condition, or a state of mental anxiety and depression; but other patients have been so unconscious of danger as to refuse to send for medical advice until the last few hours of life, or have even attempted to work. Death may be preceded by delirium, convulsions, or coma, or the mind may be clear to the last, and the end come suddenly and unexpectedly. One of Dr. Bell's patients lived only seventeen hours after he was first taken ill. Many cases have terminated fatally in from three to five days. Dr. Bell says that those who survive for a week generally recover.

The *post-mortem* appearances vary considerably. Decomposition appears to be somewhat rapid. The lung tissue may either be congested or quite natural. There may be blood-stained secretion in the bronchial tubes, and their mucous membrane may be ecchymosed. Sometimes the pleuræ contain a pint or two of fluid, and the lungs are partially collapsed in consequence. There is often blood-stained, serous or gelatinous infiltration of the mediastinal tissues, and the bronchial glands are swollen, softened, and ecchymosed. The pericardium may be marked with hemorrhages, and may contain a blood-stained fluid; or a large quantity of blood may be poured out between it and the sternum. Sometimes the pharynx and the adjacent tissues are infiltrated or ecchymosed. There may also be ecchymoses in the pia mater, in the kidneys, and elsewhere. It is remarkable that the spleen is hardly, if at all, softened. In prolonged cases hemorrhagic infarctions may be found in the lungs, attended with slight pleurisy; or there may be patches of broncho-pneumonia.

Obviously, there may sometimes be considerable difficulty in distinguishing, by clinical evidence alone, this form of anthrax from a severe attack of ordinary pulmonary inflammation. Indeed, until recently, fatal cases of the Wool Sorters' Disease at Bradford were commonly registered under the head of Pneumonia, Bronchitis, or Congestion of the Lungs. And, on the other hand, it can hardly be doubted, as Dr. Bell suggests, that a similar affection will hereafter be found to occur in many towns where carpet and blanket wools, dry hairs or furs, are used for manufacturing purposes.

Certain points in regard to the distribution of the bacilli in animals or men affected with anthrax have still to be mentioned. They have an important bearing upon the theory of infective maladies in general, because they illustrate what is perhaps a universal law, namely, that the microzymes of contagion multiply locally before they infect the blood to any considerable extent. In the ordinary form of the disease affecting the skin, the bacilli

may be found, according to Davaine, about the second or the third day, in clusters embedded in the rete mucosum at the centre of the vesicle. E. Wagner has since found that they are at this time so closely packed in the papillæ of the cutis as to conceal all the tissue elements. Thence they spread both laterally and toward the deeper structures, enter the vessels, and are carried all over the body with the blood. In the gastro-intestinal variety the bacilli infiltrate all the swollen and œdematous tissues, so that Buhl and others described this affection under the name of *mycosis intestinalis* before they recognized that it has any relation to anthrax.

At an early stage of the disease the blood may contain no bacilli. Thus, if in a doubtful case one fails to discover them in this fluid, one must not attach much importance to this result. On the other hand, the presence of well-marked rods is conclusive; but it does not seem to be sufficient to find round bodies only, as Leube did in the case of the man Schmidt, referred to at p. 339. In man, even when a fatal termination is approaching, the bacilli seem to be seldom found in such immense numbers, and so generally distributed in the blood, as they are in animals. Davaine calculated that from eight to ten millions are present in a single drop of a diseased beast's blood. In Buhl's first case of intestinal anthrax they are said to have been present in the blood of the portal vein, but not in that of other vessels. They may often be detected in the substance of the spleen and of the solid tissues generally, and also in the fluid poured out into the serous cavities.

The surest way of arriving at a diagnosis of a suspected case of anthrax is often to inoculate a rabbit or a guinea pig, or even a mouse, with fluid from the part primarily affected, or with blood. Such small animals commonly die in two or three days, or even still more rapidly, with dyspnœa, dilatation of the pupils, and perhaps convulsions. Their blood may then be seen to swarm with bacilli. However, if the experiment of inoculation is followed by a negative result, a negative conclusion is not warranted.

Foot and Mouth Disease.—In striking contrast with the dangerous epizoötic diseases hitherto mentioned is one which in cattle seldom causes more than a transitory illness, and which, when communicated to man, generally produces effects so trifling that they are not brought under the notice of a physician. This is what is termed the "Foot and Mouth Disease," or sometimes "*aphtha epizoötica*." Some writers have unwisely appropriated to it the mediæval name of *murrain*, which is so familiar from its being used in the English translation of the Bible. On the Continent the complaint in question has been known for centuries, but it is said to have been first introduced into England in 1839. In 1869 it prevailed here very extensively, and in 1871 it is said to have affected as many as 700,000 animals. It occurs chiefly in cattle and in sheep; pigs are also liable to it, but probably only as the result of infection by feeding upon the milk of diseased cows. It is very rarely seen in horses.

In cattle, the foot and mouth disease is attended with a moderate degree of pyrexia. One of the most marked symptoms is the formation of vesicles and bullæ upon the mucous membrane of the mouth, including the lips and the tongue; these parts also become swollen, and there is a dribbling of saliva. The vesicles soon break, and their floors become covered with a thick gray layer. A somewhat similar eruption appears upon the feet, round the borders of the hoofs, and in their clefts. The vessels then pass into pustules and dry up into crusts. They give the animal pain in walking or standing, so that it generally remains lying down. Another seat for the vesicles is upon the udder and the teats, which may in consequence become quite raw and excoriated. But within twelve or fourteen days all the effects of the complaint pass off. It almost always ends in recovery, except in calves,

of whom from 50 to 75 per cent. die, probably from the milk yielded by their mothers (when they also are affected) having an irritant action upon the alimentary canal, besides conveying the specific virus.

After what has been said of the extent to which the foot and mouth disease sometimes prevails, one may well be surprised that, if capable of transmission to man at all, it should not be frequently transmitted. But in the "*Brit. Med. Journal*" for 1867 will be found a list of only twenty-two notices of such an occurrence, which were all that Prof. McBride could find recorded during the previous thirty years; and most of these were taken, not from medical journals, but from the "*Veterinary Review.*" It might, therefore, be supposed that human beings are but little susceptible of it. But if so, it is curious that two foreign observers, Hertwig and Jacob, should each have readily succeeded in infecting himself experimentally by drinking the milk of diseased animals.

There are two ways in which the complaint may be communicated to man. One is by the direct *inoculation* of the specific virus into a crack or sore place, generally on the hand or on a finger. This is specially apt to happen to a person engaged in milking a cow with the eruption upon the teats; but sometimes saliva or fluid from the mouth conveys the contagion to a veterinary surgeon who is attending to the sores there; and a butcher is said to have taken it by holding his knife between his lips while dressing an infected carcass.

The other way is by *drinking the milk* of a diseased cow. This is far more important, not only because it concerns children, who suffer much more than adults, but also because it is very likely to be overlooked, at least by persons who are not conversant with the symptoms observed under such circumstances. As to the characters of the milk yielded by cows suffering from the foot and mouth disease, there have been some discrepancies of statement. Sometimes it is offensive to the smell and the taste, and it may even be mixed with blood or pus in considerable quantity; but this seems to be only when the teats are severely excoriated, or when the mammary glands themselves have become inflamed as the result of an undue retention of their secretion. In other cases it is less obviously altered; and then one of its most marked peculiarities is a tendency to coagulate when boiled, or even at a much lower temperature, forming innumerable little flocculent masses which float in a bluish whey. Sometimes it seems to have a yellowish tint, and when examined microscopically displays granular cells like those of the colostrum. But sometimes it is said to have a perfectly natural appearance. It seems generally to be diminished in quantity by about one-half. There is no doubt that it is very often drunk with impunity, not only by adults, but even by children. In any case boiling may be assumed to render it innocuous.

The *incubation* of the "foot and mouth" disease in man is said to be from three to five days. At the end of this time slight pyrexia arises, with loss of appetite. A sensation of dryness and heat in the mouth is then noticed, and vesicles quickly appear upon the inside of the lips, on the tongue, and sometimes upon the fauces and hard palate. They reach the size of peas, their contents become opaque and yellowish, and in from one to three days they break, forming shallow, dark-red ulcers. There is some pain, which is increased by mastication, by swallowing, and by talking. The lips become swollen, and mucus and saliva are poured out in excess. In a case related by Mr. Briscoe, of Chippenham, in the "*Brit. Med. Jour.*" for 1872, the tongue swelled until it protruded for two inches and a half outside the mouth, and it was so firmly wedged between the teeth that for thirty-six days the patient took no food except milk; there was great dyspnœa, and suffocation appeared at one time imminent; sloughs peeled off the tongue, and

the discharge became very fetid. Such a condition, however, is altogether exceptional. There is often slight disorder of the digestive organs, indicated by diarrhœa and abdominal pain.

In some patients an eruption appears upon the fingers, especially round the nails. It consists of small, clear vesicles which pass into pustules, and sometimes run together. It very rarely happens that the inflammation is sufficiently intense to cause shedding of the nails. The feet are seldom affected, but sometimes vesicles have been observed between the toes. Mr. Amyot ("*Med. Times and Gaz.*," 1871) mentions the case of a woman in whom the feet "became hot and covered with painful tubercles." It is said that the female breasts have occasionally presented vesicles, and even the face and other parts of the body. In Mr. Hislop's cases ("*Ed. Med. Jour.*," 1863), in which there was a red, scaly rash upon a farmer's forehead, and upon the body and lower limbs of his wife, cured by bichloride of mercury and iodide of potassium, I must confess that it seems to me doubtful whether foot and mouth disease was really present, or syphilis.

The eruptions, however, for which the foot and mouth disease seems most likely to be mistaken are varicella and slight smallpox. The affection of the lips and tongue might be set down as a simple aphthous stomatitis. Indeed, it is not impossible that a slight catarrhal inflammation of the mouth, with a little soreness of throat, may be a far more common result of infection by diseased milk in infants than has hitherto been suspected. A doubtful case might always be cleared up by the inoculation of a sheep, but the animal must be known not to have had the disorder already.

The *duration* of the foot and mouth disease in man is usually from ten to fourteen days, but sometimes not more than a week. A case fatal by septicæmia has been recorded, but with that exception death has occurred only in very young children.

The only *treatmeni* required is the application of a solution of borax to the mucous membrane of the mouth, or of the solid nitrate of silver to any particularly painful ulcers; the eruption on the fingers may be dealt with like a mild eczema.

DISEASES OF THE NERVOUS SYSTEM.

GENERAL INTRODUCTION.

PECULIARITIES OF THIS GROUP OF DISEASES—ORDER FOLLOWED—ELECTRICITY AS AN INSTRUMENT OF DIAGNOSIS.

In entering upon the study of Nervous Diseases, we find ourselves face to face with the most difficult department of regional pathology. Unlike the lungs and heart, the brain works silently, so that no auscultatory phenomena accompany its disorders. From the abdominal viscera it differs in being beyond the reach of palpation and percussion, and in pouring forth no secretions to be submitted to physical or chemical examination. Hence, *the symptoms of nervous disease are, almost without exception, those of perverted nervous function.* There is, indeed, the same broad line of demarcation, as in the case of affections of other organs, between those which appear to be wholly functional, and those which depend upon organic changes; and for the former we have a special name, that of *Neuroses.*

I shall not, however, now find it convenient to take the various diseases in a regular ascending order, beginning with those in which no lesion of structure exists, and passing gradually to those which depend upon lesions of greater severity. For some of the most destructive affections, *e. g.*, of the brain, present a very simple series of disorders of function, while some of the most trifling neuroses are attended with very complicated symptoms. I shall, therefore, deal first with the former, in such an order as may appear most conducive to their easy comprehension.

Again, one cannot absolutely separate the diseases of the nervous system into those which affect respectively the brain, the spinal cord, and the nerves. For, first, these are disorders of which the precise seat is still obscure or doubtful; and, secondly, there are affections which, although they begin in one of these great divisions of the nervous apparatus, afterward involve the rest. Still, the arrangement which I shall adopt will, as far as possible, be that of taking first the diseases of the peripheral nerves, and successively ascending to those of the spinal and of the cerebral centres.

Before, however, I pass on to consider these special subjects, it will be convenient that I should make some preliminary observations with regard to the normal relations of the nerves and muscles to electrical currents. For the conditions under which alone such currents can generally be applied to living human beings make it impossible for us to imitate strictly the methods employed by the physiological laboratory.

The electrical instruments used in medicine at the present day are chiefly of two kinds. One consists of a galvanic battery of from ten to fifty cells,

arranged in a single series; this yields what is termed a *primary*, "continuous" ("constant"), or "Galvanic" current; and the method of applying it is called *galvanizing*. The other is the common induction apparatus, which may be constructed upon various principles; the currents obtained from it are known as *secondary*, "induced," or "Faradic;" and the process of applying them is called *Faradizing*. In both cases, the poles of the instrument are generally made to terminate in sponges, or covered metals moistened with a weak solution of salt. These are placed upon the surface of the skin at some distance from one another; the skin itself should be made thoroughly wet, and it may often with advantage be soaked by means of water dressings for an hour or two before, since this increases its conducting power, and makes it more easy for the current to penetrate to the muscles. On the other hand, when one wishes to act only upon the sensory nerves of the skin, one employs a dry brush of very fine metallic wires as the terminal which is to be applied to the affected part; and the surface is carefully dried and has some vegetable powder, such as flour, dusted over it. The poles may either be kept stationary, or moved from spot to spot; one of the most usual methods is to stroke the skin gently with one of them; when this is done the current is said to be "*labile*" or varying, while it is called "*stabile*" or continuous, when each pole is kept applied to a single spot.

In healthy persons the effects of *Galvanizing* differ, not only with the strength of the currents employed, but also according as one or the other pole is applied over particular structures. To make the matter as simple as possible, the practice is to place one pole upon some indifferent part (such as the sternum or spine), and the other over a certain muscle or nerve, the reaction of which is to be tested. At first a very weak current is used, and its strength is gradually increased until the desired effect is produced. Now if the *negative* pole (or *cathode*) be the one applied over the muscle or nerve (descending current), it is found that contractions of the muscle—or of the muscles supplied by the nerve—occur sooner (that is, with a more feeble current) than when the *positive pole* (or *anode*) is placed in the same position; and it is also found that they occur only at the moment when the pole is brought into contact with the skin ("make" or "close"), and not at the moment when it is removed ("break" or "open"). A technical way of expressing this result is to say that the muscular contractions caused by a feeble current are obtained at the time of closing the current, and at the cathode; and German writers represent it by a formula of which the natural English equivalent* is C.C.C. (Cathode, Closing, Contraction). With a current of greater power, the *positive* pole or *anode* begins to cause contractions; as a rule, these seem to occur rather when the pole is withdrawn from the surface than at the moment of its application; but sometimes the reverse is the case. These results are represented by the formulæ A.O.C. and A.C.C. respectively (Anode Opening Contraction and Anode Closing Contraction). Lastly, it is only when a still more powerful current is employed that the negative pole causes contractions at the time of opening (C.O.C., or Cathode Opening Contraction). It is to be understood that as the strength of the current is increased, the contractions which can be excited by the more feeble currents are still producible, and with augmented intensity. Thus the cathodal closing contraction (C.C.C.) grows more and more powerful until it becomes "tonic," *i. e.*, continuous, at about the time when cathodal opening contraction (C.O.C.) is obtained; and for such a condition the new formula C.C.T. (Cathodal Closing Tetanus) is sometimes employed. The results will perhaps be more intelligible to the reader if they are placed in a tabular form, it being understood that variations in the energy of the contractions are roughly represented by letters of different sizes.

*These formulæ will be found more fully explained in Dr. Poore's work.

	CC	AO	AC	CO
Weak current causes.....	c			
Strong current causes.....	C	c	c	
Very strong current causes.....	T	C	C	c

Another effect of galvanization is the production of redness of the skin, more marked at the cathode; and, if it be continued for a sufficient length of time, inflammation, or even ulceration, may be caused by it. The application of the current also gives rise to a disagreeable sensation, of a pricking or burning character, which seems to be equally marked at the two poles, but which may not be perceptible when the current is first applied.

The effects of *Faradization* upon healthy persons are much less complicated than those of the application of the continuous current. When the faradic instrument is used, there is no question of the action of separate poles, nor of closing or opening the circuit. Its construction is such that the poles are reversed many times a second, and contact is as often made and broken. There is, therefore, no need to move either pole; for the "breaks" and "makes" are made by the magnetic interrupter.

All that need be said about the normal effects of this mode of applying electricity is that both the muscular contractions and the sensations of tingling or pricking or burning to which it gives rise go on increasing in intensity as the strength of the currents is augmented. The usual method of employing faradization is to place the two poles, a few inches apart, over the nerves or muscles the reaction of which is to be tested.

AFFECTIONS OF THE NERVE TRUNKS.

Peripheral Paralysis.—CAUSES—MOTOR PALSIES OF VERTEBRAL NERVES—THE REACTION OF DEGENERATION—SENSORY PARALYSIS—CAUSALGIA—GLOSSY SKIN—DIAGNOSIS, PROGNOSIS, TREATMENT—PARALYSIS OF CRANIAL NERVES—MOTOR, OF THE PORTIO DURA, HYPOGLOSSAL, AND OCULO-MOTOR—SENSORY OF THE TRIFACIAL AND OLFACTORY.

Neuralgia.—PATHOLOGY AND GENERAL CHARACTERS—TRIFACIAL NEURALGIA, ETC.—SCIATICA—TREATMENT OF NEURALGIA.

Zona.—A NEUROSIS—THE ERUPTION—SYMPTOMS—NEURALGIA—TREATMENT.

The affections of the nerve trunks may be broadly divided into two groups. In one the chief symptoms are *loss of muscular power and of cutaneous sensibility;* these are commonly called the *Peripheral Paralyses*, to distinguish them from similar conditions due to diseases of the brain or spinal cord. In the other group, which, pathologically, is of far less severity, *pain* is the principal symptom; these constitute the *Neuralgiæ.*

PERIPHERAL PARALYSIS.—Many points in regard to the causes and symptoms of the paralyses due to lesions of the nerve trunks are the same, whatever parts of the body may be their seat. It is convenient to take the nerves of one of the limbs by way of illustration, and I will choose those of the arm, because the movements performed by its several muscles are more distinct and definite than those of the individual muscles of the leg, and their functional integrity is accordingly more easy of determination.

Injuries of the *brachial plexus* may be followed by loss of power and of sensation in the whole upper limb. The surgeon sees cases in which this occurs as the result of severe accidents in which the shoulder joint is dislocated, or the humerus or scapula fractured. But similar symptoms not infrequently follow simple pressure upon the nerves, or an injury so slight that its occurrence, especially in a child, may be altogether overlooked. Thus Paget ("*Med. Times and Gaz.*," 1864) mentions the case of a boy who, when an infant, had had his left arm violently pulled by a little brother; the limb appeared altogether powerless for a long time afterward, and it remained permanently weak and much smaller than the other one. The employment of force to reduce a dislocated shoulder has sometimes been attended with like consequences: and other causes which have been noted are the pressure of a crutch which is too long or insufficiently padded; having the arm forcibly compressed by a cord (in some countries prisoners are injured in this manner); and among the water-carriers of Rennes the carrying of certain large vessels with handles through which the arm is passed. "Crutch paralysis" is not uncommonly seen in hospital practice in London; and the patient is often unaware of its cause. A similar effect may also be produced by lying on one side, with the weight of the body resting upon the arm; or, more often, by sleeping with the arm across the back of a chair. But in most instances of this kind, the sensibility of the patient has been blunted by some narcotic poison, or at

least by alcohol; and they are sometimes spoken of as cases of "Sunday morning" paralysis, on account of the frequency with which Saturday night is chosen by the lower classes for indulgence in drink. When the cause is doubtful, the axilla and neck must, of course, be carefully examined, lest an aneurism, exostosis, or some other tumor should be present. Some writers believe that the brachial plexus is sometimes affected by external cold, as we shall presently see to be the case with the facial nerve; but this appears to be doubtful. Mr. Salter has related ("*Guy's Hosp. Rep.*," 1868) two cases, in each of which caries of a wisdom tooth, besides causing severe pain in the arm, led also to a partial paralysis of the muscles (of the nature of a "reflex" paralysis, as I shall hereafter have to define that term), so that the patients could not grasp objects with the hand, nor raise the hand to the head, nor employ it in dressing, nor hold a fork at dinner. In one instance all the symptoms vanished as soon as the tooth had been extracted and the pain of the operation had passed off; in the other instance a similar result occurred a few hours after the same treatment.

It is comparatively exceptional for the whole of the brachial plexus to be paralyzed at once by any of the causes that I have been mentioning; and the loss of power may often be limited to the muscles supplied by a single nerve. In such cases the interpretation of the symptoms may be so obvious that no one acquainted with the rudiments of anatomy could possibly misunderstand them. But sometimes they are of a more recondite nature, and require a somewhat careful study.

Thus particular interest attaches to paralysis of the *serratus magnus* muscle, supplied by the *posterior or long thoracic nerve*. That nerve, indeed, leaves the plexus at so high a point that it is never involved when the cause is the pressure of the crutch, or sleeping upon one arm. But it may be affected by a blow upon the shoulder, or any other severe injury to that part; by carrying a heavy weight upon the shoulder; or by over-exertion of the shoulder muscles, as in the case of mowers, puddlers, cobblers, or rope makers. Sleeping on damp ground is also said to have given rise to it, and even exposure to draughts. The principal symptom is an alteration in the position of the scapula. Even when the shoulder is at rest, with the arm hanging down, the scapula, as a whole, is drawn slightly upward and inward, and has its inferior angle tilted, so as to be nearer to the vertebral column than the rest of its posterior border. But what is especially characteristic is that this posterior border stands off a little from the part of the back on which it should rest, forming a "wing-like" projection. These peculiarities are greatly exaggerated when the patient is told to perform certain movements. Thus, if he is directed to lift his arm straight over his head, he finds himself unable to raise it above the level of the shoulder, because the necessary rotation of the scapula by the serratus cannot be effected; but when once another person has moved the bone to the proper position for him and fixed it there, he acquires the power of lifting the limb to whatever height may be desired. During the attempt to raise the arm, the scapula, if left to itself, moves further than ever toward the spine; and where both serrati happen to be paralyzed together, the two bones may actually come into contact at their inferior angles. If the raised arm is brought forward, the "wing-like projection" becomes greatly augmented, the posterior border of the scapula standing off so as to leave a deep hollow, in which one can place one's hand. Another symptom, according to Dr. Poore ("*Clin. Soc. Trans.*," viii), is an alteration in the shape of the chest on the affected side, as shown by the cyrtometer. He finds that in a healthy person, when the arms are thrown horizontally forward, the chest becomes flattened in front, its lateral diameter being increased. When there is paralysis of one serratus, the widening fails to occur on that side.

An affection of more frequent occurrence is paralysis of the muscles supplied by the *circumflex* nerve, among which the *deltoid* is the chief. This, too, may result from direct injury to the shoulder. According to Erb, it is sometimes caused by "rheumatic" affections of the articulation. One must, however, bear in mind that osteo-arthritis (to which disease Erb doubtless refers) is commonly attended with destruction of the long tendon of the biceps, and that unless this structure is perfect the arm cannot be raised; and inability to lift the arm from the side is also the principal symptom of paralysis of the deltoid. It may be thought that a sufficient proof of the existence of paralysis would be found in the presence of atrophy of the muscle, which, indeed, is often so marked that a deep groove is formed between the acromion and the head of the humerus. But a considerable degree of wasting may be the result of disuse when the joint is diseased.

Paralysis of the *musculo-cutaneous* nerve is, of course, indicated by inability to flex the elbow joint, the biceps and brachialis anticus muscles being paralyzed. It must be borne in mind that the supinator longus (which receives its supply from another source) is also a flexor, and the patient must therefore be told to keep his hand supinated at the time when the power of the other muscles is being tested.

Among the nerves which send filaments to the hand and fingers, the *musculo-spiral* is much more apt than the other two to be separately affected. In a considerable proportion of the cases of paralysis due to the pressure of a crutch, or to lying on one side during the deep sleep of intoxication, the musculo-spinal nerve suffers alone; and as in the latter case the triceps often escapes, it is probable that the nerve undergoes compression where it is winding round the humerus, and after it has given off the branches to that muscle. The extensors of the wrist, of the two joints of the thumb, and of the metacarpo-phalangeal joints of the fingers are the chief other muscles supplied by this nerve; and accordingly one symptom of paralysis of it is a flexed condition of these joints, with inability to straighten them; the two remaining joints of the fingers being nevertheless capable of extension by the interossei and lumbricales, provided that the metacarpo-phalangeal joints are held up by another person's hand, or in some other way. Another consequence of the loss of power in the extensor muscles of the wrist joint is impairment in the extent to which the fingers can be *flexed* upon the palm. Thus there is no other nerve the paralysis of which interferes so much with the motions of the hand as does that of the musculo-spiral nerve.

An isolated paralysis of the *median* or of the *ulnar* nerve is more commonly due to a wound or injury affecting it at the elbow, in the forearm, or at the wrist, than to any morbid state of the brachial plexus above. The symptoms, in the case of the *median* nerve, are an inability to use the flexors of the wrist and fingers, the metacarpo-phalangeal joints being alone capable of flexion, by means of the interossei. Indeed, the excessive action of these muscles is apt to cause an over-extension of the two phalangeal joints of the fingers, and especially of the index finger.

On the other hand, when the *ulnar* nerve is paralyzed, the muscles of the little finger and the interossei are more markedly affected than any others. The fingers cannot be abducted nor adducted, and the patient is unable to flex the metarcarpo-phalangeal, or to extend any of the phalangeal joints of the fingers. There being nothing to antagonize the traction excited by the common extensor and flexor muscles, the joints in question become distorted, so that the two last phalanges are over-flexed, while the first phalanges, on the other hand, are over-extended. Accordingly, the prominences of the knuckles are replaced by hollows on the back of the hand, while they project toward the palm, and are the more noticeable there because the inter-

ossei and two of the lumbricales muscles are atrophied. The fingers of a hand so affected are often compared to the claws of a griffin; French writers speak of it as the "*main en griffe.*"

The production of deformities, in affections of the nerve trunks, by contraction of the muscles which are physiologically the antagonists of those that are paralyzed, has not before been alluded to; but it may occur in every form of these affections, and often complicates the symptoms very considerably. On the other hand, at very advanced periods of such cases, the paralyzed muscles themselves may waste and shorten, so as to produce distortions of precisely opposite characters.

It by no means necessarily follows, in all cases of peripheral paralysis, that the anatomical distribution of the nerves is strictly adhered to. Erb speaks of having seen several instances in which the deltoid, biceps, brachialis anticus, and supinator longus were chiefly, if not exclusively, attacked; and he throws out the suggestion that this depends upon an affection of the sixth cervical nerve where it is emerging from between the scaleni. In such patients the arm hangs by the side, and the elbow cannot be flexed. It is remarkable that a precisely similar affection has been described by Duchenne in newly-born children, as the result of traction or pressure upon the shoulder during birth. Erb says that he has himself seen two cases of this kind.

Lastly, one must bear in mind that in exceptional cases a morbid process beginning in a single nerve at a particular spot is very apt to pass upward along that nerve, and may afterward extend to other nerves with which it is connected. Thus, Dr. Weir Mitchell relates the case of a man who received an injury to the nerve which supplies the pectoralis major; on the third day he was attacked with pain in the course of the median and musculo-cutaneous nerves, and the muscles to which they are distributed subsequently became paralyzed and wasted. Another patient bruised his ulnar nerve at the bend of the elbow; and about five months afterward the flexor muscles of the wrist became affected with paralysis, those supplied by the median nerve as well as those supplied by the ulnar. In the chapter on Epilepsy, I shall have to describe some still more remote effects of injuries to nerves.

In some cases of paralysis due to lesions of the nerve trunks, the electrical reactions of the nerves and muscles present remarkable modifications. These have been minutely studied by Erb, Ziemssen, and others in Germany; and more recently by several observers in this country. Let us suppose, for example, that a nerve trunk is cut across, or that a small portion of it is crushed in an injury to the upper part of the limb. The result is that throughout the whole length of the nerve below the affected spot, and in its branches (so far as one can test them without at the same time stimulating the muscles), the excitability both to galvanic and to faradic currents undergoes a progressive diminution, until in the course of the second week it is entirely extinguished, so that not the slightest contraction can be produced by the application of either form of electricity to the nerves. But in the muscles the reactions are very different. They, too, fail to respond when a "faradic" current is applied to them. But when an interrupted "galvanic" current is employed, they contract much more readily than under normal circumstances. The method of determining this fact is very simple. We first ascertain what number of cells are required to excite contractions in the muscles of the healthy limb which correspond to those paralyzed; and then, perhaps, find that only half that number is needed to excite contractions in the muscles supplied by the injured nerve. Indeed, they can often be thrown into vigorous action by a current which

is too feeble to give rise to any sensation. Moreover, there is what is termed a "qualitative" change, *i.e.* the order in which the two poles respectively excite contractions is altered. So far from contraction at the moment of closure with the cathode (C.C.C.) being now the most marked effect, there is often as much or even greater contraction when the circuit is completed with the anode; while instead of cathodal opening contractions (C.O.C.) being the last of all to appear, they may precede the anodal opening contractions (A.O.C). Lastly, the contractions themselves are slow and protracted, and resemble tonic spasms rather than the short clonic movements which occur in healthy muscles when stimulated by galvanic currents.

Very little progress has hitherto been made toward the explanation of these remarkable facts. The seeming paradox that muscles insensible to powerful faradic currents should nevertheless respond to very weak galvanic currents has, indeed, been accounted for by Neumann, who has shown that it depends upon their requiring a current to pass for some length of time before they can react to it. Momentary galvanic currents, even when of considerable strength, are found to be as inoperative as faradic currents themselves. But why the muscles should be incapable of responding to currents which are only of brief duration, and why they should be sensitive to weaker galvanic currents than under normal conditions, we cannot at present say.

It seems, however, to be established that the peculiar reaction in question, as well as the loss of electrical irritability in the trunk of the nerve below the seat of injury, are coincident with and indicative of a very remarkable series of degenerative changes which have been shown by several observers to affect the structure of the parts paralyzed. Erb has accordingly proposed to designate as the "reaction of degeneration" the presence of increased galvanic excitability in a muscle, when associated with diminished faradic excitability. Within a few days after the occurrence of any severe lesion of a nerve trunk, the whole length of the nerve below is found to have its fibres greatly altered. Their medullary sheaths break up into fatty granules, and it is even doubtful whether the axis cylinders remain undestroyed. At the same time the neurilemma everywhere undergoes a greatly increased development; first it becomes crowded with corpuscles, and these presently develop into spindle cells and fibres, so that the nerve trunk itself becomes much denser and harder than before. In the muscles, too, similar changes take place, and these have been investigated by Erb. The fibres themselves gradually diminish in size, and their striation becomes indistinct; but their nuclei multiply, and the connective material between them is crowded with cells, which after a time undergo development into fresh fibrous tissue. If the injury to the nerve should remain unrepaired, the muscular fibres waste still further, and at length disappear. Thus the whole substance of the muscles is at last converted into a whitish yellow, flattened mass of dense fibrous tissue, in which no striated fibres can be discovered. It often includes in its interstices a large number of adipose cells.

The cause of these curious changes is at present unknown. The easiest way of explaining them would be to attribute them to the extension of an inflammatory process along the nerve from the seat of injury. But, as Erb remarks, they ought not then to be confined to that part of the trunk which is to the peripheral side of the lesion, nor should they be traceable through a plexus or be accurately limited to certain fibres; nor should they occur simultaneously in distant muscles, or in those which are placed in the upper part of a limb. He believes that in some way they depend directly upon the separation of the affected structures from the nervous centres.

It must not be supposed that the commencement of degenerative changes in the peripheral nerves and muscles is necessarily a proof that the trunk above has undergone permanent and irremediable injury. On the contrary, these structures possess extraordinary powers of regeneration. Even after the muscles have for several weeks exhibited the "reaction of degeneration" it often happens that the connection between their nerves and the spinal cord is at length gradually restored. Erb has noticed in such cases very curious anomalies in the reactions of the nerves and muscles respectively to electrical and other stimuli. Nerves that have completely recovered the power of transmitting volitional impulses may still remain devoid of all excitability by faradic or galvanic currents; or if their regeneration should take place early, they may regain their sensitiveness to both kinds of current before the muscles have ceased to present the "reaction of degeneration." That reaction, indeed, is itself never persistent for any great length of time after the original injury. If the nerve fails to undergo repair, and the muscle fibres begin to disappear, their excitability to galvanic currents quickly begins to diminish, and at length it becomes extinguished; and even when regeneration of the nerve does occur, the muscles commonly display a lessened rather than an increased galvanic irritability while it is going on, and for some time after the normal conditions have in all other respects been completely restored.

On the other hand, it is only when the original lesion of the nerve trunk is of a certain degree of severity that the "reaction of degeneration" presents itself. In the slighter forms of paralysis, such as that due to the presence of a crutch, or to compression of the brachial plexus or musculo-spiral nerve during sleep, both the muscles and nerves often retain their normal irritability both to faradic and to galvanic currents.

Affections of Sensation.—Nerve trunks possess sensory besides motor functions, and these too may be impaired by the same lesions which cause paralysis. Thus, if we still take the brachial plexus by way of illustration, we find that affections of its nerves may completely extinguish the transmission of tactile impressions from all parts of the limb to which those nerves are distributed, and may likewise render the patient unable to feel a prick or a cut, heat or cold, or the passage of a galvanic current. The resulting loss of common sensation is termed Anæsthesia, while the incapacity to feel pain is distinguished as Analgesia. They do not necessarily accompany one another. Dr. Mitchell relates two cases in which, without the slightest impairment of the sense of touch, there was great diminution in the susceptibility to pain, one patient experiencing only a slight pricking sensation when a large needle was run into the palm, and the other being scarcely sensitive to the electric wire brush. In ordinary cases, however, this instrument is a most delicate test for the presence of minute degrees of susceptibility to sensory impressions. As I have stated at p. 345, the part to which it is applied must be perfectly dry. Dr. Mitchell says that if no sensation can be produced by it, one may generally conclude that the loss is absolute, but that on several occasions the use of it has saved him from despairing of cases which had seemed altogether hopeless.

On the other hand, there are all possible degrees of anæsthesia—to a point at which very careful investigation is necessary to show that the transmission of impressions is not perfect. The best ways of detecting a very slight anæsthesia are to bring the tip of a feather gently upon the surface of the skin, or to touch a single hair with a needle or the edge of a knife. If the patient can feel this on one side but not on the other, sensation is to some degree impaired. Or a pair of compasses may be used, as taught by E. H. Weber, the object being to determine at what distance from

one another the points are felt to be distinct. This "limit of separate perception" varies greatly in different parts of the skin under normal conditions, from 2.25 mm. on the extremities of the fingers to 77 mm. on the arm. But for each region there is an average, and any very considerable departure from it may be taken as proof that some perversion of sensibility exists. Or a direct comparison may be made between the opposite sides of the body in the individual whose case is being investigated. An improved form of the instrument is sometimes employed, in which the two limbs of the compasses slide upon a graduated bar; this is called the "æsthesiometer." But experience has shown that it is useless to base accurate numerical statements upon Weber's method. It often happens that widely different results are obtained from the same spot at an interval of a few minutes. Dr. Mitchell points out that, to ensure the most correct results that are attainable, one must cover the patient's eyes, keep the part which is to be tested perfectly at rest, and apply the compass points (which should be rounded) lightly upon the surface of the skin, but with equal force, at exactly the same time, and in a line bearing a definite relation to the axis of the limb.

It would be a great mistake to suppose that in all affections of a mixed nerve trunk the muscular paralysis and the loss of cutaneous sensation are necessarily equal. Very often, particularly where the disease or injury is of but slight severity, there is little or no anæsthesia, although the loss of power is complete; or sensation may be quickly regained while the paralysis is slowly recovered from. In explanation of these facts, Dr. Mitchell suggests that the sensory centres are readily affected by very feeble impulses, whereas the muscles are unable to respond unless their stimulus is conveyed to them in large amounts. As he points out, this supposition accords with the well-known fact that by irritation of the ulnar nerve at the elbow one can excite sensation far more readily than motion. And it is further supported by the results of the application of galvanic currents, described at p. 350. For these results show that the severity of the lesion of a nerve may vary greatly in different cases, all of which are, nevertheless, attended with absolute loss of voluntary power in the muscles supplied by it. I am not aware that anæsthesia ever occurs unaccompanied by motor paralysis, from lesion of a mixed nerve trunk. And, on the other hand, it seems probable that a certain degree of intensity of lesion, as measured by the presence and duration of the "reaction of degeneration," will be found to be constantly attended with impairment of sensation.

Another symptom of affections of the nerve trunks—and one which likewise has its seat in their sensory fibres—is *pain*. It is by no means constantly present, and, indeed, is wanting in most cases of ordinary peripheral paralysis. It commonly exhibits in a more or less marked degree the features which will be hereafter described as those of neuralgia, and is referred mainly to those parts of the skin to which the diseased or injured nerve is distributed. It is sometimes due to neuritis; and the trunk of the nerve may then, in thin persons, be felt to be swollen and exceedingly tender. In some cases it is periodic, returning every day at about the same hour, generally toward the latter part of the day. There is a special variety of it, to which Dr. Mitchell has given the name of Causalgia (καῦσις, cautery), on account of its burning character. This may vary infinitely in severity, up to the most unendurable agony, which the patient compares to a "red-hot file rasping the skin," or to some similar mode of torture. In cases due to wounds, this kind of pain seldom, if ever, comes on until the process of cicatrization is beginning. Its most frequent seat is in the upper limb, the palm of the hand; whereas in the lower limb the back of the foot is more liable than any other part. Heat aggravates it, and allowing

the limb to hang down has the same effect. It is relieved by moistening the skin, and two of Dr. Mitchell's patients always carried a bottle of water and a sponge, and would not allow the affected part to become dry, even for an instant. In course of time, the severe suffering caused by this condition often leads to a general irritability of the nervous system, allied to hysteria. The patient may then complain of hyperæsthesia of the whole body, particularly when the surface is dry. Dr. Mitchell speaks of two such persons as having found some ease in walking from pouring water into their boots.

This peculiar kind of pain seems never to occur in parts which have been rendered altogether anæsthetic by the complete division of their nerves; nor does it accompany the subjective sensations that are so often referred to the distal ends of limbs which have been amputated. From these facts Dr. Mitchell argues that its immediate cause is some change in the peripheral extremities of the nerves, rather than in the trunks above. Such an opinion is strengthened by the circumstance that the integument of the parts in which the pain seems to have its seat is liable to a special change, to which Sir James Paget first gave a name—that of "Glossy Skin" ("*Med. Times and Gaz.*," 1864, i). The surface then shines as though it had been varnished. Or, in Paget's words, "the fingers"—for they are most often affected—"become tapering, smooth, hairless, almost void of wrinkles, glossy, pink, or ruddy, or blotched as if with permanent chilblains." An eruption of fine, pointed vesicles (somewhat like those of eczema) is also of common occurrence; or bullæ may form, which afterward ulcerate; or painless whitlows, leading to shedding of the nails. Or, again, the nails themselves may become arched and incurved, and the skin may shrink away from their roots, leaving the sensitive matrix partly exposed. Parts which are the seat of causalgia are slightly warmer than those on the opposite side of the body; whereas the general effect of injuries of the nerve trunks is to lower the temperature; sometimes, as in some cases of Hutchinson's, by as many as eight or ten degrees Fahrenheit.

Another occasional result of morbid conditions of the nerves of a limb is a painful *swelling* of one or more *of its articulations*, which may go on to anchylosis, and greatly increase the deformity and the disablement experienced by the patient. The arthritic effects of injuries to nerves have been especially insisted on by Dr. Mitchell; they are analogous to the joint affections which occur in certain diseases of the spine. They are in the highest degree rebellious to treatment. Erb also mentions, as sometimes following paralysis of the musculo-spinal nerve, a thickening of the tendons at the back of the wrist—a circumscribed, painless swelling, of the size of a hazel nut, evidently due to their having been exposed to mechanical strain, in consequence of the flexed state of the joint.

These various conditions appear to be sure indications of the existence of some disease affecting the nerves of the parts concerned. But they do not necessarily depend upon a definite lesion of the nerve trunks themselves. Paget mentions two cases in which glossiness of the fingers developed itself as the result of neuralgia after shingles. Dr. Mitchell relates one in which it was due to a chronic affection of the spinal cord. And some years ago Dr. Moxon had, in Guy's Hospital, a remarkable case in which a precisely similar state of the fingers was caused by a mediastinal tumor.

Diagnosis.—With regard to the diagnosis of affections of the nerve trunks I need say but little, as there is seldom much difficulty about it when once one is familiar with their various symptoms. Sometimes, indeed, progressive muscular atrophy in an early stage might be mistaken for the paralysis resulting from lesion of a nerve; particularly if the deltoid muscle should

be alone affected, or the interossei and the muscles of the little finger. The application of galvanic and faradic currents to the paralyzed muscles would clear up the doubt in some cases, but not in all.

Having ascertained that the paralysis is of peripheral origin, one must not omit to search for all possible causes of compression of the nerves. Some years ago a woman came to Guy's Hospital complaining of numbness and loss of power in the right arm, which symptoms (she said) had already been of six months' duration ; she was found to have a cancerous growth in the breast, which had extended to the axilla and involved the nerves of the brachial plexus. One must also bear in mind the possibility that the paralysis may be "reflex," and due to irritation from diseased teeth (see p. 348).

In the lower limbs, the mistake which one is most likely to make is that of attributing to an affection of the spinal cord a paralysis really due to a pelvic tumor compressing the sacral plexus, or to a mass seated in one iliac fossa and interfering with the branches of the lumbar nerves. In 1876 a woman died in Guy's Hospital who had for ten years had paralysis of the sphincters of the bladder and rectum, and who for seven years had had loss of power and sensation in the legs below the knee-joints, so that she was unable to walk, but could crawl about on her hands and knees. The cause was found by Dr. Goodhart to be a large abscess in the pelvis, dependent upon disease of the bones, through the middle of which the cords of the sacral plexus ran.

The *prognosis* of the different paralytic affections that may result from lesions of the nerve trunks of a limb depends upon their nature and severity. Most of the cases that come under the care of the physician get well sooner or later, and one can do much by treatment to hasten their recovery. Thus crutch paralysis commonly passes off in a week or fortnight ; that which follows compression of the nerves during sleep more often lasts from four to six weeks, and may be prolonged for several months ; that which is caused by violent injury, or even by severe stretching or bruising of the nerves, generally lasts more than a year, and may never be entirely removed. Even after very long periods, improvement sometimes takes place. Dr. Mitchell says that only the careful notes that he made could have convinced him of the extent to which restoration of function was found to have taken place, in some of the worst cases, at the end of three or four years.

As may be inferred from what has been stated above, most valuable aid in the prognosis of this form of paralysis is afforded by an examination of the electrical reactions of the affected parts. In cases which are to terminate rapidly in recovery, the muscles and nerves retain their normal irritability ; in those which are to run a protracted course, the muscles present the "reaction of degeneration." In cases which are incurable, the susceptibility to every form of galvanic stimulus is altogether extinguished.

Treatment.—Electricity, however, is useful, not only as a guide to prognosis, but also in treatment. There is, indeed, still much uncertainty as to how it acts, but all writers are agreed that it is beneficial. Sometimes its effects are extraordinary : after a single application of faradic or galvanic currents, the patient may suddenly find that he can perform movements which he had for weeks or months been unable to accomplish. But in such cases it may generally be assumed that the regeneration of the affected parts was already far advanced—that they were, in fact, on the brink of recovery—independently of any treatment. At earlier periods, the action of electricity is generally less striking, and any apparent success from it is still less capable of bearing a critical scrutiny. Dr. Mitchell, indeed, speaks strongly of the importance of the use of faradic or galvanic currents at the earliest possible

moment. As regards the choice of one form of electricity rather than the other, he adheres to a well-known rule which has been laid down by several other writers, namely, that whichever current is found to produce muscular contractions most readily should be used. The idea has been that by stimulating the muscles to contract one helps them in keeping themselves well nourished, besides possibly doing something toward opening up the path for the entrance of volitional impulses. But Erb maintains that, if the "reaction of degeneration" is present, one cannot by galvanization hasten the recovery of motor power, although he admits that when regeneration has once commenced its progress may be accelerated by such treatment.

In employing galvanization in the treatment of paralysis from an affection of a nerve trunk, one should use the "labile" method. The anode is placed over the seat of lesion, or the plexus above, or on some "indifferent" spot. The cathode is then gently moved over the skin, covering each of the affected muscles and nerves in turn. The strength of the current should be such as to excite distinct muscular contractions, but not to cause more than a slight sensation of burning, and a little redness of the skin. Its application should be continued for about ten minutes, and at intervals of twenty-four or forty-eight hours. If the faradic apparatus be chosen, its two poles should be held in one hand between different pairs of fingers, and they should be placed in succession over the various muscles, at a little distance from one another.

Another method of treatment on which Dr. Mitchell lays great stress is that of shampooing the affected parts, or (as it is termed) of "massage." Persons have to be specially trained for the performance of this manipulation, which requires strength and endurance as well as gentleness, for it ought to be continued for an hour at a time. The limb should first be immersed in a hot bath. Afterward every part of the skin, so far as the paralysis extends, should be lightly pinched and tapped, and moved to and fro on the tissues beneath. Then the joints are to be moved in turn, and lastly, the muscles must be gently kneaded and rolled, the power employed being gradually increased. Dr. Mitchell says that at the close of the sitting the temperature of the limb is raised by 1° or 2° Fahr., that the muscles show a marked increase of tone and respond more readily to faradization, and that the skin may be intensely reddened. The patient feels refreshed and sleeps better. It should be repeated every day. A further proof of the efficacy of "massage" is that when it is carried on for too long a time it sometimes causes lumbar pain, headache, nausea, and even great exhaustion.

For persistent anæsthesia, Dr. Mitchell recommends the application of a strong faradic current by means of the electric brush. After two or three sittings, he has commonly found sensation beginning to return in parts which were before completely devoid of it. He has also employed counter-irritants, especially rags dipped in hot spirit of turpentine and covered with oiled silk. This affects different persons very differently, causing unbearable pain in some individuals, while others scarcely feel it. In some cases he says that it is advantageous to apply irritants over a very extensive surface, and he quotes the case of a man who had lost sensation in the whole arm as the result of a fall on the shoulder, and who completely recovered after having the limb and his back severely blistered by the sun.

When the affected nerve is painful and tender, Dr. Mitchell recommends that a bag of ice should be kept constantly applied, both by day and by night.

Causalgia may to some extent be relieved by dressings with cold water constantly renewed. The injection of morphia into the tissues of the affected part was also found useful by Dr. Mitchell, but he seems to rely mainly

upon the repeated application of blisters. Sooner or later this form of pain almost invariably subsides.

The account which I have given of the affections of spinal nerves is applicable, *mutatis mutandis*, to those of the cranial nerves likewise. But the latter possess particular functions, which, when disordered, give rise to special symptoms that must be separately described. I shall accordingly discuss first the diseases of the *motor* cranial nerves (the facial, the ninth, and the nerves of the eyeball muscles), then those of the *sensory* nerve of the face (the fifth), and lastly, those of the one *nerve of special sense* (the olfactory) which is left within the province of the physician.

Facial Paralysis.—The motor division of the seventh nerve—the *portio dura*, or facial nerve—is very liable to affections which destroy its conducting power, and are consequently attended with paralysis of the muscles to which it is distributed. The patient is then said to suffer from facial paralysis, or sometimes from *Bell's paralysis*, in commemoration of Sir Charles Bell's great discovery.

Of the *causes* that may give rise to this form of disease the most frequent is the direct action of *cold* upon the side of the face. Sitting at the open window, whether of a room or of a railway carriage, sleeping near a damp, cold wall or with the face exposed to a draught of air—even the general exposure of the body to cold, as in a patient of Sir Thomas Watson's who walked about the streets for some days, without shoes or stockings, during a thaw—are each of them commonly followed by facial paralysis. Such cases are sometimes spoken of as "rheumatic," according to that wider acceptation of the term which really deprives it of all meaning. The supposition is that the peripheral part of the nerve becomes affected with a slight inflammatory swelling, which (perhaps by extending into the stylo-mastoid foramen) leads to compression of its fibres. The comparative rarity of similar paralyses of the spinal nerves from cold may depend on the larger size of the intervertebral foramina, and on their greater distance from the exposed portions of the nerves. Parotitis, tumors growing in the substance of the parotid gland, swellings of the cervical glands, wounds of the cheek, are the chief other causes that may affect the facial nerve beyond the aqueductus Fallopii. In its course within that canal it is especially liable to be involved in the severe forms of disease of the petrous bone; caries and necrosis, consequent upon scrofulous inflammation of the tympanum; or, more rarely, tumors of various kinds. Dr. Moxon, however, has recorded ("*Path. Trans.*," xx) a case in which a clot of blood was found compressing and destroying this part of the nerve. It seems probable that a similar condition, resulting from the rupture of some small blood vessel, was present in an oft-quoted case of Sir Charles Bell's, in which facial paralysis followed a box on the ear; and also in one, related by Sir Thomas Watson, of a man who was attacked within three hours of having a fall in which he struck his hip and his elbow, but not his head. On the cerebral side of the internal auditory meatus, the facial nerve may be implicated in various tumors and other diseases of the base of the skull; or its origin within the pons may be affected by morbid processes of different kinds. As a rule, such cases are distinguished by the circumstance that other nerves are involved at the same time, or that further symptoms of encephalic disturbance are present. But I have myself met with an instance in which a simple paralysis of the right facial nerve, in a man who died of granular disease of the kidneys, was found to be caused by a minute spot of softening which existed in the pons, rather to the left of its centre, with a perfect little cyst, of the size of a pea. And Dr. Gowers speaks of having "seen two cases of seizure,

evidently apoplectic, in which the only paralysis that followed the seizure was seated in the muscles supplied by the portio dura."

Lastly, it sometimes happens that well-marked facial paralysis occurs in the course of some cerebral disease which cannot be shown to affect the nerve in any special manner. I am not now referring to the partial implication of the facial muscles in ordinary hemiplegia; still less to that which is a constant feature of bulbar paralysis; but to such cases as one to which I shall hereafter refer, and in which a simple facial paralysis was the earliest symptom of tubercular meningitis. Again, I have myself had a patient who was attacked with Bell's paralysis twenty-four hours after the development of an eruption of herpes zoster on the same side of the face. He came to me about seven weeks afterward; the loss of power to move the facial muscles was then almost complete, and the "reaction of degeneration" was found to be present when the electrical test was applied. The purple scars caused by the eruption were still visible. No indication of any disease affecting the seventh nerve could be discovered; and I was inclined to regard the paralysis as reflex in its origin. He slowly recovered, but not until after the lapse of several months.

The *symptoms* of Bell's paralysis are very striking and present many points of interest. When the affection is developed to the fullest possible extent, the patient is altogether unable to move the muscles supplied by one facial nerve. Whether he frowns, or smiles, or laughs, one side remains expressionless; the forehead is unwrinkled, no creases appear round the eye, the cheek and chin are marked by no dimples, the angle of the mouth remains in the same position as before. When he tries to close the eyelids forcibly, those on the paralyzed side are motionless; but since there is a physiological association between forced closure of the eye and elevation of the globe (for protection of the cornea from injury), the latter action, which is effected by certain muscles of the eyeball through the third nerve, still goes on; and the eyeball is involuntarily turned upward, or upward and inward, until the pupil is hidden beneath the upper lid. The actions of spitting, whistling, or blowing, are alike incapable of being performed; the sides of the mouth cannot be pursed up; the cheek is not held close to the teeth by the buccinator muscle, and bulges out when the breath is propelled against it. For the same reason, when the patient masticates, the food cannot be retained within the space between the jaws, and collects outside the teeth on the affected side, so that it has to be dislodged by the finger, unless, indeed, the hand be kept pressed against the cheek as long as he goes on eating. In speaking, he pronounces the labial consonants indistinctly.

The degree to which the face is distorted when in repose varies considerably in different cases, even among those in which the loss of voluntary power is complete. In some patients the mouth is drawn far over to the opposite side, the eye stares fixedly, and the countenance has at all times a most comical aspect. This seems to be due to a contraction of the antagonist muscles, analogous to that which causes distortion of the fingers when the interossei are paralyzed. But perhaps one element in it is an absolute loss of tone in the affected muscles, which may occur only when the nerve is completely destroyed, as in cases of necrosis of the temporal bone. For I think that in most of the so-called "rheumatic" cases the patient's features are but little disturbed while they are at rest, so that at a cursory glance one may almost fail to notice that anything is amiss with him. Again, even when he is altogether unable to close the eye, the palpebral aperture may appear but little wider than on the unaffected side, and the *tensor tarsi* muscle may still be able to keep the lachrymal puncta in contact with the globe, and to prevent the tears from running down over the cheek,

as they do in the most extreme forms of the disease. It even appears to me to be probable, although I do not know that the fact has ever been observed, that during sleep, when the levator palpebræ is relaxed, the eyelids sometimes approach one another much more nearly than would be expected from the patient's inability to close them when he is awake, and when that muscle is acting in opposition to his efforts. In this way one may, perhaps, find an explanation of the not infrequent absence of conjunctivitis, which one would expect to be a necessary result of the irritation from particles of dust settling upon the exposed eyeball. As is well known, the conjunctiva is very apt to become inflamed under such circumstances, and the cornea may lose its transparency and even ulcerate; but Valleix had a case of facial paralysis of twenty years' duration in which no such results took place. In the daytime, as Trousseau remarks, the patient is able to compensate for the failure of movement in the eyelids by bringing the globe into various positions so as to wipe it upon different parts of their inner surfaces; or he may with his finger push down the upper lid and rub it gently over the eyeball.

Among the indirect effects of facial paralysis, impairment of the sense of *smell* is sometimes mentioned; and it is attributed to a dry condition of the corresponding nostril, which results from the escape of the tears over the cheek. A much more frequent symptom is a perversion of the sense of *taste*, but this appears to be directly due to interruption of the conducting power of a branch of the seventh nerve, the chorda tympani. Trousseau, for example, speaks of a man who said that his food tasted like "salt plaster;" and other patients have experienced a subjective metallic or sour taste in the fore part of the tongue on the affected side; or a loss of power to detect acid, sweet, or saline flavors. It is also said that the secretion of saliva may be diminished. On the other hand, the sense of *hearing* is often abnormally acute; there is an augmented sensibility for all musical notes and sounds, the patient hearing a watch at a greater distance from his ear on the affected than on the healthy side; there is also an increased capacity for the detection of sounds of very low pitch; and there may be a subjective sensation of hearing an acute sound. This state of the auditory sense has been termed "oxyakoia," and there have been different opinions as to its cause. Brown-Séquard propounded the theory that it is a true hyperæsthesia of the auditory nerve consequent on paralysis of its blood vessels through the vaso-motor nerves. But Lucæ has since shown that it is a result of paralysis of the stapedius muscle, which receives a branch of the facial nerve; its antagonist, the tensor tympani, being no longer opposed, keeps the membrane too much on the stretch. ("*Berl. Klin. Woch.*," 1874, Nos. 14, 16.)

Another branch of the facial nerve, which is often affected in Bell's paralysis, is that which goes to the soft palate. Dr. Sanders has shown ("*Ed. Med. Jour.*," 1865) that there is then "a vertical relaxation or lowering of the corresponding half of the velum, with diminished height and curvature of the posterior palatine arch," a condition which he believes to be due to loss of power in the levator palati muscle. In certain cases the uvula is turned to the paralyzed side; but this seems not to be constant, and it possesses little significance, inasmuch as lateral deviation of the uvula sometimes occurs in healthy persons.

It sometimes happens that *both facial nerves* are paralyzed in the same patient; the symptoms are then to a certain extent peculiar, so that such cases have been dignified with the special name of "diplegia facialis." The chief point is that there is no distortion of the features under emotion of any kind. The face remains without expression, the patient laughing or weeping as though behind a mask. Double facial paralysis may be caused

by a new growth or a syphilitic gumma, involving both nerves at the base of the skull; but more frequently it is due to disease affecting the two petrous bones separately, or to the action of cold upon both sides of the face at once or in succession; or the facial nerves may each be affected in a different way.

The commencement of Bell's paralysis is sometimes gradual, sometimes sudden. Among the lower classes it often happens that what first attracts the patient's notice is his finding himself unable to spit or to whistle; or he may find his face "all on one side" when he gets up in the morning, having gone to bed well.

The *diagnosis* of this affection is seldom difficult when it is fully developed; but one must not omit to notice whether any other nerves besides the seventh are paralyzed, for if such should be the case, there would be reason to suspect the presence of some new growth or syphilitic disease at the base of the skull. Even when the loss of power is very slight and incomplete, one can for the most part easily determine its peripheral origin; it betrays itself by a little failure of expression limited to one side of the face, and by a narrow chink remaining between the closed eyelids, which are not the symptoms that accompany a partial paralysis of the facial nerve from disease of the brain.

The *course* of Bell's paralysis varies greatly in different instances. When due to destruction of a considerable portion of the nerve, as in cases of necrosis of the petrous bone, it is, of course, permanent and incurable. And although recovery is possible after an injury, such as the division of the nerve in a surgical operation, yet this seldom occurs before the lapse of several months, and it is apt to remain incomplete. Even where the affection is the direct result of exposure to cold, it may last from four to six months; but sometimes it gets well much more rapidly, the patient beginning to regain power over the muscles at the end of ten or twelve days, and recovering completely within a few weeks.

Now, it might naturally be expected that one of the best indications of the probable duration of the affection would be the presence or the absence of paralysis of the higher branches of the facial nerve, the chorda tympani, and those to the stapedius and palate muscles. Dr. Sanders, however, maintained that the prognosis is not more unfavorable in cases in which the last-named branch is implicated. But although it is certain that the palate may be affected when the paralysis is the result of exposure to cold, and will ultimately disappear, yet I do not know that this symptom has hitherto been observed in cases which have recovered rapidly. On theoretical grounds, one cannot but suppose that it must indicate an extension of the morbid action along the nerve for a considerable distance. The point is not, however, of much practical importance, because we seem to have in the application of electricity a ready method of determining the gravity of an attack of facial paralysis from its very commencement. This point has been carefully worked out in Germany by Brenner and by Erb. From their observations it appears that there are two forms of the affection, one of which may be called *mild*, and the other *severe*. In the first form, which gets well within two or three weeks, the muscles and nerves of the paralyzed side of the face react both to galvanic and to faradic currents exactly in the same way as on the healthy side. In only a single instance, in which a rapid recovery took place, Brenner detected a slight and transient diminution of excitability to both kinds of current. But in the *severe* form, including a large proportion of those cases which are due to the direct action of cold, the currents give rise to those peculiar effects which have been described at p. 350, under the name of the "reaction of degeneration." Indeed, it was in a case of facial paralysis that such a reaction was first

noticed, namely, by Baierlacher, in 1859. The prognosis is then always so far unfavorable, that no improvement is to be looked for until two or three months have passed, that a complete recovery seldom takes place before the lapse of another period of equal duration, and that more or less weakness or stiffness of the affected muscles sometimes remains during the rest of the patient's life. Erb, however, describes an intermediate form of the affection in which the excitability of the nerves is not completely lost, although the muscles present the characteristic sensitiveness to feeble galvanic currents with absence of faradic contractility; such cases, he says, recover in from four to six weeks.

In severe cases of facial paralysis, at an advanced stage, *spasmodic affections* are observed, which present many points of interest. When the patient is just beginning to recover voluntary power over some of the muscles, it often happens that the effort to induce contraction in one of them causes simultaneous movements in muscles of a different part of the face. He may wish to move his eyebrow or his eye, and he involuntarily draws his mouth to that side; he may intend to close his lips, and he finds that he also shuts his eye. In a patient who was under my care in 1877, the distortion of the mouth was so great when an attempt was made to close the right eye, that my clinical assistant actually supposed the left side of the orbicularis oris to be paralyzed. Hitzig, who has especially studied these curious phenomena ("*Arch. f. Psych.*," 1872), notes that they sometimes occur while there is absolute paralysis, for all volitional efforts, of the muscles which are concerned in the associated movements. It seems difficult to avoid the inference that the motor impulse must find its way through the obstructed part of the nerve along the fibres which have regained their conducting power, and reach the peripheral branches by some collateral path. But it would seem that the facial nucleus in the medulla oblongata is also in a condition of exalted irritability in many cases, for Hitzig has found that touching the affected side of the face sometimes gives rise to spasms which may even involve the muscles of the opposite side. In one case of facial paralysis, contractions actually extended to the muscles of mastication, and to those of the limbs. The occurrence of associated movements is commonly a precursor of the return of voluntary power over the paralyzed muscles. They generally quickly disappear, but sometimes they seem to delay recovery, and (according to Erb) they have been known to last for as many as thirteen years.

On the other hand, when the paralysis is to be permanent, *tonic contractions* of some or all of the muscles often show themselves, which seem to have been described by Duchenne before they had attracted the notice of other observers. The result may be an elevation of the angle of the mouth, a deepening of the naso-labial groove, a narrowing of the palpebral aperture, or a general exaggeration of all the markings on the affected half of the face. The features are sometimes dragged over to such an extent that one might fall into the error of supposing that the healthy side was the one paralyzed. Duchenne asserted that whenever, in the course of facial paralysis, some particular muscle recovers its tone earlier than one would have expected, one should be prepared for the supervention of tonic spasm in it. But this statement seems inconsistent with the fact that such spasms depend upon the occurrence of degenerative changes in the muscles themselves; a fact established by a case of Hitzig's, in which a patient, who already had contraction of the orbicularis palpebrarum from a former seizure of Bell's paralysis, was attacked a second time by the same disease, without the muscle in question undergoing relaxation.

The *treatment* of facial paralysis is still a doubtful matter. When the discovery was first made that the muscles in many cases exhibit an aug-

mented susceptibility to galvanic currents, most observers jumped to the conclusion that the application of that form of electricity would cure the disease more quickly than the faradization which had before been employed. But in 1869, Erb, after analyzing the cases which had then been recorded, expressed the opinion ("*Deutsches Archiv.*," vi) that the "severe" form, in which alone the reaction of degeneration is present, cannot be materially shortened in its course by galvanization. According to him, the constant current is of little value until the conductivity of the nerves is re-established, but he believes that it then accelerates the recovery of voluntary power over the muscles. He recommends that the anode should be placed behind the ear, and the cathode moved over the paralyzed half of the face; or else that the two poles should be placed one over each mastoid process, the affected side receiving the anode. In mild cases he says that each application of the current is usually followed by increase of voluntary power over the muscles. It must not be forgotten that Duchenne believed that he attained still better results from faradization; he speaks of cases which had lasted for years, and which were, without exception, cured or greatly relieved by this procedure, although all other treatment had failed. So impressed was he with its potency, that he regarded it as capable of inducing secondary spasms, and consequently advised that when the muscles began to regain their tone, the intermissions of the battery should be reduced in frequency to not more than four in the second. He did not indeed recommend that faradization should be laid aside altogether, even when contractions of the affected muscles had set in; but he suggested that they should also be stretched with the fingers at regular intervals, or that a wooden ball should be carried in the cheek.

On the whole, however, it would appear that quite as much is to be anticipated from ordinary medicinal treatment as from electricity. All the most recent writers recommend leeches behind the ear, blisters, and small doses of mercury or tolerably large ones of iodide of potassium, as the best remedies for facial paralysis during the first two weeks. Erb, in particular, says that the iodide has appeared to him to materially shorten the duration of the disease in some "severe" cases.

Paralysis of the Ninth or Hypoglossal Nerve.—This is a comparatively rare affection, but is attended with one very remarkable effect—an extreme degree of wasting on the corresponding side of the tongue. I have seen two cases of this kind; one was in an old woman, in whom a cancerous nodule (secondary to a cancer of the breast) involved the nerve where it passes through the base of the skull; the other in a boy, who had extensive caries and necrosis of the atlas and neighboring bones. In both instances the paralyzed half of the tongue was remarkably flaccid and wrinkled; and its tip was curved round toward the affected side. Similar cases have been recorded by Dupuytren, Sir James Paget, and Mr. Fairlie Clarke. In Paget's case ("*Clin. Trans.*," iii) the affection was caused by necrosis of a part of the occipital bone, itself resulting from an injury. Several pieces of dead bone were removed by operation; a few days afterward the wasted part of the tongue began to grow larger, and within a month it had nearly regained its former size and muscular power.

Paralysis of the Third, Fourth and Sixth Nerves.—Of the movements of the eyeballs some are direct results of the action of single muscles; while the remainder, for which several muscles are brought into play, are capable of a physiological analysis, far more exact than that to which the movements of any other parts of the body can be submitted. And, since three distinct cranial nerves are devoted to them, their study has very important bearings

upon medical diagnosis. It might therefore be expected that the affections of these nerves, or of the muscles to which they are severally distributed, should be taken separately. But this is impracticable, because under normal conditions the two eyes are always moved simultaneously, and because the muscles of opposite sides which are associated together are often not the two of the same name, but one of a certain name and the antagonist of its fellow, receiving nerves from different sources. For example, the external rectus of one side, supplied by the sixth nerve, works, not with the *external* rectus, but with the *internal* rectus of the other side, a muscle which receives its branch from the third nerve. Now, one of the most important symptoms of loss of power in the external rectus is a deviation from the natural movements of the internal rectus of the opposite eye, and *vice versâ*, so that the affections of these muscles have to be discussed together.

At the same time it must be borne in mind that two of the ocular muscles receive each the entire distribution of a cranial nerve, the external rectus that of the sixth, the superior oblique that of the fourth. The consequence is that either of these muscles is more likely to be separately paralyzed than any single one of the rest, which are all supplied by the third nerve, and that the affections of the former muscles are far the most important, from a practical point of view. Moreover, these affections present differences in their symptoms which, *mutatis mutandis*, are typical of those which characterize the paralysis of all the other muscles; so that it is in every way convenient that I should describe them first, and in full detail.

Paralysis of the Sixth Nerve.—We have seen that each external rectus muscle is associated in its principal movement with the internal rectus of the opposite side. Their combined function is of the most simple kind; they merely carry the eyes to the right, or to the left, without any change in the direction of the vertical axes of the globes. Thus, if an external rectus be paralyzed, the corresponding eye cannot be moved outward when the other eye is moved inward. Let us, for example, suppose that a patient has his *left* sixth nerve paralyzed. The result is that he is unable to look with his left eye at any object to his left. If we hold a candle before him, and move it to his right side, his two eyes follow it until the right one has the edge of its cornea touching the external canthus, and the left has a small part of its cornea buried beneath the carnucle. But if we now carry the candle to his left side the right eye alone follows it; the left remains motionless and looks straight forward, or may perhaps perform a slight zigzaging movement to the left, under the combined influence of the two obliqui. The further the object is carried to the left the greater is the difference in the directions of the two eyes; and this *primary deviation* or *squint* at once clears up the nature of the case, when the paralysis is complete. But if the loss of power is partial, the affected eye may lag behind its fellow until it has already moved some distance to the left; and no obvious squint may declare itself. In such a case, one can generally make out the nature of the defect by covering the right or healthy eye; the patient then discovers that he has not got his left eye fixed upon the object, and he accordingly moves that eye further to the left. This change in its position may itself be readily detected by a close observer; but what is far more striking is a movement which is simultaneously made by the right eye. For, the left external rectus being partially paralyzed, any voluntary movement of that muscle requires the exertion of far more force than that which would have sufficed under normal conditions to produce the same degree of contraction in it. But whatever amount of force is used is at the same time thrown upon the associated muscle, the right internal rectus,

which is in possession of its full vigor; and thus the right eye moves two or three times as far as the left one.

This movement of the healthy eye is called its *secondary deviation;* and it is of the more importance, because it affords a sure means of distinguishing a paralysis of the external rectus from a mere contraction or shortening of one or both of the internal recti, such as exists in cases of ordinary strabismus. When the loss of power is considerable, this distinction is indeed made by the fact (already mentioned) that the primary deviation increases as the object looked at is carried further over toward the left side; for if the affection were a mere convergent strabismus, the axes of the eyes would remain at exactly the same angle, the one lagging at a fixed distance behind the other, but nevertheless traveling with it, so that such an affection is sometimes called "concomitant." But in slight cases this criterion fails. Its place is then taken by the secondary deviation, the *augmented amplitude* of which is (as we have seen) dependent on the existence of paralysis. In cases of ordinary strabismus the secondary is exactly equal to the primary deviation, since the muscles on both sides possess equal power.

The other symptoms of paralysis of the sixth nerve consist of subjective sensations experienced by the patient himself, and of movements to which they lead. One is that when his eyes are turned in a certain direction he sees double images of the objects at which he looks; and this "diplopia" is not infrequently the first thing which draws his attention to the fact that something is amiss with him. We will assume, as before, that the *left* external rectus is the muscle paralyzed, and we will call the image which is formed upon the retina of the affected (or left) eye "the *false* image;" the one which is formed on the unaffected (or right) eye "the *true* image." It is obvious that double vision occurs only when the eyes are directed to the left, for it is then that they fail to converge properly upon the object. And a moment's consideration will show that since the left eye has its axis turned inward (or to the right), the false image must be formed upon the inner side of its retina, and not upon its centre; and consequently that it appears to be *outside the true image*, or *further to the left*. Both images are upon the same level, and both are upright. By placing a piece of colored glass before one of the patient's eyes, we can enable him to distinguish very easily which image is formed by that eye, and which by the other; reddish violet is said to be the best color for this purpose.

It must be borne in mind that diplopia is not necessarily a proof that paralysis of any of the ocular muscles is present. Double images are sometimes formed upon the retina of a single eye; and in other cases they are due to a mere "concomitant strabismus." Making the patient look at the object with each eye separately will, of course, exclude the former condition; but the latter can only be dismissed from further consideration if it is found that the two images get wider apart as the object is moved further to the left. One must not assume that a diplopia which is of recent origin, and which began suddenly, cannot be due to a concomitant strabismus; for it sometimes happens that a patient whose ocular muscles are imperfectly antagonized is able to keep up the balance between them until he overfatigues his eyes, or until he is weakened by some illness, when he may abruptly begin to squint.

On the other hand, it would also be a mistake to suppose that whenever there is a loss of power in the external rectus muscle, the patient must necessarily notice diplopia. There is always the possibility that he may not previously have been accustomed to use the two eyes together, or that he may have been in the habit of concentrating his attention upon a single retina, in which case all that he is likely to notice is a blurring of the objects at which he looks, interfering with the distinctness of his vision. One can

generally make such a person aware that he really sees double by directing him to fix his eyes upon some bright small object, especially if a colored glass be held in front of one eye.

Another subjective symptom is that which is known as the "erroneous projection" of the visual field. We have seen that a patient who has paralysis of the left external rectus muscle refers the false image of any object to the left of him, which he sees with both eyes open, to a position outside its true position, or more to the left. And the same thing occurs, if he is told to look at an object with the right eye closed; only that his judgment is then based upon an appreciation of the degree of effort required to bring his left eye to bear upon it, which effort is augmented in proportion to the failure in the power of the muscle. One consequence is that such a patient feels giddy, and may even stagger, if he attempts to use his left eye alone; a point of some importance, because a careless observer might mistake it for a sign of serious cerebral mischief. Another result of "erroneous projection" is commonly employed as an aid in the diagnosis of paralysis of the ocular muscles. The patient is told to close the eye supposed to be unaffected, and to strike suddenly at an object placed toward the outer side of the other eye. If the external rectus muscle is paralyzed, he is sure to miss the object by going to the outer side of it. Yet another point is that some patients free themselves from the uncomfortable sensations to which such an affection gives rise, by keeping the head fixed over toward the paralyzed side, so that the images of the objects at which they look may fall upon the outer side of the retina, and be referred to their right positions.

When paralysis of the left external rectus muscle has lasted for a considerable length of time, the position of the eyeball often undergoes a further change. It is now drawn inward by the uncontrolled action of its internal rectus, so that a convergent squint is constantly present, whatever may be the direction in which the patient looks, unless, perhaps, it be very far indeed to the right. Such a condition is designated by von Graefe the "secondary contraction of the antagonist muscle;" and he has pointed out the curious fact that it is sometimes altogether disproportionate in degree to the loss of power in the muscle originally affected.

Paralysis of the Fourth Nerve.—The action of the *obliquus superior* is far from being as simple as that of the rectus externus; and its paralysis is proportionately difficult of detection. Modern investigations have shown that the upward and downward movements of the eyes, instead of being respectively the direct results of the contractions of the superior and inferior recti, require that the obliqui should be associated with these muscles. Each of the two recti, when acting alone, rotates the globe, besides raising or lowering it; but as one of the obliqui tends to rotate it in an opposite direction, the resultant of their combined action is a straight movement in a vertical plane. The left inferior rectus, by itself, would lower the cornea and carry it round to the left; the left superior obliquus, by itself, would lower it, and carry it round to the right. Acting together, they move it straight downward. It is further evident that the rotatory action of the two recti upon the (left) cornea must be more apparent when the eyeball has been previously directed to the left; that of the obliqui when it has been previously directed to the right.

Now let us suppose that the left superior oblique muscle is paralyzed. The result must necessarily be that when the patient looks downward the cornea will be carried to the right by the inferior rectus, which has now no antagonist capable of counteracting its tendency to rotate the globe in that direction. In other words, he will squint to the right and slightly upward with the affected eye, when it is directed upon an object toward his feet.

The "secondary deviation" of the sound eye will be downward and to the left. These symptoms, however, are comparatively little marked, so that the subjective phenomena of diplopia acquire a far greater relative importance in the diagnosis of the paralysis of the fourth than in that of the sixth nerve. The double images are seen chiefly when the patient looks downward, and their position in regard to one another at once indicates what muscle has lost its power. Thus, firstly, the false image lies *below* the true one; secondly, it is placed to its *left;* and, thirdly, it appears to be *tilted* in a particular manner. This last circumstance depends upon the way in which the affected eye is rotated, for since the upper end of an object necessarily has its image formed upon a part of the lower half of the retina outside the normal vertical meridian of the retina, the object itself seems to that eye to be placed obliquely *with its upper end tilted to the right.* Another point noticed by von Graefe is that the false image seems to be on a plane nearer to the eye than the true one; this, he says, depends upon the circumstance that they are both referred to positions upon a horizontal surface spread at the patient's feet, for the true image, being the upper of the two, is, of course, supposed to be the more distant. This writer also says that the patient, in order to avoid a sensation of giddiness when he looks with both eyes at objects before him, keeps his head turned downward and to the right, a position which is very characteristic, because it is not one which a person is likely to adopt under other circumstances.

Paralysis of the Third Nerve.—Since this nerve, unlike the others, is distributed to four of the ocular muscles, the symptoms produced by paralysis of it must, of course, be different, according as the affection is limited to particular branches or involves all of them alike. Thus I must first describe the effects of paralysis of each muscle singly, but I need not enter much into details, for in every instance they are analogous to one or other of the two conditions already so fully described.

Thus in paralysis of the *internal rectus* the symptoms are the converse of those which belong to paralysis of the external rectus. If we take, as before, the muscle of the left side, the "primary deviation" occurs when the eyes are directed on an object toward the right; the affected eye cannot then be moved inward while the other one is being moved outward, and a *divergent* squint is produced. The "secondary deviation" of the right eye is toward the right. Double images are perceived by the patient when he looks to the right, and the distance between them increases the further the object is moved in that direction. They are both upon the same level and both upright. They are said to be "crossed," that is, the false image (which is seen by the left eye) lies to the *right* of the true image (which is seen by the right eye). The "false projection" of the image takes place toward the right; the patient, if he aims suddenly at an object with the right eye closed, misses it by going too far to the right. To avoid giddiness, the patient keeps his head over to the right.

In paralysis of the left *inferior rectus* most of the symptoms are the converse of those which are produced by paralysis of the superior oblique muscle. As in that affection, the affected eye squints a little upward when the patient looks downward, but the rotation of the globe is now to the left. Diplopia occurs when the eyes are directed upon an object below them; the false image is below the true one, lies to its right, and has its upper end tilted to the left.

In paralysis of the left *inferior oblique* the affected eye squints a little downward when the patient looks upward; the globe is rotated to the right. The false image lies above and to the left of the true one, and has its upper end tilted to the left.

In paralysis of the left *superior rectus* the affected eye squints a little downward when the patient looks upward; the globe is rotated to the left. The false image lies above and to the right of the true one, and has its upper end tilted to the right.

Now, if the *whole of the third nerve* on the left side is paralyzed, the symptoms are necessarily in great measure identical with those which indicate affections of the muscles severally supplied by its branches. There is complete loss of power to move the eye inward or upward. It cannot be moved straight downward, but it can be lowered slightly if at the same time it is carried a little outward. In the direction horizontally outward its play is perfectly free. These last two movements are effected respectively by the muscles supplied by the fourth and sixth nerves, and in the former of these the globe is, of course, made to revolve on its antero-posterior axis by the uncompensated action of the superior oblique muscle. Indeed, the rotation is so marked under such circumstances—especially when the eye is allowed to move as little outward as possible—that it affords a striking confirmation of the modern views with regard to the action of the ocular muscles.

The visual field of the affected eye is reduced, according to von Graefe, to about one-twelfth of its normal extent; it is limited by a straight horizontal line above, but below by a curved line which sweeps downward and then outward.

The direction of the "primary deviation" of the left eye, and that of the "secondary deviation" of the right eye, necessarily vary according to the position of the object toward which the patient directs his sight; the relations of the false to the true image, of course, undergo corresponding variations, and so also does the "erroneous projection" of the different objects that meet his eye on different sides of him. The consequence of this is that giddiness is a far more marked symptom when several of the ocular muscles are paralyzed than when one alone is affected, and it cannot be obviated by the adoption of any particular posture for the head, nor, indeed, by any method except that of keeping the affected eye closed. It is only when an object is looked at which is situated to the extreme left of the visual field, that the patient ceases to squint and sees a single image with the two eyes.

But in paralysis of the entire third nerve, in addition to the affection of the four ocular muscles, there is loss of power in certain other muscles which are also supplied by that nerve. One of these is the levator palpebræ, and the result is that the upper eyelid is dropped, a condition which is termed *ptosis*. The patient is altogether unable to open the eye in the ordinary way; at most, he can only slightly separate the lids by wrinkling the forehead—by means of the occipito-frontalis. Ptosis may exist without paralysis of any other muscle; indeed, it is much more common than an isolated affection of any one of the other branches of the third nerve. Again, the sphincter muscle of the pupil receives filaments from this nerve, and paralysis of them leads to dilatation of the pupil, or (as it is termed) *mydriasis*. Ophthalmic surgeons state that the aperture of the iris never becomes so wide in this affection as it does under the influence of atropine; but it is often large enough to interfere very much with the accuracy of vision, owing to the formation of "circles of diffusion" upon the retina.* Lastly, the ciliary muscle is also supplied by the third nerve, and *paralysis of accommodation* may be one of the symptoms of affections of that nerve. Except in very short-sighted persons, this interferes greatly with the distinctness of vision for small print and other near objects. To detect it, one must,

* [From the greater or less amount of spherical aberration of an average eye not being corrected by the iris, acting as a diaphragm.—ED.]

of course, test the range of accommodation in the usual manner with a convex lens.

With regard to the *causes* of paralysis of the third, fourth, and sixth nerves respectively, our knowledge is at present imperfect. One point of very great practical importance is that almost all those cases in which the affection remains limited to a single nerve, or to a single branch of the third nerve, terminate sooner or later in recovery. Their pathology is consequently unknown, but most writers think that they are either "rheumatic" (*i. e.*, that they are like the common form of facial paralysis) or syphilitic. In 1876, a man died in Guy's Hospital of aortic aneurism, who had, about nine months before, been in another ward suffering from ptosis of the left eye and pain in the left side of the head; an old meningeal apoplexy was found. The termination of the left internal carotid artery was dilated and its coats much thickened, and the third nerve on that side was adherent to the side of the artery and stained of a deep brown color. Indeed, Sir William Gull used to say of the third nerve that it ran a "dangerous course," on account of its liability to compression in passing between the posterior cerebral and the superior cerebellar arteries, if these vessels should happen to become diseased; and, although I am not aware that such a hypothesis has ever been verified, it certainly accords well with the fact that ptosis is very apt to occur in old people whose arteries are dilated and tortuous. Whenever two or more nerves are simultaneously affected one must always suspect the presence of some malignant growth, or aneurism, or gumma at the base of the skull. But, of course, any one of these may cause paralysis of a single nerve, and, indeed, an intracranial carotid aneurism is very likely to compress the sixth for some time before it reaches the rest. It appears to be doubtful whether syphilitic affections of isolated nerves are necessarily dependent upon an actual gumma. I have seen one case in which there was ptosis of each eye, the ocular muscles escaping entirely, and it was difficult to suppose that the corresponding parts of the third nerves were the seats of such a growth on both sides. Von Graefe is said to have traced to syphilis about one-third of all the paralyses of the muscles of the eyes. We shall hereafter find that such affections may accompany various diseases of the base of the brain, and that they are often early phenomena in locomotor ataxy. But when any organic lesion of the brain or spinal cord is present, one can almost always discover other symptoms, which prevent one supposing that the case is merely one of "peripheral" paralysis of a nerve trunk.

I have already remarked that most cases of ptosis or of paralysis of the various ocular muscles get well, unless, indeed, they are due to serious organic mischief, such as aneurism or malignant growth, compressing the affected nerve. But it must be added that recovery is sometimes slow, taking many months; and that it may be imperfect, one or more of the affected muscles remaining permanently weak.

Most observers think that iodide of potassium is capable of hastening the cure, even in cases which are not of a syphilitic nature, and the application of blisters behind the ear is also believed to be serviceable. Benedikt and Erb have found galvanism very useful, a current from six to fourteen cells being applied for two or three minutes at a time, with the anode over the temple or the back of the neck, and with the cathode upon the closed eyelids. The improvement is said to be often instantaneously manifested, and Benedikt thinks that if no good is effected within the first fortnight there is no prospect of benefit from the continuance of this plan of treatment; but, according to Erb, many cases require to be galvanized for several months before any result is discoverable. Faradic currents also are sometimes useful.

Paralysis of the Fifth Nerve.—Of this affection the principal symptom is, of course, anæsthesia of the face. The loss of sensation or the corresponding subjective sensations of numbness and painful tingling may, in some cases, be traced with great accuracy to the median line of the forehead, nose, and mouth. A circumstance which commonly first attracts the patient's notice is that when he puts a cup to his lips he feels only half of it; it seems to him exactly as though it were broken. The eyelashes and conjunctiva are perfectly insensible; so also is the nostril, and liquor ammoniæ may be applied to it without causing sneezing; but there is no impairment of the sense of smell, except such as results from the dry state of the nose, which is the consequence of a diminished secretion of tears. On the other hand, the sense of taste has, in several cases, been found absent in the anterior portion of the tongue. Three instances of this are given by Romberg, in which quinine seems to have been the substance chiefly used to test the gustatory powers of the patient. It is the same part of the tongue which is deprived of the sense of taste in cases of facial paralysis; and the only possible explanation seems to be that the chorda tympani is the true gustatory nerve, and that in different parts of its course it runs both with the fifth and the seventh nerves, the connecting branch known as the greater superficial petrosal nerve being probably the means of its transference from one to the other.

Another effect of interruption of the conducting power of the fifth nerve is paralysis of the masticating muscles on the affected side. This only deprives the patient absolutely of one movement, that in which the lower jaw is carried laterally over toward the healthy side by the external pterygoid muscle. His power of chewing food is much less impaired than might have been expected, but he really masticates only with the muscles of the opposite side. If he is thin, one can see that the temporal and masseter fail to swell out when the mouth is forcibly closed; even if he is well nourished, one can easily feel that they do not harden.

In certain cases of facial anæsthesia, the circulation in the affected parts is disordered, or their nutrition is to some extent impaired. Romberg mentions one instance in which the cheek became livid when exposed to cold, in marked contrast with the healthy hue of the opposite side of the face. Sponginess of the gums, ulceration of the mucous membrane of the nose and mouth, and hemorrhages from their surfaces, have been observed. But such changes are rare and comparatively unimportant, whereas there is another affection which is of frequent occurrence, namely, inflammation of the eye, leading to sloughing of the cornea. The physiologists have made this the subject of elaborate investigations, on account of its bearing upon the question of trophic nerves. A point which seems to be established is that the inner portion of the trunk of the fifth nerve contains all those fibres which are specially concerned in the destruction of the eyeball; but there is still a difference of opinion as to whether the result is due to a mere interruption of their conducting power, or (as Charcot thinks) to some directly irritant influence. The view of Snellen, that the anæsthesia is its immediate cause, the eye being exposed to mechanical injuries of various kinds in consequence of the loss of the protection afforded by the sensitive nerves, is now generally abandoned. Indeed, Charcot cites several instances in which the cornea sloughed as the result of affections of the fifth nerve, unattended with loss of sensation.

We have seen that in the affections of the great nerve trunks of the limbs, when the continuity of their fibres is not completely destroyed, anæsthesia, if present at all, generally quickly passes off. We should therefore expect that a nerve running so protected a course as the fifth should be little liable to have its conducting power for sensory impressions interfered with, except

by lesions of a serious kind. And I believe that this is the case. Romberg, indeed, mentions that a watchman, who had to spend his nights in a hall where the left side of his face was constantly exposed to a draught, experienced violent pains in that part, and subsequently anæsthesia of the second division of the trigeminal nerve; within three weeks, under treatment with iodide of potassium, he recovered. But such instances seem to be infinitely rare. The same writer relates two or three other instances in which loss of sensation in the face was due to morbid changes in the Gasserian ganglion, that structure having been found after death to be swollen and indurated or discolored. The nature of the changes in question seems, however, to be doubtful; and we are not told why a fatal termination occurred. In the immense majority of cases of paralysis of the fifth nerve, the cause is a complete destruction of the trunk or of its ganglion, resulting from caries and necrosis of the bones, syphilitic disease of the bones or membranes, cancerous or sarcomatous growths, or, lastly, aneurism of the internal carotid artery.

Paralysis of the Olfactory Nerves.—On account of the important contrast which exists between certain of the symptoms that accompany diseases of the trifacial and of the olfactory nerves respectively, I now pass on to consider loss of smell, or, as it is termed, *anosmia.* I shall not have a more convenient opportunity, for the corresponding affections of the auditory and the optic nerves will not require notice in this work.

A man may be deprived of the sense of smell in one side of the nose, or in both. When the affection is unilateral, he is very likely to remain ignorant of his loss, unless one is particular to test the power of the olfactory nerves separately, carefully closing each nostril in turn. We shall hereafter see that this form of anosmia frequently accompanies hemiplegia. When both nerves are affected, the patient is sure to notice the defect. His inability to perceive odors is then complete; he is altogether insensible to the most delicious perfumes and to the strongest stinks. But he retains his susceptibility to pungent vapors, such as those of ammonia and of acetic acid, and snuff is still capable of making him sneeze. The impressions from all these substances are conveyed to the brain by the branches of the fifth nerve which are distributed to the nasal mucous membrane; and it is when that nerve is paralyzed that they fail to excite sensations.

But, after all, the symptom which chiefly forces itself upon the notice of a patient affected with complete anosmia is not the loss of the sense of smell (as he understands it), but that of a large part of what he considers to be the gustatory sense. He can, indeed, still recognize bitter and sweet, sour and salt tastes; and he can distinguish the rough or smooth character of the solids and of the liquids that he takes into his mouth. But in all other respects his sense of taste appears to be extinguished. He cannot tell one kind of meat from another; boiled apples, boiled onions, boiled turnips, all appear the same to him; all kinds of wine seem to have lost their flavor, tasting merely like sour or sweetish water, except that they are more or less rough to the palate. If it were not from habit and prejudice, he would probably be altogether indifferent as to the nature of his food. Evidently, therefore, those physiologists are right who maintain that the true gustatory sense is limited to the four simple tastes; all the other perceptions which are commonly ascribed to it belong in reality to the sense of smell, and depend on the fact that substances taken into the mouth give off volatile particles, which reach the nose through the pharynx.

The pathological significance of anosmia, however, was first fully worked out by Dr. Wm. Ogle ("*Med.-Chir. Trans.*," 1870). He refers, in the first place, to five cases in which this condition resulted from blows upon the

head, the part struck being generally, if not always, the occiput. He believes that in such cases the olfactory nerves are torn across as they pass through the holes in the ethmoid bone. But it seems difficult to suppose that all the filaments of both nerves should be simultaneously ruptured; and I should have been rather disposed to think that the olfactory bulbs themselves had shared in the bruising of the anterior lobes of the brain, which is so common a result of injuries to the back of the head. Or Dr. Ferrier may be right in supposing that the affected parts are the under surfaces of the temporo-sphenoidal lobes, which he believes to contain the centres of smell and taste. He mentions one case which certainly tells for his view—that of a man who, after a fall upon the top of his head, was not only unable to smell, but who actually could not distinguish sweet tastes from bitter ones.

The remaining causes of loss of smell are really independent of any affection of the olfactory nerves, but it will be convenient to enumerate them here. One is, perhaps, an absence of the pigment in the mucous membrane of the upper part of the nose; at least, a case has been recorded in which a negro boy lost the power of smell more or less completely at the same time that his skin became white. Dr. Ogle supposes that the nasal pigment also underwent absorption, and he cites some other evidence to show that there is a relation between the presence of this pigment and the olfactory sense. Another cause of anosmia is closure of the passage into the posterior nares, by adhesion of the palate to the pharynx; this prevents the patient from drawing air through the nose, and so deprives him of susceptibility to odors, while he also loses the more important power of perceiving flavors. But there are other conditions in which the one kind of impressions is lost without the other, and which, therefore, would correspond exactly with the popular conception of an affection of smell, independently of taste. Such cases always depend upon an obstruction to the passage of air upward to the olfactory region through the anterior nares, while the passage through the posterior nares is open. They generally result from thickening of the Schneiderian membrane from chronic catarrh, so that one part of the lateral wall of the nose is apparently brought into contact with the septum. Dr. Ogle mentions the case of a woman who for several years had had entire loss of smell from this cause. Some years ago a lady came to me who said that for ten months she had lost both smell and taste as the result of a severe cold. I have little doubt that what she was really devoid of was the power of appreciating flavors. I prescribed some liquor arsenici hydrochloricus, and in less than a fortnight she perfectly recovered.

Neuralgia.—In the second of the two groups into which (at p. 347) affections of the nerve trunks were divided, the principal, and sometimes the only, symptom is pain. This, however, is the chief sign of many other diseases likewise, and therefore it is of the first importance that one should lay down as accurately as possible the characters which distinguish neuralgic pains from those which are not neuralgic.

Now, pain itself, although it is so familiar to every one, seems, nevertheless, to be an ultimate fact of sensation, incapable of definition or of explanation. Erb surely throws but little fresh light on its nature when he describes it as "the reaction of the sensorium to a certain degree of excitation beyond that which would cause common sensation."

Under ordinary circumstances, the impressions which give rise to pain are made upon the peripheral filaments of a nerve, and are conveyed by that nerve to the cerebral centres; and the sufferer recognizes as the seat of the pain the part to which the filaments in question are distributed. Sometimes the irritation which causes pain is applied to the trunk of the nerve and not

to its terminations; as, for instance, when the ulnar nerve is struck or compressed, where it passes behind the internal condyle of the humerus; but such conditions are exceptional.

In neuralgia, on the other hand, the pain is never excited by direct irritation of the distal extremities of the nerve to which it is referred.

I think that two distinct affections are included under "neuralgia." The one is really due to peripheral irritation, but this is not applied to the painful nerve, so that the patient is mistaken in his interpretation of the local sign. This is sometimes called "reflex neuralgia." As an instance of it, I may cite the trifacial neuralgia which is so often excited by disease of a tooth. Such an affection can most readily be explained by the hypothesis that the irritation is directly transferred from one nerve nucleus to another within the cerebro-spinal centres. In the other form of neuralgia, of which sciatica may be taken as an example, there is every reason to believe that the morbid process begins in the trunk of the nerve which seems to be the seat of the pain.

The late Dr. Anstie was strongly impressed with the belief that the posterior roots of the nerves, and especially their ganglia, were in some particular manner concerned in the ætiology of neuralgia. But one can hardly regard such a theory as applicable to the "reflex" form of the disease. And even in the other form, as Erb points out, a strong argument against it may be found in the fact that, when the arm is affected, the pain is referred definitely to certain branches of the brachial plexus, whereas the fibres from each root are, of course, spread over several of the offsets of that intricate interlacement of nerves.

We are altogether ignorant as to the nature of the change in the nerve trunks, or in their nuclei within the spinal cord, which constitutes neuralgia. When sciatica has lasted for any length of time, the muscles of the affected limb are always flabby, and their strength is impaired, so that they may be said to be partially paralyzed. This would seem to suggest that the state of the nerve differs rather in degree than in kind from that which is present in the milder forms of peripheral paralysis; and such a view harmonizes well with the fact (already noticed at p. 353) that a slight injury to a nerve trunk causes tingling and pain along its course, whereas severe pressure causes loss of muscular power, without necessarily incapacitating it for the transmission of sensory impressions. But, according to Erb, a partial anæsthesia is constantly present in all cases of neuralgia, in the skin supplied by the nerves which are the seat of the pain.

It may be thought that the varieties of neuralgia to which I have referred ought to be separated entirely from one another, and to receive distinct names. But this would not be convenient, because they are really alike in many respects. In both of them the pain, instead of being simply referred to the peripheral extremities of the nerves, is felt to shoot or dart along the course of their fibres. And in both of them, after a time, there are constantly developed certain "tender points," pressure upon which causes a great increase of the patient's sufferings, while they are often, though not always, the seat of spontaneous shooting pains. It was Valleix who first drew attention to these *points douloureux*, as he termed them; and he maintained that they are of great importance in the diagnosis of neuralgia. He further showed that they are constant in position for each of the principal cutaneous nerves; corresponding generally with the spots at which they emerge from bony canals or from fasciæ. Trousseau has since laid stress upon the presence of an additional "point" at the spinous process of the vertebra beneath which the affected nerve escapes from the spinal canal; this he terms the apophysial point. With regard to the frequency of the occurrence of "tender points" in cases of neuralgia there

has been much diversity of opinion. I think that this depends upon the varied scope of application of the term neuralgia by different writers. Thus Eulenburg, who includes migraine, found tender points in only about half the cases which he examined. Anstie insisted on the fact that it is only when the pain reaches a certain degree of severity and of persistence, that they develop themselves. To me it seems probable that they are after all merely spots at which nervous filaments happen to be so placed that they can be readily compressed. I have always found that in cases of sciatica the external popliteal nerve is much more sensitive to pressure on the affected than on the healthy side, in that part of its course in which one can without difficulty feel it and roll it beneath the finger, namely, when it is running under cover of the biceps tendon.

Another character of neuralgia, which is remarkably constant, and which is often useful for diagnostic purposes, is its limitation to the nerves of one half of the body. Even when it attacks a large number of nerve trunks on the same side, it comparatively seldom extends across to those of the opposite side; and I believe that it is never absolutely symmetrical, presenting the same degree of severity, and reaching the same extent of distribution, both to the right and to the left of the spinal column.

According to what has been stated, neuralgia may be defined as including every pain that is not excited by irritation of the peripheral ends of the nerve to which it is referred. This obviously opens a very wide field of application for the term. The pain in the shoulder which attends upon disorder of the liver, the pain in the arm which is experienced in heart diseases, the pain in the testicle which is caused by a renal calculus, are each typically neuralgic; and so also are gastrodynia and angina pectoris, when they are not due directly to a morbid stimulation of the terminal filaments of the gastric and cardiac nerves respectively. Even the pain in the side which accompanies a hepatic abscess or a pleurisy is in part neuralgic, for it often extends along the cutaneous branches of the affected nerves far beyond the area of inflammation, and the skin itself may be tender when it is pinched up.

Now, one may fairly refuse to apply the name of neuralgia to cases in which the pain is altogether subordinate to a definite organic lesion; and also, as I think, to those in which it is transitory and (so to speak) accidental in its origin. I shall hereafter have to insist on the fact that the occurrence of a single epileptiform fit does not constitute a case of epilepsy; and so it may be said that the neuralgic pain which accompanies the passage of a renal calculus is not actually a neuralgia. But it must be confessed that there is great difficulty in drawing the line as regards some other affections. For instance, *migraine*, which is regarded as a neuralgia by all the older writers, and even by Eulenburg and Anstie, is now described as a separate neurosis. It is, indeed, certain, for reasons that will be given hereafter, that the seat of migraine is in the nervous centres themselves. But it is equally certain that the pain is referred to the cutaneous nerves of the scalp, and shoots and darts along in the course of their fibres. Moreover, in persons who have suffered from this disease for some time, true "tender points" are developed, exactly as in an ordinary neuralgia; and other trophic changes occur, which show that a morbid influence has been reflected outward upon the hair and other superficial tissues. Thus it does not appear easy to exclude migraine from the definition of neuralgia, while the common "reflex" forms are allowed to retain their places in it. The latter, however, seem to be affections of the lowest nerve nuclei, whereas the former probably has its seat in one of the higher centres for sensation, such as those in the thalamus. And I think that the difficulty may be removed by the consideration that the nerves of the scalp may fairly be supposed to bear the

same relation to the brain beneath, which has been shown by Hilton and by van der Kolk to exist between the superficial nerves of all parts of the body, and the organs or other structures which are covered by them. Just as almost any disease of the lung may be accompanied by pains referred to the nerves which are distributed to the skin over the chest, so we shall find that various affections of the brain and its membranes may be attended with pains running in the course of the frontal, the temporal, and the occipital nerves. The pains themselves are neuralgic, but the presence of other symptoms shows that the disease, as a whole, is something more than a neuralgia.

However this may be, it is certain that neuralgia bears a very close ætiological relation to the neuroses in general: I shall more than once have occasion to refer to this when speaking of the different local varieties of the disease, some of the chief among which I must now pass on to describe.

Neuralgia affecting the Fifth Nerve—Trifacial Neuralgia—Prosopalgia.—I have already remarked that many cases in which pain is referred to the branches of the first division of the fifth nerve belong to a separate neurosis, called migraine. But there remain numerous instances in which one or both of the lower trunks, or even all three of them, are affected; and among them is included the most severe of all neuralgiæ—a terrible malady, for which the most convenient name is, perhaps, *tic douloureux*. By Trousseau it is called "epileptiform neuralgia," apparently because it occurs in paroxysms with sudden onset, which so far resemble the attacks of epilepsy. But, as I shall hereafter explain, I prefer to use the word "epileptiform" in a different sense.

The absolute suddenness with which the pain of tic douloureux comes on is, indeed, one of its most remarkable characters. The patient is perhaps sitting quietly reading, when he jumps up from his seat, and rushes up and down the room, with his hand forcibly pressed against his cheek. Or he may rock himself backward and forward in the chair, crying out or uttering deep groans. In ten or twenty seconds, or a minute at the longest, the paroxysm is over. It ceases as abruptly as it began. The pain does not always affect all the branches of the fifth nerve; sometimes only those of the second or of the third division are attacked. In some cases the muscles of the side of the face are at the same time thrown into violent spasms, so that the patient makes horrible grimaces and contortions. The paroxysms may return every few minutes. Trousseau mentions one case in which there were sometimes twenty in an hour; they sometimes do not even intermit during the night. They are often brought on by movements of the jaws, as in speaking, or in eating or drinking; and sometimes pressure upon one of the teeth will instantly excite an attack. Sometimes remissions occur, the patient remaining free from the disease for several days together, or even for some months. But presently it returns, and is as severe as ever. The "tender points" are well marked in all cases of tic douloureux which have lasted for any length of time; they are situated at some or all of the numerous spots at which branches of the fifth nerve emerge from bony channels, or perforate fasciæ. Pressure upon any of them is instantaneously followed by agonizing pain, and a breath of cold air upon the face may have a similar effect. Another result of the disease, when it is of long standing, is the disappearance of the whiskers or beard from the affected side of the face, the hair being worn off by the friction to which it is exposed. According to Trousseau, even the configuration of the bones may become altered, the malar eminence and the prominent part of the lower jaw being flattened down.

A patient who labors under tic douloureux acquires an expression of intense distress and suffering; his countenance is worn and wrinkled, and looks like that of a much older person. But the disease scarcely ever

occurs in those who are under forty years of age, and very often begins at a still later period. Dr. Anstie says that the worst case he ever saw was in a woman who was eighty years old when she was first attacked. According to this writer, a special feature in the ætiology of this form of neuralgia is that the sufferers from it almost invariably come of a stock which is tainted with insanity; indeed, they are themselves often the subjects of a suicidal melancholia, and their mental condition is almost always one of moody depression. This is, no doubt, partly caused by the severe pain which they have to endure, and partly also by their inability to eat solid food, which may lead to their living entirely upon liquids; moreover, they are very apt to seek a temporary relief in the stupefaction produced by drink.

Tic douloureux is happily very rare. I believe that its pathology is as yet unknown. Sir Thomas Watson gives an account of a post-mortem examination which was made in the case of Dr. Pemberton, a London physician of great repute in his day, whose career was ruined by the disease. The os frontis was unusually thick, and a bony mass lay within the falx cerebri. It was supposed that these changes had set up an irritation which caused the neuralgia, but there is no evidence that any branch of the fifth nerve was involved in them, and outgrowths of bone of a precisely similar kind are often found in those who have had no pain in the head or face, nor any cerebral symptoms. Even in a celebrated case related by Romberg, in which the carotid artery was dilated to twice its usual size where it passed through the cavernous sinus, it seems to me exceedingly doubtful whether the so-called aneurismal condition of the vessel had anything to do with the tic douloureux from which the patient had suffered on the corresponding side of the face; for that disease had existed for eighteen years at the time of his death, and one cannot suppose that the artery had been dilated during the whole of that time. I have repeatedly seen a similar affection of the carotid at the same spot, in the bodies of those who had made no complaint of pain. It is also difficult to believe that the fifth nerve could have been pressed upon without the sixth and the third nerves having been first paralyzed. Moreover, Romberg found other changes in the affected parts, the nature of which one cannot determine from his account of them, namely, a hard, yellow nodule in the crus cerebri, and a softened state, with reddening, of the roots of the fifth nerve. It does not appear that this disease is ever of "reflex" origin or caused by peripheral irritation of any branch of the fifth or of other nerves. There is, indeed, Mr. Jeffries' oft-quoted case, in which a triangular piece of china from a broken cup remained lodged in the cheek of a girl, and gave rise, for fourteen years, to violent pains, which ceased a few weeks after its removal by excision. But that patient was too young to have suffered from true tic douloureux. Mr. Tomes says that this complaint is never, so far as he knows, caused by irritation from diseased teeth; he quotes, as a warning, a case of Trousseau's, in which the pain came on when the patient touched with the tip of his finger his few remaining teeth, but in which they were extracted without the slightest benefit resulting. Some years ago, a patient, who had in vain had a large number of his teeth removed, used to come to me as an out-patient.

On the other hand, there is a less intense but a much more common form of trifacial neuralgia, to which even young persons are liable, and which is often dependent on morbid conditions of the teeth. The pain shoots and darts along the branches of the fifth nerve, but it is generally accompanied with a dull, aching or gnawing sensation. It is often distinctly paroxysmal, and (like all other forms of neuralgia) it is especially apt to come on when the patient is over-fatigued or exhausted from want of food. It is seldom severe enough to prevent him from attending to duties or pleasures, although it

may cause him great discomfort. In many cases it subsides at once if a glass of wine be taken or a dose of quinine. According to Mr. Tomes, the most frequent cause of this form of neuralgia is chronic inflammation of a tooth pulp. But other conditions, which are mentioned by this writer and by Mr. Salter as being capable of giving rise to it, are the difficult eruption of a wisdom tooth (or its impaction in its socket), the presence of secondary dentine in a pulp cavity, exostosis, hypertrophy of the crusta petrosa, alveolar periostitis, decomposition of a dead pulp in a confined space, and even the exposure of sensitive dentine, or the crowding of the teeth together, from insufficiency of room. In many instances the affected tooth is tender, so that sudden pressure on it, or the contact of substances much hotter or colder than itself, greatly augments the pain. But sometimes there is no local indication of any dental disease. Mr. Tomes points out that this may be the case even where an inflamed pulp is exposed, if the cavity is so situated as to be out of the way of irritation by particles of food. Mr. Salter has related one instance in which, although the offending teeth were painful when touched, doubt for a time prevailed as to the real cause of the neuralgia from which the patient suffered, because this returned again and again, there being only a temporary interval of ease after each tooth in succession had been extracted, until the last two were removed at one operation. The pain then entirely ceased. All the teeth had nodules of exostosis on their fangs. It might be expected that any affection of a tooth in the lower jaw should give rise to neuralgia in the course of the branches of the third division of the fifth nerve, and that a diseased tooth in the upper jaw should set up pain in the distribution of the second division. But this is not necessarily the case. Indeed, Mr. Salter mentions, as very frequent seats of "dental neuralgia," the supraorbital nerve, the globe of the eye, the temple, and particularly a spot a little to one side of the vertex. I shall have again to advert to this when speaking of migraine. The practical rule would seem to be that the only way of avoiding the risk of overlooking affections of the teeth as causes of the milder forms of trifacial neuralgia is to have the jaws thoroughly examined by the dentist in every case. Mr. Salter has recorded some very curious instances in which the nutrition of parts, which were the seat of a reflex neuralgia due to caries of the teeth, underwent perversion; one in particular, in which the iris of the affected eye, from being of a deep hazel color, became of a dull gray.

The late Dr. Anstie believed ("*Lancet*," 1866) that in certain cases he traced a neuralgia of the fifth nerve to the "reflex" disturbance caused by injuries to distant nerves; once to a knife wound, by which the occipital nerve was divided, and twice to sickle wounds of the wrist involving the ulnar nerve. In each instance the pain first came on when the injury was already repaired, and when the cicatrix had become firm.

It must also be borne in mind that pains undistinguishable from those of neuralgia, often constitute the earliest symptom of various diseases and tumors of the maxillary and other facial bones, and that such cases may come under the observation of the physician at a time when there is no obvious deformity or swelling to lead the patient to seek surgical advice. There is in the museum of Guy's Hospital a specimen of Bright's, in which a morbid growth which had started from the sphenoidal sinuses made its way into the middle fossa of the base of the skull, and doubtless pressed upon the fifth nerve; the patient, a woman of forty, whose countenance showed strong indications of suffering, had complained chiefly of an extremely severe pain on the corresponding side of the face, which became more violent in paroxysms.

Neuralgia Affecting the Cervical Nerves.—"Cervico-occipital" and "cer-

vico-brachial" neuralgia. These affections may be dismissed after very brief consideration. Among the ascending branches of the cervical plexus, the great occipital, or posterior division of the second spinal nerve is the one to which neuralgic pain is most often referred. Such cases are often due to the direct action of cold. Dr. Anstie speaks of a patient who was several times attacked after sitting in a draught which blew strongly on the back of the neck. One must, of course, not overlook the existence of disease of the cervical vertebræ.

Cervico-brachial neuralgia generally affects several of the branches of the brachial plexus. Numerous "tender points" are developed, most of them at spots where the nerves pierce the fascia, but some (as, for instance, one at the inferior angle of the scapula) where their occurrence is not so readily explicable. One special feature of this affection is its liability to be aggravated by muscular movements of the arm. Anstie alludes to a case in which, at a time when convalescence seemed to be almost established, the act of playing on the piano for half an hour at once brought the pains back. Mr. Salter has shown that neuralgia affecting the nerves of the upper limb is sometimes due to diseases of the teeth. He says that pains in the shoulder and acromion, over the insertion of the deltoid, or at the bend of the elbow, not rarely depend on such causes; and he cites the case of a lady who, whenever any of the teeth in the left side of the lower jaw became tender from caries, was immediately attacked with severe neuralgia at a circumscribed spot in front of the forearm. Among the rarer causes of pains in the arms that might be mistaken for neuralgia, I may mention disease of the articular processes of the cervical vertebræ. In 1876 a woman was admitted into Guy's Hospital under Dr. Wilks, who had for four months been suffering from pains in the right shoulder, round the side, and down the arm to the tips of the fingers. No cause for her complaints could be discovered, but after a few weeks she was attacked with pneumonia, and died of that disease. Dr. Goodhart made a post-mortem examination, and found that there was a quantity of inflammatory exudation outside the notch between the seventh cervical and the first dorsal vertebræ. This had its origin in disease of the joint between these two vertebræ; the lowest root of the brachial plexus was surrounded by it.

The *neuralgiæ* of the *dorsal* and *lumbar nerves* do not need to be described in detail. They are less frequent than those of the face or limbs, but agree closely with cervical neuralgia.

Sciatica.—On the other hand, one of the most important of all the varieties of neuralgia is that which attacks the great sciatic branch of the sacral plexus, and which is consequently known by the special name of sciatica. The part of the nerve which most commonly suffers is that which lies behind the upper half of the femur, but sometimes it includes the whole length of all the main branches; in some instances it is even limited to the back of the knee, or to the terminal twigs in the calf or the foot. Its characters differ widely from those of some other forms of neuralgia, such as tic douloureux. Instead of consisting mainly of paroxysms of acute pain, darting like lightning through the nerve, and separated by intervals in which the patient is almost free, it is a constant, heavy, gnawing sensation; if it runs along the affected nerve at all, it does so slowly; its intensity is not, indeed, ever absolutely fixed and uniform, but the variations are comparatively slight and uncertain. It often appears to be worse at night, but this is ascribed by Dr. Henry Lawson (who was himself a sufferer from sciatica) to the fact that the patient then has less to distract his thoughts than in the daytime. Few diseases induce more peevishness, restlessness, and misery, spoiling the appetite, perverting sleep, and interfering with every kind of occupation, as well as with all games and amusements.

In advanced and severe cases, the pain sometimes radiates into the sciatic nerve of the opposite side, or to branches of the lumbar plexus, or to nerves which are still more remote.

The pain is often greatly increased by pressure on the sacral plexus within the pelvis, or at its exit from the sciatic notch, and thus the act of defecation may be attended with much suffering, and the patient may be fearful to sneeze or cough. He is also very susceptible to a draught of cold air.

When sciatica has lasted for any length of time, several "tender points" are generally to be found; one is said to be situated where the nerve emerges from the pelvis, others where its branches pierce the fascia lata, and others over the sacrum, the crista ilii, etc. The whole length of the nerve also becomes unduly sensitive to pressure, and especially (as I have already remarked) that part of the external popliteal nerve which lies under cover of the biceps tendon. I have never met with a case in which the patient did not complain of much more pain and tingling when I rolled this nerve beneath my finger on the affected side than when I did the same thing on the healthy side.

Wasting of the muscles is another symptom which is present in all cases of sciatica of which the duration has been sufficiently long. The buttock becomes flattened and flabby, the back of the thigh and calf are reduced in size and feel soft and flaccid. In one case Anstie found the faradic excitability of the gastrocnemius much diminished. It is therefore a mistake to suppose, with some writers, that the loss of power depends merely upon disuse of the muscles. Even within the first few days, the patient often begins to limp in walking, so that he puts only his toes to the ground and is glad to make use of a stick. Afterward he lies on the sofa all day long, with every joint of the limb bent, dreading to make the slightest movement. At last the hip and knee joints may become rigidly flexed, so that any attempt to straighten them causes severe pain. Painful cramps in the flexor muscles of the toes may come on, especially when the patient is just falling off to sleep. Sometimes fibrillary tremors are observed, or even shaking movements of the limb.

A certain degree of anæsthesia or impairment of tactile sensibility appears to be of constant occurrence in the more severe cases of sciatica, and subjective sensations of numbness and tingling are often present. The affected limb is sometimes paler and commonly colder than the opposite one; the patient occasionally complains of a sensation of chilliness in it, and it is said that the difference of temperature may be detected by surface thermometers.

The fact that the symptoms of sciatica are to a certain extent unlike those which are commonly regarded as characteristic of the neuralgiæ, has led to some diversity of opinion with regard to its nature. The writers of a few years ago regarded it, not as a definite complaint, but as a group of affections due in different cases to widely different causes. Thus Sir Thomas Watson spoke of it as being sometimes inflammatory, sometimes plainly a part of rheumatism, sometimes the result of irritation within the pelvis, or connected with a disordered state of the kidney, and, lastly, as sometimes purely nervous and neuralgic.

It is most important that one should not set down to "sciatica" the effects of pressure upon the sacral plexus and sciatic nerve from organic disease of whatever kind. I have myself published in the "*Guy's Hospital Reports*" (vol. x, 1864) a case that was regarded as an example of this complaint, until the patient unexpectedly died; whereupon the gluteal region was found to contain a large aneurism formed upon an abnormal artery, which passed down through the sciatic notch and along the back of the thigh, and constituted the main source of supply to the lower limb. That

case is, I believe, unique; such a course of the vessel (which is normal in birds) being one of the rarest of all abnormalities in the human subject. But a precisely similar pain might probably be caused by an aneurism upon one of the regular arteries of the buttock, or by a bony or sarcomatous outgrowth from the sacrum or os innominatum, pressing upon the nerve.

But, after all, such affections are comparatively seldom met with, and it appears to me that in all ordinary cases sciatica has as much claim to be regarded as a substantive disease as most other members of the nosology. I quite agree with Anstie, that its relation to rheumatism is altogether imaginary, at least, if any definite meaning be attached to the word "rheumatism;" and I am not inclined to think that the points which have been supposed to distinguish it from other forms of neuralgia are really fundamental. Many of them are perhaps attributable to the large size and the superficial course of the affected nerve, and to the way in which it is exposed to irritation when the patient walks about or even sits.

The first case of sciatica that came under my treatment in private practice occurred in a gentleman who has since been to me for neuralgia affecting the nerves of the brachial plexus; and Anstie cites in his work three or four cases in which there appeared to be a tendency to neurotic diseases in other members of their respective families, or the patients themselves had had other forms of neuralgia. One of them occurred in a lady whose paternal grandfather had suffered from sciatica.

Other writers, however, deny that inheritance or predisposition plays any important part in the ætiology of the disease, and there appears to be no doubt that it is far more commonly traceable than are other neuralgic affections to accidental circumstances, especially to the action of cold. Lawson states that nine-tenths of all cases of sciatica are referable to this cause, and Erb says that it is pre-eminent above other causes; exposure to a draught of air when the body is heated, wetting of the clothes, sleeping on damp ground or against a damp wall, have so frequently been followed by sciatica that no doubt can be entertained of their power to excite the disease.

Another frequent antecedent appears to be over-fatigue of the lower limbs. In Lawson's own case the complaint came on after a long walk; and some writers have described it as common in those who work hard at the sewing-machine.

The fact that certain classes of persons are exposed more than others to the exciting causes of sciatica probably explains the circumstance that it is more common in men than in women (according to Erb, in the proportion of four to one), and in adults between the ages of twenty and sixty, persons from twenty to forty years old being still more subject to it than those who are older. Lawson, however, says that he has seen one well-marked case in a boy, aged fourteen, who was addicted to masturbation.

The *diagnosis* of sciatica is seldom difficult to the practitioner who is alive to the numerous other diseases that may cause pain in the thigh and leg. Where the calf is the part mainly affected, one must think of thrombosis of the femoral vein, but this can be excluded by digital examination of the vessel at the groin, even if the complaint should be too recent to have caused œdema of the ankle. Disease of the hip-joint can be put out of consideration by the fact that neither pressure on the trochanter, nor forcing the head of the femur against the acetabulum, gives rise to pain; and disease of the sacro-iliac synchondrosis by the fact that no tenderness is elicited by a blow over that articulation. Advanced cases, in which the patient limps in walking and has wasting of the muscles, are very likely to be mistaken for spinal disease; this occurred some years ago in the case of a medical man who was under my care, and whose brother practitioners in his own neighborhood all felt sure that there was something the matter

with his vertebral column. Such disease is, indeed, very unlikely to have its effects confined for a length of time to one limb; but, on the other hand, I have already remarked that, when sciatica is at its height, the pain often radiates to the sciatic nerve of the opposite side. Even if one is satisfied as to the seat of the pain, one must still search carefully for local causes of irritation. I remember to have heard of one very instructive case in which a surgeon, feeling carefully along the course of the nerve, was fortunate enough to detect the presence of a bit of broken needle, the removal of which led at once to the cure of the patient.

Prognosis and Treatment.—The duration of neuralgia in general and its amenability to treatment vary greatly in different forms of the disease, as well as in different cases of the same kind. The most protracted and obstinate form of all is Tic douloureux; most writers speak of it as incurable. Even this, however, is a very long time in wearing out the patient's life. Trousseau mentions a case in which it went on for thirty years. In the other varieties of neuralgia, it is altogether exceptional for the disease to last more than a few months; but Anstie alludes to some instances of sciatica as defying all medical skill. In forming a prognosis, one is to a great extent guided by the age of the sufferer; the older he is the more likely is the course of the disease to be prolonged. Recovery is generally gradual, and it is apt to be interrupted by relapses. Even after the pain is gone, the affected part sometimes feels stiff for a long time, and its muscles are quickly fatigued by exertion.

The treatment of neuralgia is a matter which often taxes to the utmost one's patience and skill. Hygienic conditions must be very carefully attended to; fresh air, regular bodily exercise, freedom from worry and overstrain of mind, plenty of sleep, an abundant supply of wholesome nutriment, are each essential. Anstie laid special stress on the importance of a liberal diet, and particularly on the value of fat as an article of food. Whenever he could, he made his patients take cod-liver oil; if the stomach revolted at that, he insisted on their eating butter, cream, Devonshire cream, or sometimes olive oil, or even cocoanut oil. He strongly objected to allowing sufferers from such affections to have wine or brandy, excepting in very moderate quantities and with the meals. I entirely concur with him in the protests which he again and again uttered against the practice of prescribing stimulants for the mere relief of pain or of depressed spirits.

Among the medicines administered by mouth, *arsenic* appears to hold the first place. Any one of the pharmacopœial preparations may be given in ♏iij doses, gradually increased to ♏viij. I prefer the liquor sodæ arsenitis, because I have several times found that patients could take it without inconvenience who were attacked with diarrhœa and vomiting if they were placed on Fowler's solution, which is more commonly prescribed. Or the liquor arsenici hydrochloricus may be used, alone or in combination with the tinctura ferri perchloridi, itself a remedy to which Anstie attached a special value.

Another agent, to which great attention has been directed within the last few years, is *phosphorus*. Its employment in neuralgia has been especially advocated by Dr. Ashburton Thompson. The formula to which he gives the preference is a solution of the drug in cod-liver oil, in the proportion of one grain to an ounce and a half; of this a fluidrachm, containing one-twelfth of a grain, is administered every four hours. Other vehicles are alcohol and ether. Or the phosphide of zinc may be used in doses of one-half to one-third of a grain every two hours; the great objection to this compound is its tendency to cause vomiting. Or, again, phosphorus may be made into pills with resin or wax; the Pharmacopœia contains

directions for their manufacture; but it has been shown by Dr. Rees that such pills are apt to pass through the intestinal canal without being dissolved. However, pills made according to certain private formulæ are said to be effective. Capsules containing phosphorus are also much employed at the present time. Dr. Thompson gives a table of fifty cases of neuralgia treated by phosphorus, in several of which the effects of the remedy seem to have been very remarkable; it often happened that relief was afforded in a few hours; and, according to this writer, if marked results are not attained within three days it is useless to persevere longer.

Galvanism is often very valuable in the treatment of neuralgia. One of the poles of a "*constant current*" battery may be placed close to the spine of the affected side, or near the roots of the painful nerves, the other being applied upon the various "tender points" in succession, or being perhaps immersed in a vessel of water into which the hand is also dropped. A "descending" current is usually employed, the positive pole being put nearer to the centres than the negative one, but reversal of the poles seems to make no difference in the result. A good example of the effect of this form of electricity is afforded by a case related by Anstie in the fourth volume of the Clinical Society's "*Transactions.*" A woman had for nearly five months suffered from cervico-brachial neuralgia, which lasted for several hours each day, and had defied all kinds of medicinal treatment. The very first application of a battery of from ten to fifteen cells arrested the pain; the attack which followed was much less severe than usual; and after thirteen days the complaint ceased to recur. The treatment was continued altogether for six weeks. Another case in which great relief was afforded was read to the Clinical Society at the same time by Dr. Buzzard.

But it occasionally happens that this continuous galvanism fails. Faradization of the nerve is rather injurious than beneficial, but the application of a powerful current to the skin by means of the electric brush is said to be sometimes very serviceable as a means of severe counter-irritation. Its action is similar to that of *blisters*, which are undoubtedly of great value in many cases. Anstie recommended that they should be placed not upon the painful part itself, but close to the spine at the level of the affected nerves. Even in the desperate tic douloureux of old age he sometimes found that a blister applied to the nape of the neck removed the pain entirely for a long time. But in sciatica he obtained better results by placing flying blisters over the nerve trunk in the gluteal region. Mustard plasters, chloroform liniment, and other rubefacient applications, may also be prescribed in mild cases with advantage.

The remedies which would naturally first suggest themselves for so painful a disease as neuralgia are *opium* and morphia, and very few cases occur in which recourse is not had to them in one form or another. Yet one should be very cautious in administering them at all freely, at least when there is any hope of the ultimate subsidence of the complaint. In the incurable tic douloureux the objections to their use do not apply; and Trousseau employed opium in that affection with not a little success, increasing the dose boldly until sometimes as much as a quarter of an ounce or even half an ounce of laudanum was taken daily. In a few instances the result was more than a palliation of the patient's misery. The frequency and severity of the attacks of pain were lessened, so that after a time the drug could be discontinued. And although a relapse occurred a few weeks later, the interval of ease was a great gain; for food could be taken, which had before been almost impossible, so great was the suffering caused by the act of mastication.

But in many cases the administration of opium by the mouth disorders the digestive organs. Hence the subcutaneous injection of morphia is

greatly to be preferred, for it is attended with no such ill effects; and of late years this method of treating neuralgia has been very extensively employed. The quantity should be very carefully regulated; not more than a sixth of a grain (two minims of the pharmacopœial solution) should be used on the first occasion; in mild cases it is even well to begin with one-twelfth of a grain. If relief is not afforded by such doses larger ones may be tried; a quarter of a grain, half a grain, even a grain, is commonly injected under these circumstances; and sometimes very much larger quantities still. One seldom has to use the syringe oftener than twice in the twenty-four hours; but Anstie laid stress on the importance of performing the operation as early as possible after the commencement of an exacerbation of the pain. It seems to be almost a matter of indifference whether the morphia is injected at the seat of pain or at any other part of the body, except so far as concerns the effect on the patient's imagination.

The effect of subcutaneous injection of morphia is often marvelous. Suffering which had been almost intolerable is removed, as by magic, within a few minutes. And this is not infrequently the first step toward the complete cure of the disease. It was so, for instance, in the severe sciatica of which Dr. Lawson was himself the victim, and in which so many other plans of treatment had been tried without the least good result.

This method is not, however, altogether free from evils of its own. Dr. Oliver has related in the "*Practitioner*" a case in which immediately upon the injection the patient cried out with an expression of great alarm, and the pulse became very small, the face red, and the eyeballs prominent. Brandy was given, and in about half an hour these symptoms passed off. It is thought that the alkaloid may have been thrown directly into a small vein. But such an accident is excessively rare. A much more serious risk is one to which Dr. Allbutt has especially drawn attention, namely, the setting up of a "morphia habit," a peculiar state of depression and irritability, which can be relieved by nothing but a fresh dose. There is also reason to fear that the remedy itself sometimes renders the pain more intense than before, or at least diminishes the intervals between the exacerbations of it. Even when the original disease is cured, patients sometimes experience great difficulty in discontinuing injections of morphia to which they have been accustomed; a vigorous effort may be needed, and the endurance of much discomfort and even misery for several days. For this reason, as well as for others, the syringe should not be entrusted to the hands of the sufferer himself, or even to those of a relative or servant, unless, perhaps, sometimes when his complaint is incurable. Anstie, however, laid stress on the fact that the evils connected with the establishment of a morphia habit never arise when the dose is kept below a certain point. In a valuable paper in the "*Practitioner*," he insisted strongly on the importance of "economy" in the use of the alkaloid; and he related the case of a lady who for three years had enjoyed a complete immunity from a severe sciatica, under the daily use of a dose of morphia, which was at first one-eleventh or one-twelfth of a grain and was never raised above one-fourth of a grain.

Zona.—*Herpes zoster—Shingles.*—To complete the description of affections of the peripheral nerves, up to their nuclei in the cerebro-spinal centres, I must now give an account of a very remarkable disease, of which the most constant symptom is a cutaneous eruption. It has hitherto been claimed by the dermatologists, but appears to be the result of a morbid change in one or more of the ganglia upon the posterior nerve roots. The names for this disease owe their origin to the fact (with which the elder Pliny was acquainted) that it passes round the trunk of the

body like a girdle; even the English word "shingles" is a corruption of the Latin *cingulum*. The brief notice of it given by Pliny also implies the knowledge of another striking feature, namely, that it is limited to one lateral half of the cutaneous surface. He says "*enecat, si cinxerit,*"—"it kills, if it encircles." A popular tradition to the same effect still exists in England. Nevertheless, zoster is never fatal, and it really has no tendency to pass over to the other side of the body from that which happens to be first affected by it.

The most elementary acquaintance with anatomy could not fail to suggest, to any one who had observed the distribution of the eruption in a case of shingles, that it corresponds exactly with that of the peripheral distribution of one or more of the dorsal nerves. Accordingly this has been recognized for many years; and with it, the necessary consequence, that certain eruptions on the face and limbs, which follow the course of the nerves supplied to those parts, are identical with it. To these also the name of herpes zoster is now given by general consent, although when so applied it really loses all meaning, since the affected area no longer has the form of a belt.

The *eruption* of shingles consists of vesicles. These are of flattened form, and larger than those of eczema, being often as big as split peas; they are arranged in clusters of perhaps twenty or thirty, each cluster lying on a reddened and slightly swollen patch of skin; when the vesicles are thickly set, they often run together, and form flat bullæ of very irregular shapes. Some years ago, Dr. Haight, of New York, found an opportunity in Vienna of investigating their structure with the microscope. His observations showed that their roofs consist of the horny layer of the cuticle, with some of the superficial elements of the rete mucosum adherent to the under surface; their floors are formed by the bare summits of the papillæ, with the deepest elements of the rete occupying the depressions between them; their cavities are traversed by numerous bands, consisting of masses of the intermediate elements of the rete, drawn out into long spindle cells and cells with several tapering processes. The fluid which the vesicles contain is at first transparent, but after a time the presence of floating leucocytes renders it opalescent, and ultimately it may become purulent, or acquire a purple color, from the escape of blood through the softened tissues beneath. The cutis itself seems always to take part in the inflammation to some extent, leucocytes being scattered in the spaces between its fibrous bundles, and along the vessels and nerves. But when pus is formed, the number of cellular elements in its tissues may undergo an enormous increase, so that if the roofs of the vesicles should have been removed by the friction of the clothes (which often seems to determine the occurrence of suppuration), ash-colored surfaces are exposed, looking like layers of false membrane. In other words, the histological changes in zoster are essentially identical with those which have been described, at p. 242, for variola.

The number of clusters is very variable, from a single one to ten or even more. They are generally developed, not all at the same time, but in quick succession; those coming out first which lie most toward the upper distribution of the nerve whose branches they follow. After a very few days fresh ones cease to make their appearance. There is a short papular stage; and some of the latest clusters not infrequently abort, without going beyond it. In certain very mild cases, when only one or two clusters are formed, none of them pass into a vesicular condition. Even if the disease should be of considerable severity, the eruption begins to dry up from the fifth to the eighth day; the centres of the vesicles become depressed and horny, yellowish or brownish crusts form upon them, and in the course of the third week these fall off, leaving reddish or purple stains. But, when the cutis

is thickly infiltrated with pus cells, its superficial layer undergoes destruction, and an eschar has then to be thrown off: thus the process of healing is retarded, and an indelible cicatrix results. The distribution of such cicatrices in the course of a particular nerve shows at once the nature of the disease from which they arose. A child was once brought to me with a series of little keloid tumors scattered over the branches of some of the nerves of the arm; their origin seemed at first to be inexplicable; but I presently traced them to an eruption of shingles, which (as I learned) had occurred about a year before. I need not add that whenever zoster attacks the face (or, in a female, the neck, or the arm) one must not fail to warn the patient of the possibility that it may leave permanent scars behind it.

Writers describe, and have given names to, numerous varieties of zoster, according to the anatomy of the nerves whose distribution is followed by them; but such distinctions are useless. When the disease affects the face, the nerve which it follows is the fifth, the greater part of which is perfectly analogous to the sensory portion of an ordinary spinal nerve. Indeed, it is remarkable how exactly the clusters of vesicles sometimes map out the points of emergence of the several twigs of the trifacial nerve from their bony canals. When the first division of the nerve is affected, the loose tissue of the upper eyelid becomes extremely œdematous and swollen, so that the affection is very likely to be mistaken for erysipelas by a careless or inexperienced observer. Another peculiarity of this form of shingles is that it is often attended with ulceration of the cornea and iritis, by which the sight may be very seriously damaged. Hutchinson, who has specially investigated such cases, has remarked that the ocular affection never arises unless the eruption occupies the distribution of the nasal twig. When the two lower divisions of the trifacial nerve are involved, a few vesicles often appear on the mucous membrane of the mouth and palate. Paget has recorded an instance in which necrosis of the alveoli followed the eruption, so that some of the teeth fell out.

Cervical zoster, and that which affects the upper limb, follow exactly the distribution of the several nerves. In some instances the vesicles reach down to the fingers, but this is very exceptional; in the great majority of cases they do not extend below the elbow. On the trunk, which appears to be by far the most frequent seat of the disease, the area occupied by the eruption, of course, slants more and more downward as it approaches the pubes. It often happens that one or two vesicles lie slightly beyond the meridian plane, both at the linea alba and at the spine; this probably depends upon the fact that the nerves of the opposite sides overlap in their distribution, just as in the Siamese twins there was a part of the connecting band which received nervous filaments from each of them. In the lower limb the distribution of zoster presents this peculiarity, that it is almost invariably confined to the buttock and thigh. Hutchinson says that it never extends below the knee, and the only instance that I know of to the contrary is one figured by von Bärensprung, in which there were a few small papules as low as the middle of the calf.

I have already remarked that the eruption of shingles has no tendency to encircle both sides of the body. Hutchinson once saw a zoster in the course of the fourth dorsal nerve on the *right* side associated with a frontal zoster on the *left* side. In one of von Bärensprung's cases an ordinary zoster, limited to the *right* half of the thorax, was accompanied by a single vesicle in the *left* axilla, the patient having been suffering from severe burning pains on both sides.

An attack of shingles is very seldom attended with any febrile disturbance or disorder of the general health. In children, who are liable to it at all periods after the first year, it commonly runs its course without any

unpleasant sensation, or is merely accompanied by a little numbness and tingling. Von Bärensprung tested the cutaneous sensibility with a pair of compasses, and found that in two cases it was considerably increased, while in a third it was diminished. Sir Thomas Watson relates a curious case in which the eruption affected the scalp, and in which the patient, who had for seven years been plagued with continual noises in the head, became free from this symptom on being attacked by the shingles, and for eighteen months afterward. He also mentions another case, of a man in whom the eruption came out in February and who suddenly lost a cough which had teased him all the winter. On the other hand, von Bärensprung met with two cases of zoster affecting the distribution of the fourth cervical nerve, in each of which vomiting occurred at the commencement—a consequence, he supposes, of "sympathetic irritation" of the vagus and phrenic nerves.

But by far the most important subjective symptom of herpes zoster is pain of a neuralgic character and referred to the same nerves the distribution of which is followed by the eruption. This is entirely absent in young patients, but in adults it is generally present, and in old people it is apt to be exceedingly severe. Even the slightest touch may then give rise to extreme suffering. Von Bärensprung cites a case in which there was only a single patch, of the breadth of two or three fingers, but in which the tenderness was such that the patient kept the part covered with the palm of his hand all night and all day, lest his shirt or his bed linen should come into contact with the vesicles. When the eruption gets well the pain commonly subsides; but in some instances it continues long afterward, for months or even for years, with scarcely any abatement of its intensity. I have myself known a gentleman who was still suffering at the end of ten years; and Trousseau mentions an instance in which the pain lasted for fourteen years. Sir Thomas Watson alludes to a case of this kind in which the patient, a lady, could at all times bring on the pain (this being situated along the edge of the left ribs) by drinking some cold liquid. I believe that the persons in whom shingles leaves behind it this terrible infliction are always advanced in age.

Again, the pain sometimes precedes the development of the eruption by several days. This has led certain writers, among whom was Anstie, to regard herpes zoster as a mere complication of neuralgia, analogous to several other curious "trophic" changes which are met with in that disease. But in opposition to such a view it must be urged that in the majority of cases of shingles (in two out of three according to von Bärensprung) pain is altogether absent, and that when it is the earliest symptom the cutaneous affection always appears within a definite period, a fortnight being the longest interval that I have heard of. Some instances have, indeed, been recorded of an eruption, regarded as zoster, which seemed to be caused by pressure upon the corresponding nerve trunks, or which occurred in the course of an ordinary neuralgia. Thus Charcot and Cotard have published a case in which one-half of the neck and one shoulder were covered with the vesicles, the cervical nerves of that side being compressed by cancerous disease of the vertebræ. Charcot is said to have met with another case in which a patient, during a second attack of sciatica, presented herpetic vesicles on the lower part of the thigh. In these instances it seems not improbable that the eruption really was shingles, and that it was caused by an extension of the morbid process from the trunks of the nerves to the ganglia on the posterior nerve roots. But there are other cases to which such an explanation is less readily applicable. Thus von Bärensprung reports an observation by Esmarch (of Kiel) of abscesses about the pelvis and under the gluteus maximus, where *post-mortem* the great sciatic nerve was found to be swollen and reddened. About five weeks before the patient's

death an eruption of groups of vesicles appeared on the back of his leg and on the sole of the foot as far as its middle. In this instance the seat of the cutaneous affection was unlike that of a zoster, for, as I have already stated, that scarcely ever goes below the knee. The same objection applies to Charcot's oft-quoted case of a man who suffered from pain in the leg and back of the foot after a gunshot wound of the thigh, and in whom a herpetic affection repeatedly developed itself upon those parts. Moreover, the fact that the eruption recurred is of itself sufficient to show that it was not really a zoster, for, as Hutchinson has pointed out, shingles never relapses. He has, indeed, gone so far as to maintain that the complaint protects against itself, like the exanthemata. In a series of a hundred cases collected by him there was only one in which there was any history of a previous attack. But it appears to me that this depends simply on the fact that shingles is relatively of infrequent occurrence, and perhaps does not affect more than one per cent. of the population. Still, the fact that shingles has no tendency to recur, either in the same part or elsewhere, indicates a great difference between it and all ordinary neuralgic affections, and is, I think, conclusive against the opinion that it is a mere accidental complication of neuralgia.

If further arguments are needed, one may be found in the fact that herpes zoster is associated with a series of changes in the ganglia of the posterior nerve roots, such as are not known to occur in the neuralgiæ proper. That these ganglia are the starting points of the disease was, I believe, first suggested by von Bärensprung in 1861. His opinion was verified by Charcot and Cotard, who found (in a case already referred to) that whereas the nerve roots were healthy, the ganglia and the nerve trunks to a little distance outside the intervertebral notches were much reddened and slightly swollen, their stroma being also unduly rich in nuclei. Precisely similar appearances were afterward discovered by von Bärensprung himself in a little child which died soon after an attack of shingles.

With regard to the *causes* of herpes zoster almost nothing is known. It occurs equally in both sexes and at almost every age. Hutchinson has repeatedly observed it in persons who have recently been taking arsenic medicinally, and I have myself noticed the same thing more than once.

In the *treatment* of this disease it is generally supposed that there is but little to be done. The vesicles must be protected by a soft linen rag, with a pad of cotton wool or a flannel bandage. Some writers recommend that flexible collodion should be painted over them to facilitate their drying up; or a little starch powder may be dusted over the affected part as soon as any discharge appears. Children require no medicine whatever.

Dr. Ashburton Thompson has related ("*Glasgow Med. Jour.*," 1874) the case of a youth to whom he administered one-third of a grain of zinc phosphide every three hours, on the second day of pain in the right side. Some vesicles of zoster were then already becoming opaque, but one patch was still incipient. Next day the eruption began to dry up, and by the second day of treatment all pain was gone.

The treatment of the neuralgia which sometimes follows shingles is unsatisfactory. Bazin is said to have used arsenic with success; but very often it utterly fails. Of late years I have been in the habit of prescribing the vinum colchici; in several instances the pain has quickly subsided while the patient has been taking this medicine, and, as I believe, in consequence of its administration. Locally, anodynes are generally applied, but without much benefit; I remember one case in which a weak arnica lotion appeared to give great relief. Von Bärensprung says that he has always found blisters useful.

AFFECTIONS OF THE SPINAL CORD.

Paraplegia—ITS SYMPTOMS—INCOMPLETE PARAPLEGIA—REFLEX MOVEMENTS—KNEE-JERK AND CLONUS—THE BLADDER IN PARAPLEGIA—THE RECTUM AND GENITAL ORGANS—AMMONIACAL URINE AND ACUTE BED SORES.
Myelitis—HISTOLOGY OF ACUTE MYELITIS—OF CHRONIC MYELITIS OR SCLEROSIS — ÆTIOLOGY — COURSE — DIAGNOSIS FROM SPINAL IRRITATION, ANÆMIA AND CONGESTION OF THE CORD, REFLEX AND HYSTERICAL PARAPLEGIA, CONCUSSION, ACUTE ASCENDING PARALYSIS, ALCOHOLIC AND SYPHILITIC PARAPLEGIA, HEMORRHAGE, TUBERCLE AND OTHER TUMORS—PROGNOSIS AND TREATMENT.
Hemiparaplegia—RACHIALGIA—PARAPLEGIA FROM COMPRESSION.
Meningeal Hemorrhage—ACUTE AND CHRONIC SPINAL MENINGITIS.
Spastic Paraplegia—IN ADULTS—IN CHILDREN—ANATOMY—TREATMENT.

Within the last few years our knowledge of affections of the spinal cord has made great progress; certain lesions can now be positively diagnosed which were entirely unknown to the pathologists of the last generation. Unfortunately, however, the advance has been mainly in regard to diseases which are comparatively rare. Upon the more common ones much less light has been thrown; and their diagnosis still remains matter of presumption rather than of certainty, being based mainly upon the known frequency of one affection under particular circumstances. This, indeed, is not peculiar to the cord; as much might be said of the liver or the lungs. But I think that there is no other region of the body in which the morbid changes revealed by an autopsy are so often unexpected during life.

To avoid going over the same ground again and again, I shall begin by discussing one symptom—Paraplegia—which belongs to most spinal affections in common; afterward I shall pass on to consider each affection separately, with its other symptoms.

PARAPLEGIA.—This is a form of paralysis possessing two essential features: first, that it affects both sides of the body, and generally to an equal extent and in the same degree; and, secondly, that it affects all parts, up to a certain level, according to the functions of the several pairs of spinal nerves, ascending from the sacral to the cervical. It is easy to see why affections of the cord should produce such a symptom. On the one hand, the motor tracts belonging to the two halves of the body are in the cord so close to one another that they are generally involved in the same lesion. On the other hand, since each segment* of the cord contains fibres belonging to all the nerves below, they are all likely to suffer together. Although Sir William Gull and other writers have spoken of a "cervical paraplegia," when the arms are paralyzed without the legs, I think the expression is incorrect. In such cases, the affection, if it lies within the cord at all, must be definitely limited to a part only of the segmental area; and between them and cases of ordinary spinal paralysis there are many important differences. One cannot insist too strongly on the fact that the morbid changes which cause paraplegia acknowledge no such limitations. It is true that a lesion may extend gradually across the cord, and, therefore, the resulting paralysis may for a time be incomplete; but this is a different matter.

* A "segment" of the cord is that part of it which would be included between any two adjacent complete horizontal sections. It takes the *whole thickness* of the cord.

As regards their distribution in the length of the cord, the lesions in question vary widely. They may be strictly confined to a single spot, or may extend from the cauda equina to the cervical region.

The *upper limit* of a spinal affection is, as a rule, roughly indicated by the extent upward of the paraplegia to which it gives rise. Disease of the lumbar enlargement causes paralysis of the lower limbs; disease in the dorsal region, paralysis of the abdomen and chest, corresponding with the level to which it reaches; disease of the cervical enlargement, paralysis of the upper limbs; disease still higher up, paralysis of the diaphragm and of the cervical muscles. In some cases the feet and legs become powerless before the thighs and the hips. This is generally supposed to mean that the lesion is at first confined to the extreme lower end of the lumbar enlargement; but, Woroschiloff having found that in the lateral columns of the lumbar cord of the rabbit the motor paths for the distal parts of the lower limbs lie outside those for the proximal parts, it may, perhaps, sometimes be due to the gradual extension of a morbid process from the surface inward. When paralysis attacks the upper extremities in detail, as the result of disease spreading upward through the cord, I believe that the muscles of the hands are affected before those of the elbow and of the shoulder. The fact that the nerves to the ulnar side of the hand come chiefly from the lowest part of the brachial plexus would lead one to expect that the inner fingers should suffer earlier than the outer fingers or the thumb. I do not know whether this is the case; if not, we have here a distinction between lesions of the cord itself and those of the nerve roots. According to Erb (Ziemssen's "Handbuch," xi, 2, p. 62) paralysis of all four limbs, and of the trunk muscles generally, sometimes occurs without any interference with the respiratory movements, as the result of an exceptional limitation of disease to a part of the segmental area of the cord in the cervical region, leaving the lateral columns intact. Strictly speaking, we should not call such an affection paraplegic, in the sense in which I have defined that term.

When any segment of the cord is completely destroyed, there is, of course, an absolute loss of *sensation*, as well as of motion, in all parts of the body below. And when a total disorganization spreads upward through its substance, the gradual progress of the disease from day to day can be determined from the extension of the anæsthesia with far greater accuracy than is ever possible from that of the motor paralysis alone. But, in the great majority of cases, parts affected with paraplegia either retain perfect sensation, or have that function but little impaired. Now, where the lesion of the cord is such as may fairly be supposed to commence in the anterior part of it—as is often the case in "compression paraplegia"—one is inclined to attribute the absence of anæsthesia to that circumstance. But, in reality, the preponderance of loss of motion over loss of sensation is not only met with in all diffused spinal affections, but belongs to them in common with diseases of the peripheral nerves and of the brain itself. And the true explanation is, no doubt, that which was given when the diseases of the nerves were under consideration; namely, that sensory impulses are transmitted far more easily than motor ones, so that unless the conducting fibres are completely disorganized anæsthesia does not occur. Many patients, however, complain of *paræsthesiæ*, such as "pins and needles," sensations of "pricking" or "tingling" in the toes or in other parts, "creeping" or "crawling" feelings, subjective sensations of heat or of cold.

If the account of paraplegia in the last few paragraphs is correct, it would follow that this symptom, as it is seen in man as the result of disease, has little or no light thrown upon it by the experiments of those physiologists who have devoted such infinite pains to the investigation of the exact paths for the transmission of sensory and of motor impulses respectively

within the substance of the cord. And this, I believe, is really the case. I shall hereafter have to describe certain affections which are strictly limited to particular tracts of the gray or of the white matter ; but we shall find that none of these are characterized by a simple paralysis or anæsthesia of the parts below. On the other hand, I have already asserted that the lesions which cause paraplegia tend to diffuse themselves over the whole segmental area of the cord, and that no special localization of them is concerned in causing the preponderance of loss of motion over loss of sensation, which is so commonly observed. All physiologists are agreed that volitional motor impulses pass mainly along the lateral columns. But with regard to the paths for sensory impulses, there still is doubt. One opinion was maintained by Brown-Séquard, that they are situated in the gray matter. Another, which was first taught by Schiff, is that tactile sensations are transmitted through the posterior columns, but sensations of pain through the gray matter.* So far as I can judge, neither view, supposing its truth to be demonstrated, would at present be practically available in the regional diagnosis of affections of the spinal cord as we see them at the bedside. We shall find that a central tubercular nodule is occasionally the cause of paraplegia. I have not read, nor have I observed, that anæsthesia precedes the motor paralysis in cases of this kind, which seem to approach more nearly than any others to the conception of a lesion destroying the gray matter only, such as, on Brown-Séquard's theory, should produce that effect. On the other hand, it ought to follow, if Schiff's doctrine were correct, that such an affection should rather cause analgesia, tactile sensation being retained. But as yet, so far as I know, nothing of the kind has been noticed. It may, of course, be replied that such a nodule does not completely disorganize the gray matter before it presses on the white columns ; but this is the very point on which I would insist, namely, that the diseases which cause paraplegia are not so localized as to admit of the application of laws deduced from experiment. One question, however, which is of great clinical importance is that of the decussation of sensory fibres within the cord itself, immediately above their entrance into it from the posterior nerve roots. And another point of interest is the delay in the transmission of sensory impulses which is now and then seen in man as the result of disease, and which has also been observed in experiments on animals. Both these matters will be fully discussed hereafter.

Hitherto, I have spoken only of complete paraplegia, a condition in which the affected muscles are altogether incapable of being influenced by the will ; but in a large number of cases the paralysis is incomplete. Every degree of loss of power may be noticed. One patient can just move his toes, another can flex the knees so long as they lie horizontally on the bed ; a third can draw the thighs up to the abdomen ; a fourth can stand for an instant and then falls down ; a fifth can walk a few paces, and so on, up to a condition in which all that can be detected on the most careful examination is a slight uncertainty of step, with a tendency to catch one of the feet against anything that happens to project above the pavement. When the paresis is a little more marked than this the gait is peculiar, and it is well worthy of study because it contrasts with that which is observed in some other spinal affections that will be discussed further on. Erb describes it in the following terms: "The foot hangs down in walking, the toes are dragged, the sole is brought to the ground clumsily, and for the most part upon its outer edge ; the knee is too much raised or carried forward without being bent ; there is generally a kind of stiffness about

* [A third and better supported hypothesis places the chief sensory tracts in the deeper part of the lateral columns of the opposite side. But this most difficult problem is still far from satisfactory solution.—Ed.]

the legs. The patient uses one, or even two sticks, or he is held up by crutches or by the arms of other persons; he totters but slightly and stands quietly and steadily. When left unsupported, he sinks down upon the ground. The gait, of course, varies somewhat according to the number of muscles which are paralyzed. When only those below the knees are affected, it is waddling and very peculiar." In those cases of partial paraplegia in which sensation is also impaired the patient feels when he stands upon his feet as though they were wrapped in wadding or covered with thick woolen stockings; or a hard floor may seem to him like a thick felt carpet. I believe that he then always requires the guidance of vision to enable him to stand firmly or to walk evenly. We shall presently find that where a patient who has his feet close together totters or falls, as soon as he is made to close his eyes, many observers infer that he is suffering from a special disease of the cord—"sclerosis of the posterior columns" or "locomotor ataxy." I quite agree with Erb that although the symptom in question is often present in that disease, it is yet by no means peculiar to it, being met with whenever there is an imperfect transmission of those sensory impulses from the soles of the feet, and from the other parts of the lower limbs, by which the equilibrium of the body in the erect posture is normally maintained.

The *lower limit* of a lesion of the cord cannot be determined with any great accuracy. We have seen that disease in the cervical region renders the legs as well as the arms paraplegic, and this equally whether the affection is confined to a small part of the length of the cord or involves the whole of it down to the cauda equina. A very simple observation, however, enables us in many cases to say that at least some of the lower segments retain their functions, although their connection with the brain is cut off. We have but to see whether *reflex movements* can be excited in the legs by impressions upon the cutaneous nerves. If this experiment succeeds, it proves that the lumbar enlargement is still undestroyed. On the other hand, its failure is not to be taken as conclusive evidence that the disease extends throughout the whole of the lower part of the cord. On the contrary, it often happens in cases of very limited lesions that reflex movements can only now and then be elicited. For example, surgeons find, when there is fracture with displacement of the upper dorsal or cervical vertebræ, that for a few days after the accident there is an entire absence of reflex excitability in the cord; it seems to have been in some way deprived of its functions by the shock.* So also, we shall hereafter learn that the movements are generally less readily obtained when disease has begun in the interior of the cord than when it is merely compressed from without, the reason being that in the former case an "inhibitory" influence is transmitted downward upon the lower centres. Even when these retain or have regained their susceptibility, it may sometimes be easily exhausted for a while by repeated calls upon it. In a well-known paper, read before the Royal Medical and Chirurgical Society in 1839, Dr. William Budd mentions the case of a young lady in whom the slightest disturbance of the bed clothes caused very energetic contractions of the right leg, which was paralyzed; but there was a successive diminution in the vigor of the spasms on each renewal of the stimulus. The impressions that produce reflex movements are sometimes felt, and in the case of this young lady the movements themselves were attended with pain like that of cramp. On the other hand, Dr. Budd relates an instance in which all the limbs were paralyzed by an injury to the cord in the cervical region, but in which convulsions in the arms were excited by drawing on the hair of the head or by tickling the

* [See the instructive remarks of Prof. Goltz on this subject, in his "Verrichtungen des Grosshirns," S. 78–81.—Ed.]

chin; the efferent impulses must then have traversed the seat of lesion, through which no volitional efforts could be made to pass. As a rule, however, cases in which all communication with the brain is absolutely cut off are those in which the reflex activity of the cord is most marked; the patient then has no consciousness either of the application of the stimulus or of its effect, unless he sees his legs jerked upward. It seems probable that the susceptibility of the lower spinal centres is often directly augmented by an irritant influence transmitted downward from the diseased part, just as it constantly is by the administration of strychnia, which excites spasms in paralyzed limbs much earlier than in those which remain obedient to the will. Sensitive impressions upon the skin of the soles of the feet seem to set up reflex movements in the legs much more easily than similar stimuli applied to any other part of the cutaneous surface; lightly touching the hollow of the foot with a feather often throws the whole limb into spasm. As might be expected, a hot metal plate produces much more marked effects than one at an ordinary temperature, even when the transmission of sensory impressions is so imperfect that the patient cannot tell the difference. But the most violent convulsions of all are those which are sometimes excited by micturition and by defecation. One of Dr. Budd's patients was obliged, whenever he was placed upon the night stool, to have his feet inserted into two large loops of saddlers' webbing which were nailed to the floor, to have two larger loops adjusted over his knees, and to get an attendant to hold down his thighs; otherwise he was liable to be thrown forward upon the ground.

Reflex movements are always more or less perfectly co-ordinated. A single muscle may sometimes be thrown into contraction, as in the common experiment of making the cremaster draw up the testicle by touching the inner side of the thigh, but only when a similar action is performed normally. There seems to be no reason for supposing that the muscles in general use are in any way individually represented in the spinal centres. And whatever may be the encephalic machinery by which the several groups of muscles are harmonized and co-ordinated in their actions so as to produce definite changes in the position of a limb, it is certain that cross-connections exist within the cord by means of which a similar result can be brought about. We do not in man see such marked purposive reflex movements as are observed in decapitated frogs. The nearest approach to them that I remember to have read of was in a case of Dr. McDonnell's, recorded in the "*Dublin Quart. Journ.*" for 1871. The patient was paralyzed in all four limbs; when a catheter was passed into his bladder his left arm would "wriggle up and down, and the hand come fluttering over the genital organs, although he was absolutely unconscious of the operation, and even when his eyes were shut." Another curious case is related by Virchow at p. 684 of his "Gesammelte Abhandlungen." It is that of a man who was paraplegic, and who generally lay in bed with his lower limbs rigidly flexed. When he wanted to stand upright he would pinch the right thigh sharply, or give it a smart blow on its outer surface; this threw both the legs into a position of extension. Still the gastrocnemii remained contracted, and before he could put his foot to the ground he had to bring down the heels with both hands. As a rule, when reflex movements are excited by impressions upon the cutaneous surface they remain confined to the limb on the same side. Leyden says that they do not spread to the opposite limb except when the lesion is in the medulla oblongata, but many instances to the contrary may be found. The movements themselves generally consist in a series of jerking spasms rapidly following one another, in which all the joints are flexed, but sometimes the contraction is rather of a tonic character. A curious illustration of the way in which one set of reflex actions may be

made to counteract another set is afforded by a case of Dr. Budd's. The patient was recovering from paraplegia and could walk a few steps when the contact of the soles of his feet with the ground would make his knees bend beneath him; this he was able to overcome by rubbing the surface of his belly, the legs being then extended with a jerk.

A special kind of reflex movements has recently been described by Erb under the name of "tendon reflexes." It is a well-known schoolboy trick to give a smart tap with the side of the hand upon the tendon of the great extensor muscle of the leg, above or below the patella, while the knee is crossed over the opposite thigh, thereby causing the foot to be jerked upward. In 1875 Westphal and Erb independently published observations on this phenomenon; and Erb seems to have proved that it is of a reflex nature, the afferent nerves being those belonging to the tendon itself.* He has also shown that under morbid conditions, when the susceptibility of the lower spinal centres is increased, the same movement of the limb may be induced by a blow upon the periosteum of the tibia, below the ligamentum patellæ, and that similar contractions may be excited in many other muscles by irritation of their tendons respectively. The reflex actions set up in this way, and those which have their starting point in cutaneous impressions, are not necessarily affected in the same way by disease in the same patient. The former kind may fail altogether, while the latter kind are still to be obtained; or they may be greatly exalted, the others being, on the contrary, below the normal. No explanation of these differences can at present be given. According to Erb, a phenomenon described by Charcot and some other French writers is a particular form of tendon reflex. I refer to the violent tremor that can sometimes be produced in a paralyzed limb by grasping the foot with one's hand, and suddenly pressing it upward, so as to bring down the heel. Erb attributes this "ankle-clonus" to the irritation of the tendo-Achillis, which is forcibly stretched.

The condition of the reflex centres in the lower part of the cord bears a close relation to the state of nutrition of the paralyzed muscles, and to their electrical reactions, in cases of paraplegia. We have seen that where disease destroys the entire substance of the cord up to a certain level no reflex movements of any kind can be obtained in the limbs below. Under such circumstances the muscles become very rapidly deprived of their electrical contractility, and they undergo marked atrophy. Erb seems to think that before there is a complete loss of susceptibility to galvanic currents there is generally a period in which the "reaction of degeneration," described at p. 350, is present. On the other hand, when paraplegia is due to a lesion limited to one of the upper segments of the cord, so that reflex movements persist in the paralyzed limbs, the muscles, as a rule, retain perfectly normal electrical relations, and they remain well nourished for months and years, or at most show a slight flaccidity and wasting, which may fairly be attributed to disease. Indeed, they may actually become rigid, so that the legs, for example, are either forcibly extended, or drawn up in a state of flexion. But cases of this kind will be separately described hereafter, under the name of "spastic paraplegia."

Non-striated, as well as striated muscles can be excited to reflex contractions. Erb mentions a curious case of paraplegia, in which a fluid fecal evacuation was passed whenever a large bed sore was dressed. And in other instances micturition was directly produced by pressure over the bladder, or by the introduction of the finger into the rectum. It does not follow that the impulses were reflected upon the vesical muscular fibres after

* [There is, however, much to be said against the reflex character of this phenomenon. See papers by Tschirjew ("R. und R.'s Arch.," 1879), Gowers ("*Lancet*," i, p. 156, 1876, and Waller ("Brain," July, 1880). Knee-jerk, therefore, is a better name than tendon reflex.—Ed.]

being conveyed to the cord by cutaneous nerves, for the slightest compression of any part of the bladder appears to be sufficient to make its mucous membrane the recipient of stimuli from the urine contained in its interior. Indeed, some physiologists now believe that when micturition seems to be a volitional act, the influence of the will is really limited to relaxing the sphincter, and throwing the abdominal muscles into forcible contraction, so as to press upon the bladder and to set in action its reflex machinery. The experiments of Goltz upon young dogs ("*Pflüger's Archiv*," 1874) have shown that in these animals the bladder is capable of emptying itself at regular intervals, and in a perfectly normal manner, after the cord has been completely cut across by a sharp knife. Thus physiology would lead us to expect that micturition should take place naturally in those cases of paraplegia in which the lesion is limited to the upper part of the cord. In practice, however, it is found that retention of urine almost invariably occurs, even when reflex movements can be easily excited in the lower limbs, proving that the lumbar centres are intact. The explanation doubtless is that an inhibitory influence is transmitted downward upon those centres from the diseased parts above. Goltz, in fact, states that in some of his experiments it was not until several days after the operation of section of the cord that the animal was able to micturate properly; and he suggests that in cases of injury to the spine in man the bladder would after a time be found to have regained its functions, if catheterism were not continued as a matter of routine. However this may be, it is certain that the theoretical accuracy of Goltz's views with regard to micturition is established by some very exceptional cases of paraplegia, in which the urine is passed quite naturally. It is remarkable how many of the patients whose cases are related by Dr. Budd in his well known paper were able to micturate, though not always in a perfectly normal manner. In the case which I have already cited as presenting such violent convulsive movements of the legs, the urine at first began to flow in interrupted jets, but as the spasms grew more feeble its stream became continuous. Another patient, at a certain period in his illness, emptied his bladder involuntarily and with a sort of jerk, about every two hours. In a third case, one of fracture of the spine, in which there was at first retention, the power of voiding urine quite naturally was regained on the fifth day, whereas no volitional movements in the legs occurred until the ninth day.

So, again, one of the cases related by Ollivier in his classical work* is that of a man who had complete paralysis of his lower limbs as the result of caries of some of the higher dorsal vertebræ, but in whom the bladder performed its functions normally.

It is an interesting question whether the converse can occur, that is, whether the lumbar centre for micturition is ever the only part of the cord diseased, so that while the bladder is paralyzed the lower limbs nevertheless retain the power of movement. I have notes of more than one case in which retention of urine has preceded all other indications of the onset of paraplegia by an interval of a few days, but I do not remember any instance in which it has continued to be the sole or even the main symptom of a spinal affection. Some years ago I saw a gentleman who was one of the sufferers in the Thorpe railway collision, and whose chief complaint was that he could pass his water only in a very feeble stream; he said that "it flowed from him like oil," and it quickly become ammoniacal and very fetid. He had pain and tenderness in the back, but the only sign of any loss of power in the lower limbs was that in walking he sometimes seemed to totter, especially if his foot caught against any slight obstacle; he also had "twitch-

* ["Traité de la Moelle Epinière et de ses Maladies," par C. P. Ollivier. 1st Ed., 1821, 3d, 1837, with plates.—Ed.]

ings" in the legs three or four times a day. He was not long in getting perfectly well.

In some instances in which the power of micturition is partially impaired one can distinguish paralysis of the *sphincter* from that of the *detrusor*, the muscular coat of the bladder itself, by which expulsion of the urine is mainly effected. It might have been supposed that in the former case there would be complete incontinence, the fluid dribbling away incessantly. But physiologists have found that even after destruction of the lumbar cord the neck of the bladder can withstand a pressure of six inches of water (as compared with one of twenty inches when the cord is intact). And this result is entirely confirmed by some observations of Hutchinson ("*Brit. Med. Journ.*," 1877) upon persons who have the remains of a shrunken spina bifida, involving the fourth pair of sacral nerves, so that the sphincters of the bladder and rectum alone are permanently paralyzed. In such cases he finds that the bladder may be able to hold a large quantity of urine, but if once its reflex contractions are excited the will is, of course, powerless to restrain them, and therefore there is a constant risk of accidents. So in many cases of partial paraplegia the patient says that as soon as he feels the inclination to micturate he is obliged to indulge it, so that the urine is apt to escape before he can get to the chamber vessel. But when the *detrusor* is paralyzed, the complaint is that he has to strain for a minute or two before he can pass any water, that it comes away slowly in a feeble stream, and trickles for some little time after voluntary effort has ceased.

On the other hand, when the "micturition centre" in the lower part of the cord is wholly deprived of its functions by disease or injury, the sphincter and the detrusor of the bladder are, of course, paralyzed together. The result is that the organ becomes distended, and that the excess of urine beyond what it can hold dribbles away through the urethra. The Germans call this condition "ischuria paradoxa." It has often been mistaken for incontinence; an unskilled nurse is very apt to suppose that there can be no retention of urine in the case of a patient who is constantly wet, and her statements may mislead a careless practitioner; but one can always avoid such an error by passing one's hand over the lower part of the abdomen. In real incontinence of urine the bladder keeps firmly contracted, and almost perfectly empty. I believe that its walls invariably become greatly hypertrophied. It seems to be still doubtful whether this symptom always depends upon an exaltation of the reflex activity of an intact lumbar centre, or whether it may occur after that part of the cord has been destroyed, as the result of contractions of the detrusor, which one could either regard as spontaneous or suppose to be dependent upon minute ganglia connected with the vesical nerves.*

Some other points connected with the pelvic organs may be conveniently mentioned here. One of them has reference to the *rectum*. It is well known that the peristaltic movements of the intestine are not directly dependent on the spinal cord. But the sphincter ani is under the control of a lumbar centre. Consequently, as a rule, it becomes paralyzed with the bladder; and incontinence of the fæces is generally associated with retention of urine. Another point concerns the male genitalia. *Priapism*—a more or less complete erection of the penis—is of frequent occurrence in paraplegia. It is one of the results of the reflex activity of the lower part of the cord. Sometimes it seems to be simply dependent upon distention of the bladder; sometimes it is directly excited by the operation of catheterism. Goltz, in his experiments on dogs, traced out carefully the various ways in which it could be induced by irritating the skin of

* [See a paper on these intra-vesical ganglia by Dr. Francis Darwin, "Quart. J. Micr. Sc.," 1874, p. 109.—Ed.]

the abdomen, or of the thighs, or the surface of the penis itself. He also found that he could easily inhibit this reflex action by simultaneously irritating the sciatic nerve, or even by pinching the hind foot. The last observation—showing that the centre for erection is very sensitive to disturbing influences—may serve to explain the fact that a temporary loss of sexual power is not infrequently observed in persons who have been in a railway accident, but in whom there are no other symptoms of serious spinal injury. When the lower part of the cord is destroyed, priapism is, of course, altogether absent. Lastly, as regards the female organs of generation, the only fact worthy of mention seems to be that *parturition* may take place naturally in a woman suffering from paraplegia, provided that the lumbar centres remain intact.

Returning to the subject of micturition, we have still to consider certain changes (in the urine itself, and in the organs that secrete and expel it) which are of the highest clinical importance. Sir Benjamin Brodie, many years ago, pointed out (in the "*Med.-Chir. Trans.*" for 1836) that in cases of injury to the spine the urine is often voided alkaline, ammoniacal, and turbid, even as early as the second or third day; it is now well known that in most cases of disease of the cord, attended with paraplegia, the same thing occurs, but generally not until a longer time has elapsed. Its explanation is still a matter of doubt. Charcot believes that it is in some way due to a direct action of the nervous centres upon the urinary organs. Most of the other writers who have recently expressed opinions upon the subject think that it is simply a result of decomposition of the urine, after secretion, while it is stagnant in the cavity of the bladder. Such a view must not be taken to mean that only those patients who have complete retention pass urine which is alkaline and fetid, for there can be no doubt that a very slight impairment of the power of the bladder may prevent it from emptying itself completely. But the difficulty, at the present day, is to understand how the change in the urine is brought about, apart from the access of air. Traube, of Berlin, was, I believe, the first to suggest that a badly-cleansed catheter might set up decomposition in it; and the importance of employing antiseptic precautions before one introduces any instrument into the bladder cannot be overstated. But I have seen more than one case in which catheterism was never necessary, but in which the urine, nevertheless, became putrid. I do not know whether the difficulty is met by a suggestion made by Dr. Goodhart, in the "*Guy's Hosp. Rep.* for 1874, that an almost stagnant column of urine filling the urethra would suffice to enable organisms, without which it is believed that putrefaction does not occur, to pass into the bladder.

Moreover, although we may admit that decomposed urine is capable of exciting inflammation of the bladder and of the urinary passages, it does not appear that there is always such an interval of time as would be required for the production of this effect. Charcot cites cases in which the urine contained blood as early as the third and the fourth day respectively. And the presence of blood is generally followed in a very few days by that of pus, or of the stringy mucoid substance which is formed out of pus in a strongly alkaline liquid.

On post-mortem examination, in cases of this kind, one finds the most intense cystitis and nephritis; and, indeed, these affections have most commonly been the direct cause of death. The mucous membrane of the bladder is ecchymosed or swollen, infiltrated with inflammatory products, lined with a diphtheritic layer or with a granular deposit of phosphates, or actually sloughing. Its muscular wall is often free from hypertrophy, but sometimes I have found it greatly thickened; once it measured at least one-twelfth of an inch across, although the disease had lasted only four or five

weeks. The kidneys are enlarged, intensely congested, and full of suppurating points and streaks; in other words, they present all the characters of an "ascending inflammation."

A circumstance which points strongly toward the conclusion advocated by Charcot—that morbid changes in the urinary organs are at least sometimes directly dependent upon an influence transmitted to them from the nervous centres—is that their development is commonly coincident with that of a gangrenous condition of the skin over the upper part of the sacrum and the gluteal regions adjacent. An "acute bed sore," in fact, sometimes begins to form as early as the fourth day. Many instances of this kind are quoted by Charcot; but, perhaps, there is not one more striking than a case recorded by Sir William Gull, of a man who, on November 22d, 1856, was working in the docks, when he felt a sudden pain in the back after lifting some deals; he became paraplegic on the 24th, and on the 26th was admitted into the hospital with a small bed sore already present. It is clear that in such cases the affection cannot be regarded as a mere result of pressure upon the skin, nor of the irritant action of excreta passed without the patient's knowledge; but there seems to be no doubt that each of these conditions is concerned in the production of the more chronic bed sores which do not appear until after the lapse of some months. An acute bed sore may sometimes be made to heal, if carefully attended to; this occurred in the case which I have just quoted; but, afterward, another one formed at the same spot, and it rapidly increased in size, so that, when the disease ended fatally, six weeks from its commencement, the whole of the sacrum was exposed. Indeed, this affection may directly destroy life, either by exhaustion, or by pyæmia, or by extension of putrid inflammation into the spinal canal, when the dura mater has sloughed.

The morbid conditions that give rise to paraplegia fall naturally into two groups. The first includes all those affections which have their seat *within the cord itself*. The second is made up of those in which the primary lesion is *outside the cord*, this being subjected to a *slow compression*, annihilating its functions. The division is not, indeed, altogether exhaustive; but it is one of great practical importance, because in the second group there is a very characteristic chain of symptoms, dependent upon the interference with nerve roots at the level of the lesion.

Myelitis.—Among the affections beginning within the spinal cord itself and capable of causing paraplegia, the most important, and by far the most frequent, is one which is now believed by all pathologists to be a more or less diffused inflammation of its substance, and which they therefore term *myelitis*. This, again, presents many varieties in its symptoms and course, and in the morbid appearances to which it gives rise; but in the main they may be arranged into two classes, according as their progress is *acute* or *chronic*.

Acute Myelitis.—It has long been known that in many cases of paraplegia the cord is found after death to be more or less extensively softened; and from the time of Ollivier, the opinion has been maintained by many pathologists that the morbid process in question is of an inflammatory nature. There are, indeed, obvious objections to such a view. It involves the admission that a remarkable contrast exists between softening of the cord and a similar change in the brain. The latter (as we shall hereafter find) is most frequently the result of a deficient supply of arterial blood to the affected part of the cerebral substance; and, when it is inflammatory, it is almost always traceable to some definite local irritation. The former, on the other hand, is very seldom secondary to any interruption of the blood

supply, and its chief causes are of a general kind, such as functional exhaustion, exposure to cold, etc. A more serious difficulty is that the histological appearances correspond only partially with those which are seen in inflamed tissues generally, and that even the naked-eye changes are not altogether such as are met with in the spinal cords of animals, when inflammation has been artificially set up in them. Leyden, for example, induced myelitis in dogs by injecting Fowler's solution of arsenic, and obtained greenish patches of purulent infiltration, surrounded by vascular zones. Now, in human pathology, the presence of anything that can be recognized as pus in the spinal cord is in the highest degree exceptional. I find a reference to a case of Jaccoud's as one of the chief instances of it that have been recorded ("Les Paraplégies," etc., p. 545); but this writer, although he speaks of a "*myelite suppurée*," does not actually say that the material which took the place of the affected part of the cord was proved to be true pus.

The color of the spinal cord when softened is very variable. Sometimes its tissue is pink or bright red, from vascular injection; sometimes it is mottled with small ecchymoses; it may then be so saturated with extravasated blood that doubts arise as to whether the case is not simply one of hemorrhage, an affection, however, of which the occurrence is denied by many modern pathologists, as we shall presently see. Probably all these appearances are confined to an early stage of the inflammatory process. Later on the blood undergoes changes which give to the diseased parts a tawny yellow or brownish tint, or, if no hemorrhage has occurred, it may be perfectly white. Whether white softening is ever observed when death has occurred at a very early stage I am not sure. I have not found any instance of it among those cases which have proved rapidly fatal at Guy's Hospital.

But the fact that one part of the spinal cord is softer than the rest, or that it looks diffluent when cut across, is not of itself a proof that disease is present. It may result from bruising with the bone forceps during the autopsy though made by the hands of a practiced assistant. The microscope here affords valuable assistance. In the great majority of cases one has only to spread out a little of the softened tissue between slide and cover in order to find with a quarter objective numbers of opaque, mulberry-like aggregations of fatty granules, the well-known "compound granule masses" or "corpuscles of Gluge." The exact pathological nature of these bodies is, indeed, still very uncertain; but there is scarcely any doubt as to their practical significance, as indicating that the part of the cord in which they are detected is in a morbid condition. Leyden, indeed, quotes statements made by different observers that they are normally present in the fœtus, and that they may also be found in persons at an advanced age, or after death from tuberculosis, pneumonia, or anæmia, when no spinal symptoms had existed during life. But at any rate one need not hesitate to regard them as a proof of disease when they are thickly scattered through some parts of a cord and absent elsewhere. The difficult question of their origin may be left to be discussed hereafter, when I shall be describing softening of the brain. The most remarkable points about them are that they are observed under such diverse pathological conditions, and are fully developed at so early a period. Thus, there were large numbers of them in the spinal cord of a patient who was under the care of Dr. Frederick Taylor in Guy's Hospital in 1873, and who died within eleven days from the commencement of his symptoms, and not more than fourteen days after the occurrence of a slight injury that perhaps set up the disease. In such cases they are not generally accompanied by an obvious infiltration of leucocytes, or by the presence of any diffused granular matter, but lie embedded among nervous

elements which to a superficial observer seem to have a normal appearance.

Recent investigations, however, have shown that the histological changes are really far more considerable than would at first sight appear, and it is to be noted that granule masses are not infrequently discovered when there is no obvious softening, the texture being, of the two, rather firmer than is natural; and, again, when there is no marked change of color, or, at most, only a pinkish-gray tint, with some blurring of the pattern on a cut surface. Nor is even the failure to detect granule masses a proof that morbid changes may not be recognized where the cord has been specially prepared for the purpose. In many cases after it has been soaked in a diluted solution of chromic acid for two or three weeks one can see at a glance what parts of it are diseased, since they remain colorless instead of turning green. And a microscopical examination of thin sections shows that the nerve fibres have their axis cylinders swollen and varicose, that the ganglion cells are enlarged and granular (or perhaps vacuolated), and (according to Erb) that the network of the neuroglia is thickened, while its cells are increased in size and augmented in numbers, and sometimes contain more than one nucleus. Erb also says that a colloid substance is sometimes found diffused along the course of the swollen connective-tissue bundles, and Leyden that a scanty infiltration of leucocytes is now and then seen in the interstices between the nerve fibres. Such appearances are doubtless conclusive as to the inflammatory nature of the process, and so is the presence of recently effused lymph in the tissue of the pia mater or in the subarachnoid space. This I have myself found in sufficient quantity to be plainly visible to the naked eye, and it probably might be discovered with the aid of the microscope in many cases in which it has hitherto been overlooked.

Chronic Myelitis. Local Sclerosis of the Cord.—The disease which I have just been describing does not in all cases run such a course as might seem at first sight to warrant our speaking of it as an *acute* inflammation. Sometimes, indeed, it sets in with fever and ends fatally in a few days, but in many instances it is unattended with febrile disturbances, and goes on for many months before it terminates, either in the death of the patient or in his more or less complete recovery. What then justifies its name is the fact that its onset is more or less sudden and its development rapid, the paralysis being complete within a week or two.

In marked contrast with that affection—even in the most protracted cases—is one which always begins insidiously and gradually, of which the duration is never less than two or three years, and which, instead of leading to a softening of the cord, rather renders it firmer and harder than before. The naked-eye appearances in this form of myelitis are variable. Sometimes it is altogether impossible to distinguish those parts of the cord that are diseased from those that are healthy; in such instances no morbid change was recognized until the accurate microscopical investigations of the last few years revealed its presence. In other cases the substance of the cord is obviously tougher than natural. Ollivier long ago very aptly compared its appearance to that of the boiled white of egg; the knife meets with resistance in cutting through it, and the exposed surface is smooth and even. On close scrutiny it often looks gelatinous and has a grayish or grayish-yellow tint. It is rather shrunken than increased in size.

With the aid of the microscope the neuroglia is found to be thickened and to have a fibrillated texture, or even, in extreme cases, to be converted into a dense mass of connective tissue with delicate, parallel, wavy fibres. Its cells and nuclei are increased in numbers, and are far more conspicuous than in the healthy spinal cord. Cells possessing a large number of radiating processes (Deiter's cells) are often seen; it is said that similar

cells may be discovered in the normal neuroglia, but they are, at any rate, much more obvious in cases of chronic myelitis. The nerve fibres are generally atrophied; they have lost their medullary sheaths, and their axis cylinders are described as having a hard, glistening look. The ganglion cells are shriveled and granular, or may even be converted into homogeneous, bright-looking angular bodies without processes, and having apparently no nuclei, so that they can hardly be identified. At the same time granule masses are present, often in large numbers, but sometimes very few of them. Corpora amylacea are generally abundant. The smaller blood vessels have their walls thickened.

This morbid process—the account of which is taken chiefly from the work of Erb—is now generally known as "sclerosis." It is not, however, peculiar to cases of chronic myelitis, such as are attended with paraplegia, but lies at the foundation of several other affections of the spinal cord, which will be described separately, because their limitation to special parts of its substance gives them an independent place in nosology. In the cases now under consideration, the lesion is so far diffused over the whole segmental area of the cord, that it interferes with or altogether prevents the transmission of motor impulses from the brain to the parts below. Its extent may, of course, vary greatly. Sometimes it is confined to a single spot, and it is often spoken of as "simple transverse myelitis;" sometimes it creeps slowly along the entire length of the cord (generally from below upward) until it may even reach the bulb.

Ætiology.—According to Erb, inflammation of the cord is more frequent between the ages of ten and thirty years than in older persons. But I find that a series of twenty-five fatal cases of primary diffused myelitis at Guy's Hospital were pretty uniformly distributed over the several decennial periods from ten to fifty, and that three of them occurred in patients between fifty and sixty years old. Most cases occurred in men, the proportion being as 19 to 6. This, however, depends, not on the existence of any predisposition to the disease in men, but on the fact that they are much more exposed than women to its various exciting causes. For, although it certainly is not always due to any one morbid influence, its direct ætiology can yet be traced in many cases with remarkable accuracy. Sometimes it is clearly the result of exposure to cold. Several instances of this have been related by writers on spinal affections, and our records at Guy's contain others scarcely less striking. Thus, in 1876, a custom-house officer was admitted into the hospital with nearly complete paraplegia, who had been well until eight days previously, when he was obliged to remain for twenty-four hours in a barge, where everything was soaking wet. On the very next day he was attacked with pains in the joints, which he believed to be rheumatic; his urine began to run away from him; and two days later his feet and hands and back already felt numb. Another patient, who was brought in on January 29th, 1875, had, on the 18th of that month, got wet and caught cold; three days afterward he passed his water into his bed without knowing it, and his legs became numb and powerless. Among the conditions which are mentioned as especially apt to set up myelitis are sleeping on the damp ground, or in snow, and prolonged immersion in water, as when a person narrowly escapes drowning. Bodily fatigue also seems often to play an important part in the ætiology of the disease. When it occurs in soldiers on active service, as is said to be frequently the case, it may either be due to exhaustion of the spinal cord in long marches, or to exposure to cold and wet, or to both causes combined. A case in point has been recorded by Sir William Gull ("*Guy's Hosp. Rep.*," 1856). A young man, after walking twenty-eight miles to seek for work, passed the night of July 8th,

1855, in a brick field. On the 15th he walked thirty-two miles; the day was wet, and he allowed his clothes to dry upon him. Next day he felt as well as usual, but in the afternoon, while he was sauntering in a garden, his legs suddenly gave way under him, and he fell down. He was admitted, six days later, completely paraplegic. He was healthy and florid looking; but it should be stated that he had, about a fortnight previously, been unable to pass his water for a period of twelve hours. He died in less than a month.

A great muscular effort, such as often causes a pain in the back, is now and then followed, after an interval of a day or two, by paraplegia. An instance of this has already been cited (at p. 396) in the case of the dock laborer. Another example of it occurred in Guy's Hospital, in 1863, in the person of a railway porter, aged thirty-two, who was pushing a carriage with his back, when he suddenly gave a jump to escape falling into a pit, and so ricked his spine. For a moment he felt powerless, but he afterward resumed his work, and worked as usual on the next day. The day after that, however, he was walking in the street, when he suddenly became paralyzed and fell down. Each of these cases terminated fatally in about six weeks; at the autopsy no injury to the bones or the ligaments could be discovered; but in the dock laborer there was softening of the cord in the dorsal region, the affected part having a greenish or brownish color, no doubt from admixture of a small quantity of extravasated blood. The microscopical examination revealed no trace of inflammatory exudation; but I do not doubt that, if modern methods of investigation could have been used, we should have been more successful. I shall, however, have to revert to this question further on—when I shall have to discuss in some detail the question of the causation of myelitis by mechanical injury.

Violent mental emotions—of terror, grief, or anger—are mentioned as possible causes of myelitis by some writers. Thus, Leyden met with a case of paraplegia, of which the symptoms and course corresponded exactly with those of myelitis, and which appeared to be due to fright from the breaking out of fire.

Again, it occasionally happens that paraplegia develops itself in the course of an acute disease, especially during convalescence; and in cases of this kind myelitis has been discovered on post-mortem examination. I have already referred to such observations in discussing the diphtheritic paralyses. Virchow has recorded ("Ges. Abhandl.," p. 683) an instance in which chronic myelo-meningitis came on a few months after recovery from enteric fever, and seems to have thought that this occurrence was more than a coincidence. Westphal examined the bodies of two patients, each of whom became paraplegic during the eruptive stage of smallpox (variolois), and in both cases he found disseminated myelitis. Either pregnancy or the puerperal state, too, may be attended with paralysis of the lower limbs; and it would seem that the cord has been found softened. But the most remarkable case of all—if it was correctly interpreted—is one which Baumgarten supposed to be due to infection with the poison of the epizoötic splenic fever (Milzbrand); it ended very quickly in death, and he found the characteristic bacilli in the blood as well as in the spinal cord ("*Arch. f. Heilk.*," 1876).

Syphilis is said by Erb to be an undoubted and important cause of myelitis. He states that the affection may then run either a very rapid or a chronic course. Of cases of the latter kind he saw at least a dozen in the course of a few years, and some of them came to an autopsy. This question, however, is one which I shall have to discuss further on.

Lastly, there is reason to believe that sexual excesses play a considerable part in the ætiology of diffused inflammation of the spinal cord, although it is very difficult to say exactly how far this influence extends. Men who

have been indulging their passions too freely, especially if they are weakly or very young, often experience pains in the back and limbs which seem to be due to exhaustion of the lower spinal centres, and not merely to fatigue of the muscles. And it is probable that these symptoms may after a time pass into those of actual myelitis. Whether coitus in the erect posture is especially injurious I do not know, nor whether onanism is so, except in so far as the stimulation is apt to be repeated very frequently in the absence of instinctive desire, and at an early period of life. On these points no evidence is to be obtained, nor can one desire to collect it, since the possible physical effects of such practices are far outweighed by their influence upon morals.

In passing on to discuss the *symptoms* and *course* of myelitis I must remind the reader that on grounds of practical convenience I have set aside for separate description certain groups of cases which are included under that designation by the morbid anatomist, namely, those of softening due to direct extension from some affection of the parts outside the cord ("compression myelitis"), and also those of sclerosis limited to certain definite tracts within the cord or disseminated in a number of isolated patches throughout its substance. I am at present confining myself to the *primary* form of acute myelitis, and to the more or less *diffused* forms of chronic myelitis. Now, in such cases the principal symptom is paralysis of parts below the seat of lesion. Indeed, the whole account of paraplegia, from p. 387 to p. 396, may be taken as applicable to the disease now under consideration. But it is important to note, that not only may the slightest possible general impairment of muscular power in the legs be due to myelitis, but even, in all probability, a partial loss of power in a part of a limb, or even in a particular set of muscles.

But it is important to note that besides causing paralysis, impairment of sensation, dysæsthesiæ, disorder of the reflex functions of the cord, and the like, myelitis is often attended with another set of symptoms which I have hitherto avoided mentioning, namely, with *painful sensations* referred to various parts of the body. In some instances the seat of pain is in the back; it there varies greatly in intensity; it may either be confined to one or two spinous processes, or diffused along the whole length of the cord; there may be extreme tenderness on pressure, or this may be altogether absent. Occasionally spinal pain may be the earliest symptom of the disease, but the notes of cases which I have before me seem to show that this is rare. In other instances the pain is referred to the front of the chest, or to the epigastrium, or to one or more of the limbs. One patient, a girl of nineteen, was attending as an out-patient for pain in the chest, when she one day fell down in the waiting room, with paraplegia. Another, a man aged forty-nine, had complained of a fixed pain in the left hip for eight weeks before any definite sign of spinal mischief showed itself. A third patient, a man aged fifty-one, suffered at first from severe burning pains in the soles of the feet, which continued night and day for a considerable time. These cases all terminated fatally, and in each of them the bones and ligaments and membranes were found to be healthy, the disease being limited to the substance of the cord itself, so far as could be ascertained by ordinary methods of examination. Sometimes, when pain is referred to the fore part of the body, it is accompanied by a sense of constriction, as if a cord were tightly bound round the waist, or as if the chest were fixed in a vise. We shall hereafter see that this symptom, to which German writers give the special name of *Gürtelgefühl* (girdle feeling), is particularly marked in that other form of paraplegia due to compression of the cord by disease of the surrounding structures. But I believe that there can be no question as to its occurring now and then in cases of

primary myelitis. On the other hand, I would strongly insist on the fact that a large majority of cases of this kind are altogether unattended with pain, whether in the back or elsewhere.

The *course* of myelitis differs very widely, not only according to the nature and seat of the morbid change in the cord, but also in individual cases which appear to resemble one another closely. Erb speaks of the disease as sometimes setting in with malaise and fever, and even with rigors. A case in point occurred at Guy's Hospital in 1872. A man, aged fifty-one, who was much exposed to weather, one day began to shiver, and was attacked with severe pains running down the back of the thighs and calves and the insides of the arms. He remained in bed for four days, after which the pains left him, and he went to work again. Next day he lost all power in his limbs, and he died of dyspnœa about eight days afterward. The paralysis often shows itself quite suddenly in that form of myelitis which leads to "softening" of the cord. Indeed, as Erb points out, the rapid development of this symptom affords the chief justification for our calling cases of this kind "acute;" they are, in fact, often unattended with febrile symptoms, and their duration may be prolonged over a period of many months.

Prognosis.—Until within the last few years there were no grounds upon which writers could speak with confidence of the possibility of complete recovery from myelitis. It was well known that many patients suffering from paraplegia got perfectly well, but whether inflammation of the cord existed in such cases was uncertain. The discovery of the remarkable power of regeneration possessed by nerve fibres (see p. 352) did much to clear up the doubt, and Eichhorst and Naunyn found in experiments upon young dogs, in which they cut through the spinal cord in the lower dorsal region, that the affected part, although it became completely liquefied, yet ultimately underwent a partial repair, nerve fibres with double contour being developed in small numbers in the new material which filled up the gap, while motor power and sensation were to some extent regained. These results have not been confirmed by the more recent investigations of Goltz and Freusberg; nor in any case could one lay much stress upon them, so far as human pathology is concerned, since it is certain that in man the cord is very far from possessing such powers of restoration. But, on the other hand, I shall presently have to cite an observation made by Michaud, which shows that in spite of the occurrence of well-marked myelitis, recovery from compression paraplegia may take place, the affected part of the cord continuing to display obvious morbid appearances. Surely this case may be taken as proving the possibility of the complete subsidence, with return of normal function, of those local and slight forms of myelitis which may fairly be supposed to be present in partial and transitory forms of spinal paralysis. Such a conclusion is further supported by the fact that the recovery is often incomplete in cases of this kind, since it is more reasonable to suppose that an organic affection of the cord should in some part subside than that a merely functional disorder should in any part be permanent.

However this may be, it is certain that, as a rule, the prognosis must be unfavorable in cases of complete paraplegia with paralysis of the sphincters which depend on a primary myelitis. But it is another matter if the paralysis is incomplete or limited to certain muscles.

The *diagnosis* of myelitis as a cause of partial or complete paraplegia is generally effected by a process of exclusion. We have first to determine that the original seat of the disease is in the cord itself, and that the case is not one of compression of the cord, nor of meningitis. I cannot, however, discuss the grounds upon which these inferences must be based until I shall have described the diseases in question. Secondly, we have to decide

what affection of the substance of the cord is most likely to be the cause of the paralysis. Now, myelitis is at once the most frequent of these affections and by far the most varied of them all in its symptoms and course. But I have already virtually admitted that doubts still prevail as to which cases should, and which should not, be set down to it, at least so far as concerns the less fatal forms of paraplegia. I therefore think that the only convenient way of dealing with the subject of diagnosis will be for me here to interrupt my account of myelitis, and to introduce, as briefly as may be, the necessary descriptions of the other morbid conditions, beginning in the cord itself, with which it may be confounded. These I shall take in an ascending series, beginning with some which are comparatively of but little gravity, and afterward passing on to others which are as dangerous to life as myelitis itself.

Neurasthenia Spinalis.—"*Spinal Weakness.*"—Among the patients who seek medical advice, believing that they are suffering from spinal disease, there are some—chiefly youths and men not far advanced in adult life—whose principal complaint is of muscular fatigue and weakness. If they attempt to walk far they experience aching pains or stiff sensations in their limbs. When they have to stand for more than a short time their legs tremble, and seem to give way under them. They feel tired and weary all day long, even before they get out of bed in the morning. On account of the resemblance which such cases undoubtedly bear to some others in which there is early mischief in the cord, it is desirable that a special name should be given to them; and that of "Spinal Neurasthenia," originally proposed by Drs. Beard and Rockwell in 1871, appears to be very suitable. It has recently been adopted by Erb, from whose account of the complaint mine is taken, and who in 1876 had already collected notes of more than two dozen cases. Most of them occurred in persons belonging to the middle or upper classes and to families with marked tendencies to suffer from nervous disorders. The exciting causes appeared generally to be either over-exertion of body or mind (especially if carried on during the hours that ought to have been devoted to sleep) or the various forms of sexual excess. Among the symptoms were an "irritable weakness" of the generative organs, sometimes a little dribbling of the urine after micturition, and very rarely a slight sensation of numbness or formication in the feet. In some cases dragging or tearing pains in the limbs were complained of; but generally these were not very severe. Very often there was a peculiar pain in the back, seated apparently in the muscles, increased by movements of the spine or of the scapulæ. Sometimes local tenderness of certain spinous processes was present, exactly as in the affection which will hereafter be described as "rachialgia" or "*spinal irritation;*" it may then be said that the two neuroses were combined in the same patient. Indeed, it often happened that there was also functional disturbance of the brain, indicated by sleeplessness, timidity, hypochondriacal depression of spirits, etc.

It is supposed by Erb that the state of the nervous elements in the spinal centres in such cases is, in fact, the same as in the healthy cord when exhausted by the discharge of its functions. The difference is that a natural period of rest fails to restore to them their vigor and activity. This view appears to me to be very reasonable. The diagnosis from myelitis and from other serious spinal diseases must be based mainly upon the absence of all objective symptoms of a definite lesion of the cord, in contrast with the vehemence with which the patient complains of his subjective sensations. Erb says that spinal neurasthenia sometimes sets in rather suddenly, developing itself fully in the course of a few days. It may reach such an intensity as to compel the sufferer to give up his occupation, and even to renounce all society. It

is often obstinate, lasting for many months, or even for years. Relapses of it sometimes occur. Whether it ever is incurable, or passes on into any organic affection, is as yet doubtful.

Anæmia of the Spinal Cord.—Unless the modern interpretation of a very old observation is incorrect, one of the possible causes of paraplegia is an arrest of the arterial blood supply to the lower part of the cord. Nicolaus Stenon, in 1667, described, as a result of tying the abdominal aorta in animals, that the lower limbs became powerless, and remained so until the ligature was removed. His experiment was long afterward repeated by Sir Astley Cooper and by others; but until 1869 the explanation universally accepted was that the muscles were paralyzed because they and the peripheral nerves were deprived of blood. Schiffer, however, has recently shown that if this were the case an interval would elapse, instead of the effect being observed a few seconds after the operation. Whether a similar form of paralysis is ever seen in man as the result of disease appears to be somewhat doubtful. The most frequent cause of obstruction of the abdominal aorta is embolism; but in that affection, as might be expected, the clot becomes impacted close to the bifurcation of the vessel—too low to interfere with the circulation in the lumbar enlargement of the cord.

In the "*Guy's Hospital Reports*" for 1857, Sir William Gull gave the clinical history of a patient whose aorta was from some cause or other obstructed, and who was attacked with paralysis which, as he thought, was peripheral, rather than spinal, in its origin. The man was a shipwright; and in the beginning of March, 1855, while apparently in perfect health, he was suddenly seized, being in a stooping position, with pain in the loins. This went off after he had rested for a few minutes; but on his resuming his work it returned and extended down the legs, with a sense of numbness, soon followed by entire paralysis, both of sensation and motion, from the loins downward. The sphincters were involved. After a few days sensation returned, and he was able to take a few steps unsupported. He gradually improved, but the legs remained unsteady. The paraplegic symptoms led to his coming under Gull's care in June of the same year. It was found that there was no pulsation in the abdominal aorta, nor in the arteries of the lower limbs. The right superior epigastric artery was already distinctly enlarged, and could be seen pulsating. In the course of the two following years he regained power to walk tolerably well, but at the end of that term his muscles were still thin and his gait was languid, and from time to time he had slight returns of weakness and numbness in the legs. His feet also were cold and damp. Leyden has suggested in reference to this case that it is improbable that a sudden obstruction of the abdominal aorta should have occurred without more serious symptoms developing themselves, and that a collateral enlargement of the epigastric artery would hardly have been seen at so early a period afterward. And it must be admitted that the case resembled one of "coarctation of the aortic arch" in many respects; and, among others, in the presence of a systolic murmur at the lower angle of the left scapula. But, on the other hand, the arterial anastomoses over the back and the abdomen underwent gradual enlargement while he was under observation, apparently to a very marked extent. And even if the obstruction had existed for a length of time before the paralytic symptoms appeared, it still may have caused them; a supply of blood adequate to the ordinary requirements of the lower limbs (or, on the other view, of the spinal cord) may have been insufficient for some specially prolonged effort, or when the man was exhausted by overwork, or by some other cause. One remark made by Dr. Gull is interesting, namely, that "using the legs soon brought on increased weakness and

numbness." For an intermittent paraplegia has been noticed in horses, as a result of obstruction of the aorta; a loss of power in one or both of the hinder limbs, coming on when they are driven, and subsiding when they stand still. Charcot is said to have observed a similar condition in a patient whose right common iliac artery was obstructed. When this man was walking, he was obliged to stop every fifteen or twenty minutes, on account of paralysis of the corresponding limb; after a few minutes' rest the symptoms passed off and he was able to go on again.

Leyden appears to have found capillary embolisms in the cord in ulcerative endocarditis, but I am not aware that any impairment of its functions have been traced to them; probably the arteries which enter it are not large enough for obstruction of any one of them to produce clinical effects.

There is some evidence that a defective blood supply to the spinal cord may lead to the occurrence of paraplegia in persons who have suffered severely from hemorrhage, or who are highly anæmic. Among the cases of this kind quoted by Leyden there is one of a woman, aged twenty-four, who had lost blood profusely after a confinement; she was exceedingly weak, but she was resuming her domestic duties when, at the end of about a month, she suddenly became unable to stand. Another case, published by Dr. Montard Martin, occurred in a man as the result of severe hemorrhage in the course of dysentery. Others were in persons who had suffered from menorrhagia, but it must have been difficult, if not impossible, to exclude hysteria, which, as we shall presently see, may itself cause paraplegia. The same source of fallacy exists in reference to those cases which are recorded as examples of paraplegia due to chlorosis in women. I am not aware that such an affection has hitherto been observed in the intense and fatal form of idiopathic anæmia to which men are liable.

But, after all, the mere fact that persons who have lost blood are now and then attacked with this form of paralysis is far from showing that anæmia of the spinal cord is capable of directly producing the effect in question. For the proof to be complete there must be no interval of time during which impairment of the nutrition of the cord could have occurred as an intermediate link in the chain of events. In any future case it would be important to note whether the patient, after the hemorrhage, took violent exercise or worked hard in any way, using his legs more than his arms, so as to cause the lower spinal centres to make special claims upon the nutrient powers of the blood. We might thus be able to see why the lower limbs should alone be paralyzed, and not all parts of the body alike.

However this may be, I do not find the smallest reason to suppose that paraplegia ever depends upon a defective supply of blood to the whole cord, or to any part of it, in persons who have a good color, whose extremities are warm, and who, to all appearances, have an active circulation in the organs generally. Some years ago, however, Brown-Séquard professed to be able to distinguish a paraplegia due to anæmia from one due to hyperæmia, by the simple circumstance that, whereas the former was benefited by keeping the patient recumbent on the back, the latter underwent aggravation when this posture was adopted, but was lessened if he lay on the abdomen, or stood upright, or walked. This method of diagnosis has recently been endorsed by Dr. Hammond, of New York, who mentions a case which he considered to be one of congestion, and in which "all the symptoms were worse in the morning." However, he does not give any details which might show in what manner he was able to exclude other possible causes of an aggravation at this period of the day, such as stiffening of the muscles when disused for some hours. And it seems to me that the value of his opinion is greatly diminished by the readiness with which he adopts this kind of hypothetical pathology, assuming, as he does,

the existence of two altogether distinct forms of spinal anæmia, the one limited to the antero-lateral, the other to the posterior columns. The last affection is, indeed, one with which we are all well acquainted, under the name of "spinal irritation" (or rachialgia), which I shall describe in the next chapter. Nor could one have a more striking proof that such speculations are utterly baseless than the fact that Stilling and Ollivier had each referred this disease to the very opposite condition—congestion of the cord—for reasons which are neither better nor worse than those of Dr. Hammond.

Spinal Hyperæmia.—Passing now to consider whether paraplegia may depend upon congestion of the cord, I will frankly state my own conviction that we are never justified in making such a diagnosis. All pathologists are agreed that distention of the blood vessels within the spinal canal, if found in the dead body, affords no proof that they were overfull during life: it may be due either to the mode of death or, if the corpse lies face upward, to post-mortem gravitation. But there are sometimes good reasons for believing in the occurrence of morbid processes, in spite of the fact that they cannot be recognized by the anatomist. What chiefly leads me to reject the doctrine of a persistent congestion, independently of any mechanical cause for it, is my opinion as to the origin of that doctrine. In the early days of pathology, writers proceeded to assign to all diseases their anatomical lesions in an ascending order of severity. They found that almost every organ was liable to certain chronic changes which could be regarded as inflammatory, and any cases which seemed to stand lower in the scale were handed over either to hyperæmia or to anæmia, according as the one condition or the other seemed the more easily to be brought into harmony with the various symptoms.

Even now a similar method prevails. In 1866, Dr. Radcliffe (Reynolds' "System," ii, p. 619) had under his care a woman who had been found paralyzed in all her limbs the morning after having had her menstrual flow checked by an alarm of fire. Among the other symptoms were tingling in the fingers and toes, some degree of general hyperæsthesia, and a dull, burning, aching in the limbs and along the spine, without special tenderness on pressure over the vertebræ, but with increased sensitiveness to a hot sponge. The bladder and rectum performed their functions naturally. There was no over-excitability of the reflex centres in the lower part of the cord. Within two or three weeks she began to improve, and at the end of five months she left the hospital convalescent. Considering the circumstances under which her attack commenced, we can see that the hypothesis of spinal congestion is not unreasonable, although other interpretations are possible. It is easy enough to carve out of the whole mass of cases of paraplegia a group presenting the same symptoms as those which existed in Dr. Radcliffe's patient and to label them accordingly; but I fail to see any ground for supposing that these cases have more claim to the title of spinal congestion than any others in which the paraplegia is incomplete and terminates in recovery. Dr. Radcliffe lays stress upon the sudden commencement as characteristic. Now, it is obviously a question that can be settled by direct observation, whether a paraplegia of which the onset is sudden does really get well more often than one which begins gradually. But, in the meantime, I find that Erb lays stress upon a *slow* development of the symptoms as distinguishing hyperæmia from other morbid states of the spinal cord. He also mentions, as one of the most important signs of this affection, its fluctuating and changeable course. I have, however, seen at least one case of myelitis in which inexplicable variations in the severity of the paralysis and of the anæsthesia occurred from day to day. (See Dr. Moxon's "Croonian Lectures," R.C.P., 1881.—Ed.)

Reflex Paraplegia.—It has long been suspected that a loss of function

in the spinal cord, causing paralysis of the lower limbs, may be an indirect result of certain visceral diseases. Such an affection, indeed, would be fairly comparable with one to which I have already alluded at p. 348; two cases are there mentioned, in each of which a paresis of the muscles of one arm was caused by a carious tooth. Dr. Hammond relates the case of a young lady, aged twelve, who was brought to him on account of a paraplegia which had developed itself very suddenly. He administered several doses of santonine, followed by castor oil; this led to the discharge of a number of round worms, and the paralysis disappeared in the night.

Within the last few years there has been much discussion as to the way in which this kind of paralysis is brought about. Mr. Stanley, who was perhaps the first to describe it, was contented ("*Med.-Chir. Trans.*," 1833) to refer it to "irritation, propagated through the sentient nerves to the spinal cord," whence he supposed that "the impression" was "transmitted through both the motor and sentient spinal nerves to the limbs, here occasioning an impairment both of sensation and of the power of motion." Obviously, however, this explanation goes but a very little way, and there is nothing gained by the notion of a positive transmission outward toward the periphery. In 1860, Dr. Brown-Séquard published in the "*Lancet*" a theory, which he had elaborated in detail, and according to which irritation of afferent nerves in a diseased organ was supposed to set up a reflex spasm in the blood vessels of the spinal cord, rendering it anæmic, and so impairing its nutrition and its functions. In the following year, however, Sir William Gull refuted this opinion in the "*Guy's Hospital Reports*," and no one now accepts it. Nor has any support been accorded to a not very intelligible suggestion of Jaccoud's, that reflex paralysis is due to an "exhaustion" of that portion of the cord upon which fall the stimuli conveyed upward by the sensitive nerves belonging to an irritated part. Thus the field remained clear for the entrance of the modern doctrine of "inhibition," which in theory meets all the difficulties of the case satisfactorily. Its application to paraplegia seems to have been first made by Lewisson ("*Reichert's Archiv*," 1869). In a series of experiments on rabbits, he succeeded in paralyzing the lower limbs by forcibly squeezing between his fingers the kidney, or the uterus, or a large loop of intestine. The loss of power is described as lasting only as long as the pressure was continued, or a little longer, and as always disappearing abruptly. Now, nothing is easier than to refer nervous phenomena of all kinds to "inhibitory" influences; but this very fact makes it essential that the pathologist should never extend the area of inhibition a hair's breadth beyond the limits to which a strict interpretation of physiological observations would confine them. The paralytic affections already referred to as being caused by diseased teeth, and the paraplegia from worms in Dr. Hammond's case, were all in perfect accord with Lewisson's experimental results, since they subsided as soon as the source of irritation was removed. Dr. Graves has placed on record the case of a man who was admitted into the Richmond Hospital in 1835 with partial paraplegia of two weeks' duration, and a tight stricture, which had existed for some months. He had recently been much exposed to cold and wet, and this might have been regarded as the cause of the paralysis. But in a very few days after the first introduction of a catheter a remarkable improvement took place in his legs and in his back, where he had had much pain. In fact, the change was almost sudden; and within a month the power of the lower limbs was completely restored. It would seem that we may fairly regard this as an instance of reflex paraplegia. I believe that cases have been recorded of women in whom the reduction of a displaced uterus has been followed by a marvelously rapid recovery from paralysis; but it is then scarcely possible to exclude hysteria as a cause of the loss of

power. On the other hand, the immense majority of cases which are supposed to be of reflex origin run a protracted course. It is true that the disease which is supposed to be the starting point of the inhibitory influence is often itself permanent and incurable; but sometimes it subsides entirely, and yet the paralysis persists.

In such cases, it is only reasonable to admit that there is something more than a mere reflex paraplegia; and, indeed, myelitis has sometimes been discovered after death. A particular group, in which the paralysis has appeared to be secondary to some affection of the bladder or of the urethra, has been known by the distinctive name of "urinary paraplegia." Three instances of it were recorded by Gull in 1856 ("*Med.-Chir. Trans.*," xxxix); in each of them a part of the spinal cord was found softened. Two similar cases have come under the observation of Leyden; the paralysis seemed to have started from a vesical affection, which in one patient was itself the result of a long-standing stricture; there was a limited inflammation of the upper portion of the lumbar enlargement, with granule masses. Leyden remarks that the seat of this lesion, corresponding exactly with the spot at which the nerves of the bladder enter and leave the cord, suggests the conclusion that the morbid action had extended along their fibres. This opinion has since been supported by experiments made by Tiesler and Feinberg and Klemm, each of whom succeeded in generating an inflammation of the lower part of the spinal cord in animals by setting up neuritis of the sciatic nerves. In a dog operated on by Tiesler there was actually a collection of pus in the substance of the cord "at the point of exit of the sciatic plexus." Roesingh, however, has repeated some of these experiments with negative results, so that the question can hardly be regarded as finally settled. And, as Erb points out, no observer has as yet traced an inflammatory process step by step along the whole course of the nerves.

Sir William Gull several years ago proposed a different route for the transmission of the morbid action, namely, by the veins. In a man who died of paraplegia, consecutive to a long-standing stricture, he found a small quantity of pus lying outside the sheath of the dorsal part of the cord; and one of the vertebral veins in the lumbar region was full of pus. But it is mentioned that there was a large slough over the sacrum, and, as we now know that a putrid inflammation often enters the lower end of the spinal canal from a bed sore, I think that most probably the suppuration there and the venous thrombosis arose in that way shortly before death, and were, in fact, indirect consequences of the paralysis.

There would be no difficulty in collecting from medical works and journals a large number of cases of supposed reflex paraplegia, but they would need to be most carefully criticised. In the Guy's Hospital Library copy of the "*Medico-Chirurgical Transactions*" for 1833, containing Mr. Stanley's paper, there is a pencil note by Dr. Wilks, to the effect that the majority of his cases were wrongly interpreted, and were really examples of a primary inflammatory softening of the cord, attended with secondary cystitis and nephritis. There are, indeed, two cases to which this remark does not apply: one is that of a patient admitted into St. Bartholomew's Hospital for retention of urine (attributed to a severe gonorrhœa, the discharge of which he had stopped by injections), who became paraplegic and died in about a fortnight; the other, of a man who was getting well of a gonorrhœa when he was seized with paralysis extending up to the umbilicus, and fell back dead in his bed sixteen hours afterward. But I shall hereafter have to relate instances in which septicæmia set up rapidly fatal cerebral symptoms for which no adequate explanation could be found at the autopsy; and it seems to me likely that the cases which I have just quoted,

of Mr. Stanley's, were really of that nature, rather than merely reflex, or due to inhibition of the spinal centres.

On the other hand, when a chronic paraplegia develops itself in a person who has for years had a stricture, or who has recently had dysentery or some other intestinal affection, or who has a retroflexed or prolapsed uterus, one must not forget that this may be a mere coincidence. Brown-Séquard formerly laid down a series of fifteen criteria, by which he thought that a reflex paraplegia might be distinguished from one due to an organic lesion in the cord. But now that we know that myelitis is actually present in most of the cases supposed to be of reflex origin, these criteria would have to be set aside, even if experience had not long since shown that no reliance could be placed on them. And I think the only way in which one can exclude the possibility that the relation between the primary disease and the paralysis is purely accidental, is to show that the cases in question are too numerous to admit of such an explanation. But can this be shown? I must for my own part confess that I have never yet had under my care a patient who appeared to me to be suffering from reflex paraplegia.

Hysterical Paraplegia.—It is a well-recognized fact that hysteria is a not infrequent cause of paraplegia; the proof being that young women who have previously suffered, or are actually suffering, from other hysterical symptoms are apt to be affected with paralysis of the legs, which after a time quickly gets well, or even suddenly, under the influence of some mental or moral shock. To define as accurately as possible the characters of such an affection must evidently be of great importance in reference to prognosis and treatment, for the diagnosis cannot be taken for granted upon the mere fact that the patient has hysteria, and (on the other hand) affections really hysterical are occasionally met with in persons who do not present obvious indications of that disease. But there is a remarkable want of agreement among writers as to the kind of paraplegia which is most likely to be due to such a cause. Radcliffe says that the paralysis is usually incomplete; Wilks, that its very completeness is, in a doubtful case, an argument against its being due to real disease. According to Wilks there is loss of sensation, and most writers agree with him. Duchenne attached much importance to the presence of a diminution of "electro-sensibility" without loss of "electro-contractility;" but Reynolds has related two cases in which tactile sensibility, sensibility to electricity, and electro-contractility were all perfect. The features on which he seems to lay most stress were that although each of these patients could raise her legs from the bed, either separately or together, and could move all her toes, yet when she was made to attempt to walk, even if supported by a strong arm on each side, no amount of help could prevent her from staggering and tumbling down to within a few inches of the ground, from which position she would nevertheless recover herself without assistance. As regards micturition, Reynolds states that while retention of urine is itself common in hysteria, he has found that loss of power over the bladder and rectum is not generally present in hysterical paraplegia; Wilks, that the patient "at all events, does not wet the bed, she merely has retention;" Radcliffe, that "the bladder and rectum are little, if at all, under control; less so, as a rule, than in common paraplegia." Wilks seems to insist somewhat on the fact that hysterical girls remain plump, in spite of their being affected with paralysis. I remember that Sir William Gull used to point out the coldness and pallor of the surface of the legs in such patients, as contrasting with their condition in cases of organic disease of the cord.

Concussion of the Cord.—In ordinary cases of fracture and dislocation of the spine, such as come under the care of the surgeon, the paralysis, which is commonly present from the first, is attributed either to crushing of the

cord by a displaced vertebra or to effusion of blood. A case recorded by Gull shows how a comparatively slight accident may be attended with fatal consequences in this way. A man was carrying coals on his back down some cellar stairs when his foot slipped, so that he fell, and the sack of coals upon him. He died in thirty-four hours. Two of the cervical vertebræ were found torn asunder, but this was not the cause of the paraplegia from which he had suffered immediately after his fall, for there was no displacement of the bones nor any injury to the posterior ligament or the spinal membranes. There were, however, several spots of ecchymosis in the posterior gray cornua of the cord, as well as in the posterior white columns and the posterior half of the left lateral column. It is perhaps worthy of notice that even these lesions did not actually explain the paraplegia, for there is no proof that the motor tracts on both sides were injured. This, however, illustrates exactly the view which should be taken of capillary ecchymoses of the nervous centres, whether cerebral or spinal. It is not that they themselves produce paralytic or any other symptoms, but that their presence, showing that the violence was sufficient to tear asunder vessels, shows also that it must have been enough to lacerate the nervous elements themselves, which are more delicate. There can be no doubt that any accident which fractures the vertebræ is also capable of setting up myelitis, and that secondary paralysis from this cause would be often observed were it not that the direct effects of the accident altogether conceal it.

These remarks tend, I think, to explain the fact that spinal injuries are sometimes followed by paraplegia, and may even terminate fatally without any morbid change whatever being discoverable, even with the microscope. Leyden says that he met with an instance of this kind in which death occurred within five days, and in which he failed to detect any lesion, even after having hardened the tissues with chromic acid. Such cases are commonly described as cases of *spinal concussion*, and it is probable that they depend upon a molecular change in the nerve fibres and cells of the cord, due to the jarring force of the injury. The occurrence of this change would, of course, be prevented if the vibrations should in any way be hindered from reaching the nervous centres. Mr. Erichsen, for instance, says that after a railway accident those persons who were sitting with their faces to the engine are less likely to suffer from spinal injuries, because they would be thrown forward and could break their fall with their arms and hands, whereas those who sat the other way would be driven against the back of the carriage when its motion was suddenly arrested so that there would be scarcely anything to prevent the spinal cord from being violently shaken. But I must confess that I am unable to understand another statement of Mr. Erichsen's, namely, that persons who are asleep at the time are less apt to be affected than those who are awake. Even if we admit that the functional activity of the spinal centres is diminished during sleep, it seems impossible that this can prevent their undergoing the direct physical effects of the shock.

The cases of which one can most confidently speak as being examples of concussion of the cord are those in which a blow or a fall is instantly followed by paralysis, and in which recovery takes place so rapidly that the alternative diagnosis—hemorrhage—can be satisfactorily rejected. Such an instance is, perhaps, one related by Erb. A man, aged fifty-five, fell twenty feet from a tree, upon his feet and buttocks. He was not unconscious, but was at once unable to walk, and had to be carried home. He experienced severe pains in the sacrum and in the lower limbs, but these passed off after a time. There was no loss of sensation, but the legs were said to be entirely motionless for a week. When he came under medical observation at the end of four weeks he could only make a few steps at a

time, and slowly and with hesitation. Reflex movements could be produced as usual. The excitability of the nerves and muscles by electricity was much diminished. Galvanic treatment, applied both to the spine and to the legs, was attended with great success. He was soon able to walk well, and in three weeks he was discharged cured.

But in most cases in which railway accidents are followed by paralysis this does not manifest itself until some time has elapsed. The interval is often of several days' duration. Mr. Erichsen says that it may even last two or three months. During this time the patient is not, indeed, well; he is suffering from other effects of the injury, but he frequently has no idea that his spine has been hurt. I really do not see how the diagnosis of "concussion" is to be established as against that of myelitis when the symptoms are thus remote. However slight they may be, one may surely conceive them to be caused by an inflammation of the cord limited to a sufficiently small area of its substance. And the fact that they generally subside after a time affords no argument to the contrary, now that we know that myelitis is a disease from which recovery is possible.

Acute Ascending Paralysis.—I have already alluded to the fact that myelitis sometimes spreads rapidly upward along the cord, and destroys life in a few days. To such cases, which at Guy's Hospital have not been very infrequent, the name of acute ascending paralysis might be fairly applied. The most recent writers, however, reserve it for a different affection, one in which the most minute scrutiny has hitherto failed to reveal any lesion of the tissue of the cord. The earliest notice of this form of paraplegia appears to be one of Landry, in the "*Gazette Hebdomadaire*" for 1859; and it is often called "Landry's paralysis," by way of distinction. It is said to be definitely characterized by its symptoms. For in a case of quickly fatal myelitis there is generally marked anæsthesia, the bladder and rectum are totally paralyzed, bed sores are formed at a very early period, and the faradic excitability of the muscles soon becomes extinguished. But in the acute ascending paralysis of Landry none of these phenomena are observed.

I take the following description from the excellent account given by Erb in Ziemssen's "Handbuch." Sometimes there are prodromata, consisting of slight febrile disturbance, malaise, dragging and shooting pains in the back and limbs, sensations of numbness and formication in the feet and in the tips of the fingers, and, above all, a feeling of great muscular exhaustion and weakness. The patient may go on complaining in this way for a day or two, or for a week, or even (in one recorded instance) for six weeks. Or no such symptoms may arise, the earliest indication that anything is the matter with him being a loss of strength in the lower limbs, which rapidly passes into a complete paraplegia. The feet first become motionless, then the legs, and afterward the thighs. Soon the trunk is involved; straining becomes impossible during defecation, and in coughing or sneezing; the intercostal muscles are paralyzed in succession from below upward. At the same time, or even earlier, the hands are affected; the patient is unable to write or to feed himself; his grasp rapidly becomes enfeebled; the loss of power extends to the arms and shoulders. Lastly, the muscles supplied by the upper cervical nerves, including the diaphragm, fail in their turn; the act of swallowing becomes impossible, and death by suffocation closes the scene. Toward the last it is sometimes noticed that the speech is embarrassed, that liquids regurgitate through the nose in consequence of paralysis of the palate, and that the masticating muscles and those of the face are weakened. The pupils are sometimes unequal, and a transitory diplopia has twice been observed. There may also be a very rapid action of the heart; this is believed to depend on the lesion having reached the upper part of the cervical cord.

In some cases the order in which different parts are paralyzed is said to be reversed. The fatal illness of the great naturalist Cuvier was of this kind, and terminated in less than seven days. His first symptoms, a sensation of discomfort at the epigastrium, appeared on May 7th, 1832. Next morning he experienced a difficulty in deglutition, and in the evening he could swallow nothing, and had marked loss of power in the upper limbs. The paralysis generally became absolute, and affected the lower limbs also. Such a course of events can only be explained on the supposition that the morbid change (of whatever nature) descends the cord, instead of ascending; and that it is limited to the gray matter, leaving the white columns unaffected, which, of course, contain at each level fibres belonging to all parts of the body below. It might be thought doubtful whether these cases can fairly be brought within the very narrow definition of Landry's paralysis; but they resemble the typical cases, not only in there being no recognizable lesion, but also in the fact that the sensibility of the paralyzed parts remains perfect.

For, as I have already observed, what is really most remarkable and characteristic of this disease is the complete absence of all other symptoms, with the exception of the loss of motor power. The affected limbs lie flaccid and free from spasm. At first one can excite reflex movements in them; after a few days, however, the susceptibility to stimuli diminishes and becomes extinguished, but the electrical contractility of the muscles persists to the last; nor do they ever waste. The functions of the bladder and rectum are unimpaired; there may for a time be retention of urine, but this quickly passes off, so that a catheter seldom has to be used. The patient may complain of slight subjective feelings of numbness, or of formication, but the paralyzed parts are, in the main, still perfectly sensitive to touch and to painful impressions. No pains are experienced in the affected limbs, nor is there any tenderness of the spinal column. The patient may be so completely free from discomfort, and from all the ordinary signs of serious illness, that it may be difficult for those about him to realize the gravity of his state. In the case recorded by Landry, M. Gubler, who had the charge of it, thought for a day or two that the paralysis was feigned, nor was there any apprehension of danger when the disease was within eight hours of a fatal termination.

As I have already stated, pathological anatomy has hitherto thrown no light whatever upon the nature of this disease. Among the most recent observations are those of Westphal ("*Arch. f. Psych.*," 1846), who examined the spinal cord by the most approved methods in three typical cases, and with absolutely negative results.

The mean duration of fatal acute ascending paralysis is said to be from eight to twelve days. It has been known to destroy life in two or three days, but sometimes it runs on for as many weeks. Some cases, however, end in the patient's recovery. Landry even spoke of eight out of ten cases, which he had collected from various sources, as having terminated favorably; but his paper contains no details which might have enabled one to form an opinion as to the validity of this conclusion. Erb says that the disease may stop at any period of its course, even when the breathing is embarrassed and the power of swallowing and that of chewing are impaired. One must therefore be careful not to give a bad prognosis too positively.

With regard to its ætiology very little is as yet known. It appears to be more frequent in males than in females, the proportion given by writers being as twelve to four. Some cases have seemed to owe their origin to exposure to cold, or to some of the other conditions which have been described at p. 399 as causes of myelitis; some have occurred during convalescence from acute diseases, among which are mentioned typhus, diphtheria, pleurisy, and varioloid—a statement that must not be taken as

referring to the ordinary diphtheritic paralysis, of which the characters are altogether different. Westphal thinks that the affection of Landry may be due to the operation of some hitherto unrecognized poison; Erb suggests that its nature is the same as that of tetanus—an analogy to which one may fairly object that it explains nothing. It would seem to be of exceedingly infrequent occurrence in England, if I may judge from the fact that I cannot find a single typical case recorded at Guy's Hospital.

Alcoholic Paraplegia.—Under this name Wilks has described a form of incomplete paralysis of the lower limbs, of which he has observed many instances, chiefly "in ladies who have given themselves up to the pleasure of brandy drinking." Pains in the legs and anæsthesia are also present. I doubt whether I have ever seen a case of this kind in hospital practice, but I can call to mind one or two to which I have been summoned in private. Wilks speaks of the affection as a meningo-myelitis; and thus I might perhaps have contented myself with mentioning alcohol among the causes of inflammation of the cord. But I do not know whether a definite local change in it has ever been found on post-mortem examination. I rather infer, from Wilks's account of this variety of paraplegia, that it should be regarded, not as an independent affection, but as forming a part of a general change in the nervous centres, the effects of which happen in certain persons to be more apparent in the lower limbs than elsewhere. But there can be no doubt that it is of the utmost importance to recognize the true nature of such cases, nor that it may very easily be overlooked. For Wilks has found that entire withdrawal of alcohol, with administration of good food, quinine and opium, will often bring about a more or less complete restoration to health.

Syphilitic Paraplegia.—That paralysis of the lower limbs may be one of the remote effects of syphilis has for several years been a well-known fact, but morbid anatomy has hitherto done very little to define the exact nature of the changes which occur in such cases. As to their relative frequency there is some discrepancy of opinion. Dr. Buzzard has expressed his conviction that, in a person between twenty and forty years of age, a case of paraplegia, when not associated with Bright's disease or embolism, is (like hemiplegia under the same conditions) the result of syphilis in nineteen cases out of twenty. But in this statement he appears to ignore the important distinction between the pathology of the brain and that of the cord, which is represented by the common occurrence of idiopathic myelitis as compared with the infrequency of a primary local encephalitis leading to softening; and I cannot doubt that Heubner is right when he asserts that the *lues venerea* affects the spinal cord far less often than the brain. On the other hand, Buzzard's limits of age appear to be too narrow, for in a case observed by Wilks the patient was a woman of fifty-three, and among the five or six other cases which have been examined in the post-mortem room of Guy's Hospital within the last few years, one occurred in a man aged fifty-seven, another in a woman aged forty-seven. But, in truth, this form of paralysis has hitherto been studied chiefly at the bedside. Many years ago Sir Benjamin Brodie said that he had several times seen paraplegia stopped in its course by bichloride of mercury or by Plummer's pill. The inference has since been drawn that the cases in question were syphilitic, although there is nothing to warrant it in Brodie's brief statements concerning them. A similar conclusion has sometimes been based upon the striking results which now and then follow the administration of iodide of potassium for paraplegia, a wonderful instance of which I remember seeing in the practice of Dr. Wilks about twenty-five years ago.

The frequency with which nodes are seen upon the inner surface of the skull, pushing the dura mater inward, at one time led to the supposition

that a similar affection of the bony walls of the spinal canal might occur. But in the few autopsies which have been made of late years, since the attention of pathologists has been specially directed to the subject, the vertebræ themselves seem always to have been healthy. In some instances gummata have been found upon the membranes, or growing from them into the substance of the cord. Such cases, however, come properly under the head of *compression paraplegia*, and I shall therefore refer to them in the next chapter. A gumma *beginning* in the interior of the cord is, I believe, an affection as yet unknown to pathologists.

On the other hand, in certain cases supposed to be examples of a syphilitic paraplegia, no obvious morbid change has been discovered, or only a softened state of the cord; and, as I have already remarked, Erb mentions syphilis as an important cause of myelitis. In 1878 I examined the body of a man who died of the effects of paraplegia, and who was said to have suffered from venereal disease seven years previously, this having been followed by an eruption and by recurrent sore throats, and by the occurrence of a miscarriage on the part of his wife—whom he married after having the chancre. I found a common myelitis with a recent slight meningitis, but as there was no other evidence of syphilis in any organ, I came to the conclusion that this affection was probably not syphilitic. A year before, however, I made an observation which seems to me of considerable importance. A man, aged twenty, was actually under treatment by Mr. Davies-Colley for syphilis when he became paraplegic, and was transferred to the care of Dr. Wilks. He died two months later. The cord in the mid-dorsal region was flattened and soft for about an inch and a half of its length; the antero-lateral columns and the gray matter, etc., were especially affected, the latter being of a rusty brown color. To the naked eye there was no obvious change in the pia mater. But when a piece of it, corresponding with the softened part, was placed on a microscopic slide and examined with a lens, the walls of the arteries were at once seen to be enormously thickened and degenerated. By reflected light they looked like solid, opaque, white cylinders; by transmitted light their tissue appeared black. Neither Dr. Goodhart nor I could find any in which the affection was in an earlier stage, so that we might have compared it with that of which Heubner has given so complete a description, as occurring in the cerebral arteries. I think it is very likely that many cases, such as have hitherto been supposed to be examples of softening from syphilitic myelitis, may hereafter be traced to the defective blood supply consequent on a syphilitic affection of numerous vessels in the spinal pia mater; and it may be that the same lesion will be found to account for those other cases in which as yet no morbid change whatever has been detected.

Hemorrhage into the Cord.—As a primary lesion this is very rare; so rare, indeed, that Charcot and Hayem have recently expressed doubts as to whether it has ever been observed, and have endeavored to show that in all the cases which have been recorded as examples of it there was an antecedent softening, the result of myelitis. This view would dissociate it altogether from the common cerebral hemorrhage of old people; for the softening which so often precedes that affection is not inflammatory, but depends upon disease of the arteries of the brain, and appears to have no representative among the morbid changes to which the cord is liable. But there certainly seems to be some instances in which extravasations of blood into the spinal cord and those into the brain are strictly comparable with one another. A case in point is one of Jaccoud's—that of a woman, aged sixty-two, who was brought into the hospital with complete paraplegia which had suddenly appeared four days before. The diagnosis was given that there was hemorrhage into the lumbar enlargement of the

cord. Six days afterward she was found dead in her bed. The autopsy showed that the gray matter, up to the highest roots of the lumbar plexus, was occupied by a clot which was beginning to undergo disintegration. The cause of death was an immense effusion of blood into the left lateral ventricle, the crus cerebri, the pons, and the bulb. Erb says that the part of the cord into which hemorrhage is most apt to occur is the central gray substance. Sometimes the blood is diffused throughout its whole length; in other cases it destroys the entire thickness of the cord at a certain level, so that a bluish discoloration can be seen through the pia mater.

According to Erb, this affection is of more frequent occurrence in persons between the ages of ten and forty than in those who are older. A very striking instance has recently been recorded by Goltdammer ("*Virchow's Archiv*," 1876) in a girl between fifteen and sixteen years of age. She was one day about noon sitting quietly on a chair, when she suddenly experienced a severe stabbing pain in the back between the shoulder blades, which compelled her to cry out. The pain quickly passed into the right arm, and then into the left arm and the lower part of the chest: at the pit of the stomach it extended round the body like a girdle. She fell from her seat, and at once noticed that she could not move her right leg; half an hour later her left leg also was paralyzed; within two hours she was taken into the hospital and was found to be perfectly paraplegic, with anæsthesia up to the nipples, and loss of power over the bladder. Reflex movements could be excited in the lower limbs. The case was diagnosed as one of hemorrhage into the cord below the cervical enlargement. She lived almost exactly a year. At the autopsy there was found a firm cicatricial mass in the gray and white substance of the upper part of the dorsal region of the cord; the nervous elements were completely destroyed; hæmatoidin crystals and granular pigment were present in large quantity, giving a reddish-brown color.

Tumor within the Cord.—For practical purposes, one may include under this head tubercle of the cord, just as we shall hereafter classify the so-called "solitary" tubercles among tumors of the brain. Even so, however, the group consists of a very small number of cases in comparison with any of the more common causes of paraplegia.

Tubercle in the cord forms a rounded or elongated mass, which seldom reaches any considerable size. Two cases were observed in Guy's Hospital in the year 1870; in one the tubercle was no larger than a pea, in the other it was compared to a small cherry. It is worthy of note that in one of them no sign of the lesion was visible on the surface of the pia mater. I made a series of sections at the usual distances from one another, and missed it altogether; it was afterward found by Dr. Reginald Stocker when cutting across the cord in fresh places. The main substance of a spinal tubercle is firm and caseous; its centre may be softened into a creamy liquid; its periphery is sometimes gray, showing a well-marked lymphoid structure; it is surrounded externally by a soft, vascular, pinkish zone, so that it readily slips from its bed when it has been cut across. Leyden says that tubercles more often occur in the upper than in the lower part of the cord. But of three cases of which I have notes there are two in which the lumbar region was the seat of the disease, while in the third it was in the lower dorsal region. Two of the patients were women, aged twenty-eight and fifty-eight respectively; one was a man, aged thirty-six. Dr. Fr. Taylor has told me of a case that he saw in a child. In two of my cases there was extensive pulmonary phthisis; in the remaining one the peritoneum was tuberculous, and the mesenteric glands were caseous.

A new growth in the cord may be either a *glioma* or a *sarcoma*. One that was found in the body of a woman, aged twenty-six, at Guy's Hospital, in 1860, was an oval, soft mass made up of spindle-cell tissue. They are

somewhat apt to undergo mucous softening; but the most remarkable change that is observed in connection with them is the occasional development of an elongated cavity in the centre of the cord, which looks exactly like a dilated central canal, but which is really not so, as is proved by the fact that in transverse sections the remains of the canal can still be seen in front of it. This condition is described by some recent writers under the name of "syringomyelus." Dr. Frederick Taylor has shown me a beautiful specimen of it, in which, however, it is doubtful whether the cyst is not a primary lesion, for no very clear indications of the presence of a new growth can be made out ("*Path. Trans.*," 1878 and 1884).

The symptoms produced by tumors of the cord vary widely in different cases. As a rule, a paraplegia is present which seems to differ in no respect from that which might be dependent upon any other affection. But sometimes the paralysis is of a more limited kind; to such cases I must briefly refer hereafter when I am discussing those diseases which are confined to special parts of the segmental area of the cord. Lastly, Theodor Simon has recorded several remarkable cases in which there would appear to have been a complete absence of all symptoms ("*Arch. f. Psych.*," 1874).

Reverting now to the question of the *diagnosis of myelitis* from these various affections, we shall, I think, find, if we reflect upon it, that the descriptions given in the last fourteen pages point pretty uniformly to the conclusion that not very much is to be done in this direction. The occurrence of paraplegia in a person known to be suffering from phthisis or any other "scrofulous" disease would, indeed, lead one to suspect the presence of a *tubercle*, but, as I shall presently have to point out, it would be very difficult to exclude caries of the vertebræ (without deformity) producing compression of the cord. So far as I can judge, there is no possibility of distinguishing the paralysis caused by a *tumor* within the cord from that due to a circumscribed transverse myelitis, unless, indeed, there should be very severe and long-continued pain, in which case one would be almost certainly driven into the opposite error of supposing that the lesion was in the bones or in the membranes. I have cited two cases, in both of which *hemorrhage* was, in fact, correctly diagnosed, but, taking into consideration its extreme rarity, I must confess to thinking that each of them was quite as likely to have proved an example of myelitis. At any rate, it is certain that suddenness of onset is not of itself a sufficient ground for attributing a paraplegia to effusion of blood. As regards *syphilis*, the recognition of it as being positively the cause of a paralytic affection of the lower limbs must be based upon the clinical history and upon the presence of other syphilitic lesions rather than upon any peculiarities in the spinal symptoms themselves. But I have no doubt that, as is the case with all the other remote effects of the venereal poison, there are many instances of syphilitic paraplegia in which the most careful scrutiny would fail to elicit any evidence of their real nature; and, seeing how important it is that such cases should not be overlooked, I think that we are often fully justified in prescribing antisyphilitic remedies in the dark. There is, however, one positive indication of myelitis that must always be carefully noticed, namely, a slow creeping up of the paralysis and anæsthesia from the thighs to the groins, and from thence to the abdomen. I believe that it warrants a confident diagnosis of the presence of that disease. We have seen that an *acute ascending paraplegia* may be independent of any recognizable morbid change in the cord.

Among the other forms of paraplegia which have been enumerated, the *hysterical* seems to be the only one that can be placed definitely apart from myelitis. I do not wish positively to assert that all the rest are really examples of a partial or local inflammation of the cord, but I think I am

justified in maintaining that, if they are not so, one cannot at present give a more accurate account of them than by saying that they are among the spinal representatives of the neuroses, which form so large a group among the diseases of the encephalon, and of which the true pathology is as yet unknown.

Prognosis.—I have spoken of myelitis as occasionally terminating in recovery; but at p. 402 I have laid down the rule that the prognosis is generally unfavorable when there is complete paraplegia. To this rule, however, there are some remarkable exceptions. Among the most striking that I know of are two which have been recorded by Wilks, in his "Lectures on Diseases of the Nervous System." One is the case of a gouty man, aged fifty-two, who had been losing power over his lower limbs and his bladder for a fortnight, so that on admission he could not move his legs, and had partial loss of feeling up to the umbilicus. Reflex movements persisted. The urine, which was ammoniacal, had to be drawn off twice daily. Afterward he had a feeling of tightness round the lower part of the chest and the abdomen, and he experienced numbness passing down the arms into the fingers. He gradually got worse, and about a fortnight after his admission he became feverish, with a quick pulse, a red tongue, rigors, and hiccough; a bed sore was forming, and the urine ran away from him; his mind was clouded. Dr. Wilks thought that he was suffering from suppurative nephritis and had not many hours to live. For some days he remained in a precarious state, and then his constitutional symptoms abated, and he began to regain some degree of power in his legs. From this time he made a rapid recovery; he sat up in bed, he ceased to require the catheter, he got into a chair, he asked for crutches, he walked about the ward, and finally he left the hospital; this taking place exactly two months after his admission, and six weeks from the period when his paralysis became complete.*

The other case is that of a woman of middle age, who came in suffering from almost complete paraplegia, which had begun a few days previously. It was accompanied by pain and swelling of the joints—a symptom of spinal disease which I have not yet mentioned, but which I shall presently have to describe. There was some febrile disturbance. She complained of great pain in the limbs, with twitching of the muscles, and of a sense of constriction round the waist. The urine was ammoniacal, and was passed involuntarily, and the sphincter ani was paralyzed. A bed sore formed, which extended until there was a deep slough on the back. All the other symptoms continued for three weeks, after which she began to recover. She then took tonics and was galvanized; and at the end of five months she was discharged from the ward perfectly well. Dr. Wilks, who, I may say, rejects absolutely all refined diagnoses of reflex paraplegia and of anæmia and hyperæmia of the cord, confesses that he does not know what was the nature of the disease in these cases.

Treatment.—The question now is, whether we can by medical treatment influence the course of paraplegia so as to increase the proportion of cases in which such a happy result occurs. And here I fear that we have very little positive knowledge to guide us. At Guy's Hospital of late years it has not been the practice to adopt very active measures in the acute form of the disease. The modern German plan seems to be to use leeches or cupping glasses freely, to apply bags of ice along the spine, to rub in blue ointment or to give calomel internally, in fact, to carry out the "antiphlogistic" treatment very much as English physicians used to employ it thirty years ago. In proof of the value of *ice bags*, a case recorded by Dr. Tibbits, of Clifton ("*Med. Times and Gaz.*," 1871), is quoted: that of a man suffering from myelitis, which was rapidly spreading upward, which had already pro-

* [This patient showed himself several weeks later, still quite well.—Ed.]

duced paralysis of the arms, and which was attended with a temperature of 103.6°, while the pulse was at the rate of 132, and the breathing at 40 a minute. Soon after trial was first made of the cold applications he became easier; the temperature fell to 99.2°, the pulse to 100, and the respirations to 24. In the course of the next ten days he completely regained the use of his arms, but no recovery of power took place in the parts which were first paralyzed, and he died six weeks later, of exhaustion from bed sores. The suggestion is that the issue of the case might, perhaps, have been more favorable if the ice bags had been employed earlier; paralytic symptoms had been coming on for twelve days, and there had been complete paraplegia for eight days. Erb recommends blistering the back and even the actual cautery, undeterred by the risk of setting up bed sores, which he fully recognizes. In "Landry's acute ascending paralysis" he would adopt the same course, besides rubbing the body with cold water, the cold pack, tepid baths, with cold affusion, etc.

A point of great importance is that the patient should, at the earliest possible period, be placed upon a *water bed*. Probably he should not be allowed to lie constantly on his back, but should be shifted from time to time. I believe it is sometimes dangerous to allow him to sit up. Some years ago a man, who had recently been admitted into the hospital with symptoms of acute myelitis limited to the lower part of the cord, was taken out to "have his bed made." When put back he seemed much exhausted, and very soon afterward he died.

The utmost care must be taken to prevent the formation of *bed sores*. The sacral region must be kept dry by being cleansed and wiped with soft towels. Some good may also be done by sponging the skin with whiskey, or alternately with hot and with cold water. Or a large piece of felt plaster may be applied, having a hole in the centre, if there should be a spot already reddened. When an ulcer has formed, Dr. Hammond speaks in the highest terms of Golding Bird's plan of placing a thin plate of silver over it, of exactly the same size as the sore; while a wire six or eight inches long is carried from this plate to another, made of zinc, which is laid on some part of the skin above, but separated from it by a piece of wash leather soaked in vinegar. Galvanic action is set up; and Dr. Hammond states that he has "frequently seen bed sores three or four inches in diameter, and half an inch deep, heal entirely in forty-eight hours." Mr. Spencer Wells is said to have obtained no less striking results. I regret to say that I have no experience with regard to this plan of treatment.

Retention of urine must not be allowed to occur, or the bladder to remain distended. A soft French *catheter* should be used, if interference is necessary, and care must be taken to depress the free end of the instrument between the thighs, so as to draw off the whole of the urine; for if any should be left it will quickly decompose. Probably carbolic oil should be employed to purify, while it lubricates, the catheter. But I believe that the tendency to putrefaction may be completely checked by the administration of salicylic acid, or of some other antiseptic medicine, by the mouth. This practice seems to have been first proposed by Fürbringer. I believe Dr. Pavy introduced it at Guy's Hospital, and I have seen excellent results from it. From half a drachm to a drachm must be taken in the course of the twenty-four hours; it often restores the urine from an offensive alkaline condition to one of normal acidity and odor. When pus is discharged from the bladder in any quantity, it should be regularly washed out with a weak antiseptic solution.

In some of the less complete and acute forms of paraplegia there is evidence of the value of *ergot* administered internally; and I have prescribed it in one or two instances, though not with any striking results. Dr. Ham-

mond relates a case which he considered to be one of "congestion of the cord," and in which it seemed clearly to be of great service. The patient was a Mr. W—, of Tennessee, who had become affected with partial paraplegia, and afterward with paralysis of the bladder, from exposure to cold and damp. He was unable to walk unless he used crutches and had a man on each side of him holding his shoulder. He also had a constant dull aching pain in the loins, and occasional startings of the legs while in bed. All his symptoms were worse in the morning. A teaspoonful of the liquid extract of ergot, taken three times a day, cured him entirely in a month. He had a relapse a few weeks later, but recovered in ten days under the same treatment. No other medicine was given. The tincture of *belladonna* in fifteen-minim doses is said to be useful in similar cases, especially when there is severe pain in the back.

Writers are agreed that *galvanism* should not be employed early in cases of acute myelitis, but Erb says that it may be used from the very first in the ascending paralysis of Landry. The electrodes should be large sponges placed at a considerable distance apart, one upon the neck, the other over the lumbar vertebræ; they may in turn be moved gently up and down, and their direction may be from time to time reversed. When there is reason to believe that the disease is limited to a segment of the cord, one pole may be placed on the affected spot, the other over the front of the chest or of the abdomen. Faradization of the paralyzed muscles may be useful if they are flabby or wasted.

So, again, *strychnia* seems to be valuable only in the later stages of myelitis. But both electricity and strychnia are often very useful in cases such as raise doubts as to their being hysterical or reflex, or due to concussion of the cord. And in spinal neurasthenia their employment from the first is strongly indicated. In this disease, quinine, iron, and other tonics are also of service. Early hours, good food, and exercise, short of fatigue, in the open air, are essential.

On the Continent, certain spas have great reputations for the treatment of the more chronic forms of paraplegia. Erb says that hot baths are apt to be injurious, except at very advanced periods of such cases. Among the places most frequently visited by patients suffering from paralysis I may mention Schlangenbad, Ragatz-Pfeffers, Gastein, Wiesbaden, Toplitz. It is said that in proportion as the elevation above the sea is greater baths can be borne at higher temperatures without ill effects.

HEMIPARAPLEGIA.—The description of the forms of paralysis produced in common by various affections of the spinal cord remains incomplete until I shall have given an account of a variety which results from a lesion limited to one lateral half of it, but destroying that half completely at a certain level. For this is attended with certain symptoms possessing great interest. As might be anticipated, there is loss of power in that half of the body which is on the same side as the disease in the cord, and the upper limit of the paralysis varies with the distribution of the highest nerves coming off from the part of the cord below, or involved in, the disease. Such a condition may fairly be termed a hemiparaplegia; and this equally, whether it is limited to the leg or involves also the corresponding arm. But what could not have been foreseen, at least until Brown-Séquard in 1860 demonstrated its occurrence in animals, in which one-half of the spinal cord had been cut through with a scalpel, is that anæsthesia is present, not on the same side as the disease, but on the opposite side. That half of the body which retains its motor power loses its sensibility, while the paralyzed half has its sensory functions unimpaired or even rather exalted. The explanation of the crossed anæsthesia is the fact—which is now after a long contest admitted

by physiologists—that the fibres belonging to each sensory nerve root decussate in the substance of the spinal cord itself, immediately above their entrance into it. This applies equally to the fibres which convey all sorts of impressions—whether of touch, or pain, or heat and cold; but, according to Brown-Séquard, with the curious exception that the fibres belonging to the muscular sense run upward without crossing. Thus, there is said to be loss of muscular sense, and with it, of electro-muscular sensibility, on the paralyzed and not on the anæsthetic side of the body. Why there should be hyperæsthesia of the skin on the paralyzed side—the susceptibility to tactile and painful impressions and to change of temperature being all increased—is not so clear. The supposition is that the inflammatory action which develops itself in the neighborhood of the lesion must render the fibres unduly sensitive, or else that there must be a suppression of some influence which usually controls or "inhibits" the sensory nerves, being transmitted on the opposite side of the cord to that which conveys the fibres running upward from them to the brain.

Thus it would appear that no disease of the spinal cord proper can give rise to paralysis and anæsthesia limited to one and the same side of the body. If a patient is found to have one of his lower limbs, for example, affected in this way, we are justified in concluding that the lesion must concern the corresponding half of the cauda equina, unless, indeed, it is seated above the decussation of the pyramids in the opposite half of the encephalon.

On the other hand, it must not be supposed that every affection of one side of the spinal cord necessarily causes paralysis of the same side, and anæsthesia of the opposite side of the body. This effect is observed only when the abolition of function in that half of the cord is complete or nearly so. I have repeatedly had occasion to insist that morbid changes in the nervous structures produce loss of motor power more constantly than loss of sensation. Unilateral lesions of the cord offer no exception to this rule. It is quite possible for such a lesion to give rise to complete paralysis of a lower limb, or of arm and leg on one side of the body, without there being any anæsthesia of the limbs on the opposite side. Such cases were formerly known under the name of "spinal hemiplegia." The explanation of them is that the disease, while it is all included within one-half of the cord, nevertheless leaves a part of that half functionally active; perhaps it is limited to the lateral or to the antero-lateral column; at any rate, it does not destroy at once all the gray matter, and all the postero-lateral column, which together constitute the sensory tract.

The vaso-motor nerves are said to be affected on that side which is the seat of the lesion, especially when this has occurred suddenly or developed itself rapidly. The temperature of the paralyzed limb or limbs is at first higher than that of the corresponding parts, by a difference amounting to 1° or 2° Fahrenheit or even more. After a time, however, it becomes normal, and sometimes falls to a still lower level.

With regard to the state of the reflex excitability of the lower spinal centres in cases of hemiparaplegia there is still some uncertainty. On the paralyzed side it has in some cases been increased, in others it has been diminished; on the anæsthetic side it has generally appeared to be normal, but occasionally it has been exalted. Erb says that the bladder and rectum have generally been paralyzed—always when there has been a sudden traumatic lesion—with either complete retention or complete incontinence of the urine, and involuntary passage of fæces. This appears to lend powerful support to the view that the paralysis of these parts in cases of ordinary paraplegia, due to a bilateral lesion limited to the upper part of the cord, depends on an inhibitory influence transmitted downward from the diseased parts to the healthy centres below. An analogous fact,

which has now been noticed in several cases, is that the muscles on the paralyzed side in hemiparaplegia sometimes have their faradic contractility lowered in a marked degree; they may also undergo rapid wasting.

Hemiparaplegia may be due to many different causes, but hardly to so many as those which may produce paralysis on both sides of the body alike, and some of the more rare lesions of the cord are far more apt to give rise to it than others which are of more frequent occurrence. Thus it has often been observed in patients who have been stabbed in the back by a knife or dagger. At first one is apt to wonder that a chance wound should make a section of one-half of the cord with the accuracy of an incision performed by an experimental physiologist, but the shape and size of the spaces between the arches of the vertebræ are probably such as to prevent a cutting instrument from passing across the median line within the spinal cord. Now and then a fracture or a dislocation of the spine has been attended with the symptom in question, and perhaps an effusion of blood into the membranes on one side may cause it, although Erb does not refer to any instance in which this has been observed. A tumor outside the cord, compressing one-half of it, has more than once caused hemiparaplegia.

In other cases this form of paralysis has been due to disease limited to the interior of the cord on one side, whether this be an effusion of blood, a patch of sclerosis or of some other form of myelitis, an intra-medullary tumor, or a syphilitic lesion.

It is obvious that of these various kinds of unilateral lesions some must be more apt than others to affect a considerable length of the cord, and at the same time to involve the roots of a greater or less number of spinal nerves, whether within or without its substance. This is a point of some importance, because it leads to the development, in many cases, of hemiparaplegia of a fresh set of symptoms due to interference with nerve roots. Thus there is commonly an anæsthetic half-zone of greater or less depth passing round the paralyzed half of the body from back to front, and dividing the hyperæsthetic part of the surface below from the normal part above, and the upper edge of this anæsthetic space is said sometimes to present a narrow hyperæsthetic border. The explanation is obvious: the nerves of the region deprived of sensation have had their roots destroyed by the disease; those of the region which is over-sensitive have merely had their roots irritated. The other (or anæsthetic) half of the body sometimes also has a narrow hyperæsthetic half-zone, limiting the anæsthesia above, and due to irritation of fibres which have just decussated at the upper edge of the lesion in the cord. In some cases the patient experiences a disagreeable sense of constriction or severe burning or shooting pains round the trunk, at a level corresponding with that of the nerves whose roots are involved in the disease, and such sensations may be limited to one-half of the body or affect both sides alike. We shall presently see that exact counterparts for all these symptoms are met with in certain forms of paraplegia, especially those which depend on slow compression of the cord.

The course taken by hemiparaplegia varies in different cases, according to the nature of the disease that gives rise to it. It very rarely remains stationary. Most commonly it soon undergoes conversion into paraplegia, as the result of the development of a myelitis affecting the whole thickness of the cord around the original lesion. This, again, often ends fatally, but sometimes, according to Erb, it subsides and allows the symptoms of a unilateral lesion to reappear. They may then persist for years without change, and in certain cases they have been known to terminate in recovery. According to Brown-Séquard, the power of motion is then regained earlier and more completely than that of sensation. If this is the fact, it is in

opposition to what is observed in all other affections, whether of the centres or of nerve trunks.

In the present chapter I have to describe the second group of affections causing paraplegia, to which I referred at p. 396, namely, those in which the primary lesion is outside the cord, and in which the cord is subjected to *slow compression*, annihilating its functions. I have already remarked that in the diseases in question there is a characteristic and special series of symptoms dependent upon the interference with nerve roots at the level of the lesion. Now, the chief of these symptoms is pain, but it so happens that there is an independent spinal neurosis, which is also characterized by pain referred to the back and to other parts. Consequently, although it involves a digression, I must give an account of that neurosis before I describe compression paraplegia.

Rachialgia. "*Spinal Irritation.*"—In 1828 Dr. Thomas Brown, of Glasgow, drew attention to an affection attended with pain and tenderness in one or more of the vertebræ, and termed it *spinal irritation*. He was followed by Mr. Teale, of Leeds (1829), by the brothers William and Daniel Griffin, of Limerick (1834), and by Stilling, of Cassel (1840), the last of whom devoted a volume of 540 pages to the subject. All these observers gave to it a very wide scope. They detected tenderness on pressure over certain spinous processes in persons suffering from various neuralgic and other affections, and they maintained that in all such cases a morbid state of the spine was really the fundamental disease. They even enlarged their definition so as to include transitory paralytic affections and a variety of other neuroses. In this they were no doubt partly influenced by the name which they had adopted, for it would obviously be difficult to exclude from "spinal irritation" almost any "functional" disorder of the lower nervous centres, from tetanus to pleurodynia.

It is not surprising that these views met with much opposition. They were criticised in trenchant style, but very justly, by Romberg, and they have never met with general acceptance in this country. As I have already remarked, Stilling and Ollivier maintained that "spinal irritation" was due to congestion of the cord. More recently Dr. Hammond has asserted that the essential condition in that disease is a spinal anæmia. Thus the boundary lines between it and other affections have been repeatedly shifted, and, on the other hand, many observers have ignored it altogether. This is much to be regretted, because there are undoubtedly some cases which naturally fall under this head, and which have hitherto found no other place in the nosology. For my own part, while I altogether reject the doctrine that the discovery of tenderness over one or more of the vertebræ affords grounds for referring to a particular morbid condition of the spinal cord whatever other pains or neurotic symptoms a patient may present, I yet think it of great clinical importance to recognize an independent neurosis, characterized by pain and tenderness in the back, but unattended with any symptoms of arterial lesion of the cord, of the bony column, or of the cartilages and ligaments. All the most recent writers do, in fact, describe "spinal irritation," but many of them still seem to me to give it too wide a range. I think that they all regard it as an affection of the spinal marrow itself. It may, however, be urged that the organic affections which most closely resemble it lie outside the cord and (at least in this early stage) affect only the spinal nerves. I would, in fact, propose to look upon it as a neuralgia (in the sense in which I have defined that term in pp. 371–3) belonging to the posterior branches of the spinal nerves, and perhaps to filaments distributed specially to the various structures which enclose and support the cord. It is true that the law of Hilton

and of Van der Kolk—by an application of it precisely similar to one which we shall find to be necessary for migraine and some other painful affections of the brain—would explain the circumstance that pain should be referred to the cutaneous nerves of the back, even though its starting point might be within the spinal marrow; and such a view as to the nature of "spinal irritation" is undoubtedly supported by some facts which will presently be mentioned as to the transference of impressions from the posterior to the anterior branches of the nerves in cases of this kind. But, after all, this difficulty as to the centric or eccentric seat of the affection is not peculiar to it, but concerns every other form of neuralgia likewise, as I have already remarked above at p. 373.

It might be thought that if spinal irritation is a neuralgia, its description would properly come elsewhere, but I prefer to give it in this place on account of the importance of the diagnosis between it and the more grave diseases of the vertebræ and of the cord—just as it is convenient to classify pleurodynia with affections of the chest, and gastralgia with those of the stomach; and the analogy leads me to suggest that, seeing how open to misconstruction the term "spinal irritation" is, we shall find great advantage in discarding it, and in substituting for it the name of *Rachialgia*, which was long ago employed by J. Frank, and which is fairly comparable with those that are commonly applied to other local neuralgic affections.

The severity of the pain varies infinitely in different cases. Sometimes there is no spontaneous pain at all; on pressing upon the different spinous processes we may find that some of them are tender; the patient shrinks or complains that we are hurting him, but he may hitherto have had no idea that anything was the matter with his spine. Upon cases of this kind great stress was laid by those who developed the theory of "spinal irritation" in its widest proportions, but I very much doubt whether any clinical significance can in general be attached to such a discovery. The observers in question were in the habit of directing their treatment mainly to the supposed spinal affection, and applied leeches, blisters, tartar-emetic ointment, over the vertebræ. Romberg, however, tells us that very little was effected by these measures, and in my view of the nature of rachialgia one does not see why attention should be concentrated upon it to the exclusion of the more obvious complaints from which the patient may be suffering.

In other cases there is a dull, aching sensation in the affected part of the spine, or even severe pain. Its development is usually gradual. At first it may be felt only after great fatigue or excitement, but as time passes slighter causes excite it, and it is less ready to subside; so that it ultimately becomes constant. It is almost always increased by muscular efforts and by movements of the vertebræ. Thus the patient is unable to walk far, or to stand upright for more than a short time; even sitting at the piano, the writing desk, or the sewing machine may be too painful to be borne.

Sometimes the best way of bringing out the increased sensitiveness of the affected nerves is to pass one's fingers along the spine, so as to press upon the vertebræ in succession; sometimes it is more manifest when a sponge wrung out of hot water is drawn down the back. To the patient this seems to scald the tender spots, whereas elsewhere it should not produce anything that can be called discomfort.

There is often a close anatomical relation between the seat of rachialgia and that of any other neuralgia which happens to be present in the same case; the one corresponding with the posterior, the other with the anterior main branch of a spinal nerve. This statement is, indeed, almost identical with one which used to be made, and insisted on, by Trousseau; namely, that a "*point apophysaire*" is discoverable in most instances of neuralgia. And pressure on the tender vertebra will often bring out, or

increase, the pain in any other part that happens to have its nerves in an irritable condition. Thus one of the cases related by Dr. and Mr. Griffin (at p. 19 of their work) is that of a girl who complained of headaches and pains in all parts of her body. Her whole spinal column was acutely tender. Pressure upon the first or second cervical vertebra caused a pain which shot forward from the occiput to the brow; a little further down, it excited pain in the larynx; over the lowest cervical spine, at the spot where the trachea dips behind the sternum; and still lower, at the middle of the sternum, the ensiform cartilage, and the pubic region successively. This certainly was not due to any direct mechanical impression upon the cord; for when pressure was made behind the trochanter pain was felt at the iliac crest, at the inside of the thigh, and even in the opposite hip; while pressure upon the thigh or knee set up pains in the shins and toes. In other cases there is a fixed spot in the front of the body, to which pain is always referred, whenever any one of several tender vertebræ is pressed upon. Thus another of the patients of the brothers Griffin had great tenderness of all the dorsal and lumbar, but not of the cervical spinous processes. Pressure on the upper dorsal vertebræ caused pain at the middle of the sternum; from the third or fourth dorsal down to the sacrum it excited pain, not in the corresponding points, as usual, but at the ensiform cartilage. Pain at this spot was even brought on by pressure behind the trochanter, upon the muscles of the thigh, or over one knee joint, and the patient felt the same pain if she chanced to tread on uneven ground, or if a pebble came beneath her feet in walking. To ascertain whether the seventh and eighth dorsal vertebræ were, as usual, more affected than other parts of the spine, they made rather firm pressure upon them. The result was that she suddenly tumbled forward insensible, and would have struck her face against the floor had not some one caught her. Such an occurrence might well appear almost incredible; but the same observers have placed on record other similar cases. One of their patients was a lady who complained of pain in the face, but who "had no conception that her spine was at all affected." When the second cervical vertebra was touched she sprang up with frightful suddenness, as if a needle had been driven through the cord, and then fell in a state approaching to insensibility. Out of this stupor she twice started up in the same way, and as often dropped back powerless, her countenance evincing the utmost terror and agitation. As soon as she could speak she said that she had felt a numbness and sensation as of pins and needles in all parts above the ensiform cartilage. She would on no account permit her neck to be touched again. A third instance was that of a young gentleman, who described himself as suffering from a chronic liver complaint. Pressure upon the spinal column was excessively disagreeable to him. When the finger rested on one of the dorsal vertebræ he grew pale and terrified, experiencing a sudden sensation or thrill through every nerve in his frame. He had an unpleasant feeling about the part for the remainder of the day, and shuddered at the idea of allowing the pressure to be repeated. After a few weeks, however, the experiment was tried again, and with precisely the same results. A fourth case was that of a boy, aged twelve, who fell forward insensible, as if he had been shot, as soon as slight pressure was made upon the second lumbar spine. I believe that phenomena somewhat similar, although in a less marked degree, are not uncommonly observed. They undoubtedly lend support to the view that the seat of rachialgia is in the cord itself rather than in the nerves. The late Dr. Anstie met with a case in which pressure on one spot, over the lowest cervical vertebra, caused exquisite pain, a sensation of extreme nausea, and disappearance of the pulse at the right wrist, that on the left side remaining unaltered. The patient,

a young lady, was seen by Dr. Walshe, Dr. Reynolds, and Dr. Bridge, as well as by Dr. Anstie. Dr. Frederick Taylor has mentioned to me the case of a young child, brought to him at the hospital, in whom it had been noticed for a considerable time that pressure upon the seventh cervical vertebra at once gave rise to a marked pallor of the left side of the face, which lasted a few minutes.

Dr. Radcliffe has suggested that the *cause* of rachialgia (or, as he terms it, "spinal irritation") is often a strain of the back, or a blow, the occurrence of which may have been forgotten by the patient. This supposition seems to me to be strikingly confirmed by the fact that slight railway accidents so often give rise to it. The so-called "Railway spine" is, in fact, in the immense majority of cases an affection of this kind. Dr. Anstie's patient, to whom I just now referred, had received a very slight contusion in a collision. At the same time her sister was severely injured; she nursed her assiduously for three or four months, and then her own health began to be seriously impaired. Many observers think that no reliance can be placed on the statements of persons who have met with such accidents, so far as their subjective symptoms are concerned. But I have seen several cases in which there was abundant collateral proof of good faith. A general "nervous susceptibility"—if I may use the term—also plays an important part in the ætiology of the complaint. It is far more common in women than in men, and in persons between fifteen and thirty years of age, than in those at earlier or later periods of life. An inherited "neuropathic tendency" predisposes to it. Like other neuralgiæ, it often affects women who at the same time are obviously suffering from hysteria; and thus it may be associated with any of the various symptoms of that morbid state. Among other causes which are mentioned are over fatigue, exhaustion by night watching, sexual excesses, onanism, unsatisfied sexual desire, mental emotions, insufficient food; but probably none of these are capable of directly causing it.

I propose to leave the *diagnosis* of rachialgia until I shall have described those more serious diseases which in their early stages are liable to be mistaken for it. There may, indeed, be a question whether it ever undergoes an actual conversion, or development, into some one of those diseases in progress of time. Erb leaves this question an open one; but such a supposition seems to me to be improbable. The course of the affection is toward recovery, but relapses are common. Very often, too, the subsidence of pain in a particular spot in the back is followed by the appearance of neuralgia elsewhere, or of a different neurosis, either hysterical or of some other type.

In the *treatment*, a most important point to be determined is whether or not rest in the horizontal posture should be insisted on. Some of the patients whom the late Mr. Hilton used to keep lying on their backs for months together, with sand bags to prevent any movements of the spine, were, I believe, sufferers from rachialgia rather than from actual spinal disease; and he undoubtedly obtained good results. Mr. Teale, however, says that he succeeded in curing several persons belonging to the poorer classes while they were still pursuing their laborious avocations; and in many cases the general nervous state renders it exceedingly desirable that the patient should have fresh air and change of scene, and should even be encouraged to take moderate exercise, short of fatigue. An abundant supply of good food is of great importance. Dr. Radcliffe says that a somewhat bold use of alcoholic drinks is a cardinal point in the treatment; but I strongly incline to the opinion of Dr. Anstie that they should be taken in great moderation, and only with the meals. Quinine, the tinctura ferri perchloridi, strychnia, and cod-liver oil are each of them valuable medicines.

The application of blisters to the spine is recommended by all English observers. The older writers speak no less favorably of leeches and of cupping. These measures are now out of fashion; but in one very severe and obstinate case which arose out of the Thorpe railway accident, and which I saw with Mr. Erichsen and with Mr. Robinson, of Norwich, nothing gave so much relief as the repeated application of leeches at intervals of a few weeks, and especially at the catamenial periods. I must, however, add that the patient, a married lady, was stout and florid. Other applications that have sometimes proved useful are turpentine liniment and unguentum veratriæ; or a bag of hot sand may be placed along the spine, or a galvanic current passed through it in the manner already directed at p. 381, for neuralgic affections in general.

Compression Paraplegia.—Forty or fifty years have passed since Ollivier and Cruveilhier described as a separate form of paraplegia that which is produced by the "slow compression of the cord." The importance of thus grouping together various affections of the parts which surround and support the spinal marrow seems not to have been recognized by the writers who followed them. But, recently, several observers have worked out the idea in detail; among others, Charcot, who, however, seems to me to err in including in his description tumors and tubercles lying in the interior of the cord itself. For, as he himself admits, some of the most characteristic phenomena are absent in such cases. In the present section I propose to deal only with those lesions of the vertebræ or of the spinal membranes which exert pressure from without upon a more or less limited part of the cord, and so cause paralysis. And the simplest method will be first to enumerate the diseases themselves and the conditions under which they occur, and then to describe their symptoms and how they are to be distinguished from one another. This will be dealing with some of them in rather a fragmentary manner, for they are not always attended by paraplegia, and of cases in which this symptom is absent I shall take no heed. But the boundary lines which I shall follow will be almost exactly those which divide the practice of the physician from that of the surgeon.

In the first place, it is to be remarked that some affections of the spine never cause paraplegia. Thus the cord seems always to escape compression in cases of lateral curvature (or *scoliosis*, as it is often called), however extreme may be the deformity. Leyden, indeed, alludes to one instance in which the patient was unable to stand or walk for more than a short space of time, but such an effect is quite exceptional. Whether the vertebral canal is ever narrowed in cases of osteo-arthritis I am not sure. In 1838 Aston Key related, in the third volume of the "*Guy's Hospital Reports*," two cases of paraplegia in each of which Wilkinson King—a pathologist of deserved reputation—found an intervertebral substance projecting backward, with raised lips upon the edges of the two adjacent vertebræ, or even (in one instance) with an ossified posterior common ligament bridging over the space between them. It was taken for granted that this was the cause of the paralysis, but, so far as I am aware, no similar case has since been observed. I have already remarked, at p. 413, that syphilitic nodes seem never to grow from the bodies or arches of the vertebræ inward, so as to interfere with the cord.

1. *Caries of the Spine. Pott's Disease.*—The most frequent, and by far the most important, of all the affections that are really capable of giving rise to a "compression paraplegia," is that which, from the kind of deformity produced by it, is in this country commonly known as "angular curvature," but which on the Continent is universally called after one of the greatest English surgeons of the last century, Percival Pott. It consists in the

destruction by ulceration, with or without necrosis, of the body or bodies of one or more vertebræ, which then fall together beneath the weight of the head and upper part of the trunk. The necessary result is a displacement of the corresponding arches and spinous processes, which become bent into a sharp angle or a rounded curve according to the number of the bones which are diseased. Opinions differ with regard to the mode of commencement of this morbid process. German pathologists agree in stating that it begins in the bodies themselves, the intervertebral discs escaping or being implicated only at a late period, and I have seen at least one case in which several of the vertebræ contained caseous masses in their interior, or even irregular cavities, which nowhere touched their surfaces. But, as Wilks long ago pointed out, there is in some cases a source of fallacy in the fact that when a disc is completely destroyed the adjacent vertebræ often lose each about half of its substance, and the remaining halves, coming together, look exactly like the fragments of a single bone ulcerated through by the disease. And I have repeatedly found intervertebral discs above and below the main seat of mischief presenting early changes of such a kind as to convince me that in the particular case under observation they, rather than the bones, were primarily affected. So, in an instance recorded by Sir William Gull ("*Guy's Hosp. Rep.*," 1856, p. 179), death actually occurred from an affection of the cord at a time when the only change was softening of three of the discs, with the formation of a cheesy substance in the middle one and a little "absorption" limited to the adjacent part of the bone.

Another question is whether caries of the vertebræ is to be regarded as "scrofulous." According to my view, this question is to be decided mainly by the presence or absence of associated changes in other parts. Cases of scrofulous kidney and of Addison's disease are, each of them, now and then accompanied by an affection of the vertebræ immediately adjacent; and I could cite several instances in which a similar affection has occurred in conjunction with pulmonary phthisis, or tubercular disease of the testis, or disease of other bones or joints. Their frequency seems to me to prove that Pott's disease of the spine is often scrofulous; but I must confess that in looking over a list of sixteen cases, all fatal by paraplegia, I find less evidence of this kind than I should have expected. It may be that a simple spinal caries will hereafter be recognized as distinct from the scrofulous affection. Dr. Moxon is inclined to such an opinion; he would trace the former disease to accidental injuries, and thinks that it may be characterized by occurring in adults, and by its having a tendency to indurate the bones and to terminate in repair. But it seems to me, on the contrary, that in most instances of permanently cured angular curvature with anchylosis, the active stage of the disease was passed through in childhood. Of the sixteen cases just referred to, in fifteen the patients were adults; four between twenty and thirty years of age, three between thirty-one and forty, seven between forty-one and fifty, and one at the age of fifty-six. The only case in which anchylosis existed was one in which the spinal disease began when the patient was six years old; he died at thirty-two.

Considering how great is the deformity in many of these cases, and how much it alters the relations of the arches of the vertebræ to one another, one could not be surprised if the displaced bones often compressed the cord directly. That this does sometimes occur appears clear from a case of Brown-Séquard's in which a paraplegia which had set in suddenly was removed in twenty-five hours by extension of the spine. But every pathological museum contains specimens which show that the spinal canal generally remains of its full width, however much its direction may be altered. Moreover, as Charcot points out, paralysis often occurs in cases in which there is no curvature at all, while in other cases, in which curva-

ture exists, the patient regains the use of his limbs notwithstanding that the state of the bones remains unaltered. What really presses upon the cord when there is caries of the spine is, as Gull showed in 1856, a mass of cheesy *débris*, which collects between the affected vertebræ and the dura mater, having perhaps been extruded from the diseased fibro-cartilages or from the carious bones as the result of destruction of the posterior common ligament. Michaud has further observed that the outer layers of the dura mater become in their turn converted into a thick, yellow, caseating mass.

2. *Malignant Disease of the Spine.*—This is scarcely less important than caries as a cause of paraplegia. It assumes a variety of forms. Sometimes it is secondary to carcinoma of the breast, or to sarcoma occurring in the neck or in one of the long bones, or to some kind of infecting growth in the testicle. The nature of the disease is sufficiently obvious if there has been a primary affection in any of these parts, especially if a surgical operation should have already been performed ; but it is otherwise when it is seated internally, as (for instance) in the mediastinum. A case in point occurred in Guy's Hospital in 1864; there was no suspicion of any mischief outside the spinal canal until it was revealed by the autopsy. Sometimes the vertebræ become affected with malignant disease by direct extension, as from a cancer of the kidney. Sometimes the spinal lesion appears to be itself a primary new growth. In these cases it is generally a sarcoma of one kind or another, and not infrequently it affects simultaneously a number of vertebræ in different regions as well as other bones, such as those of the limbs, the ossa innominata, or the skull. Thus a careful search may lead to the discovery of a swelling in some distant part of the body, and so may clear up a case that would otherwise have remained altogether obscure.

Sometimes there is a distinct projection of one or more spinous processes, or a mass of the growth can be felt within the substance of the erector-spinæ muscle. Sometimes a tumor in connection with the front of the spinal column is discovered when deep pressure is made in the middle of the abdomen, or, if the cervical vertebræ are diseased, manipulation of the neck may reveal an enlargement on one side corresponding with the transverse processes. But in the great majority of cases one can feel nothing abnormal. The growth lies entirely within the spinal canal except in so far as it occupies the substance of the bodies or laminæ of the vertebræ themselves.

Of fourteen cases of malignant disease of the spine which I have collected from the records of post-mortem examinations at Guy's Hospital ten were in males, four in females. The patients were of all ages, from sixteen to sixty-eight.

3. *Erosion of the Vertebræ by Aneurism.*—Common as it is for an aneurism of the aorta to eat out the bodies of the vertebræ with which it comes into contact, it very rarely penetrates the spinal canal so as to interfere with the cord. We have in Guy's museum specimens from two cases, in each of which paraplegia developed itself before death at a period long after the discovery of a pulsating tumor in the back. But the most remarkable instance of this kind with which I am acquainted is one on which I made a post-mortem examination in 1871. A man, aged thirty, was admitted into the hospital for paraplegia which had begun three months previously. He had first complained of pain in the shoulders, then his left leg became weak and numb, and afterward his right leg, and he experienced a pain as though the abdomen were constricted by a cord. He died without any suspicion of the real nature of his disease. When the erector spinæ were cut into, a large mass of laminated clot was found in its substance on the left side. This belonged to an aneurism of the descending aortic arch, which had destroyed several ribs, and laid open the spinal canal for a space

two inches in length. Some of the clot adhered to the outer surface of the dura mater; the cord itself was flattened, white, and very soft.

4. *Erosion of the Vertebræ by a Hydatid.*—This also is very rare. A striking instance of it is one recorded by Cruveilhier. A large echinococcus lay behind the vertebræ; it filled the groove on each side of the spine beneath the lumbar muscles so as to form two elongated pouches, which were connected together by a narrow neck that also penetrated into the spinal canal. A somewhat similar case occurred to Dr. Moxon in our post-mortem room in 1871, a few months before I met with the aneurism just referred to. The parasite was "multilocular," budding externally. It formed a large elastic swelling on the left side of the spine, and made its way into the canal through the second and third lumbar vertebræ. It compressed the cord, but did not penetrate the dura mater. The patient, a woman of fifty-eight, had suffered for eleven months from pain in the side, and had been paraplegic for six weeks.

5. *Meningeal Tumor.*—New growths of various kinds may occur in connection with the spinal membranes. Sometimes a lipoma or an enchondroma is formed outside the sheath of the cord, having developed itself in the connective tissue which is usually present there. Much more frequently, one of the spaces within the sheath is the seat of the affection. According to Charcot, tumors are particularly apt to grow from the inner face of the dura mater—generally sarcomata or psammomata. In the post-mortem room I have met with one specimen of a growth in this position; it consisted of perfectly developed fibrous tissue; its upper end lay close to the foramen magnum; it extended downward for more than two inches, and pressed upon the back of the cord on the left side. In another case I found a soft, granular, reddish-gray mass, smooth and lobulated on the surface, which lay loosely attached beneath the arachnoid membrane, between it and the pia mater, in the dorsal region. It measured an inch and a quarter in length; it was made up partly of fibrous and partly of spindle-cell tissue.

6. *Meningeal Gumma.*—This affection seems to be very rare, even more so than tumor. Virchow has related a case in which the dura mater in the lower cervical region was increased to three times its normal thickness, and was bound down to the bodies of the fifth and sixth cervical vertebræ by a large quantity of firm connective tissue, which doubtless had been developed out of a syphilitic new growth. Wilks once found a hard, irregular, yellow mass, three-quarters of an inch long, lying on the right side of the cord within its sheath, and adherent to the pia mater and to the posterior roots of the spinal nerves, which last were compressed by it. In a case of Dr. Moxon's ("*Guy's Hosp. Rep.*," 1871) there were a number of brownish or blackish patches, from the size of a barleycorn to that of a pea, with soft, yellowish centres, penetrating into the substance of the cord from the pia mater. In one described by Heubner, in Ziemssen's "Cyclopediæ," a gelatinous mass extended from the floor of the fourth ventricle into the bulb, for a depth of one-twelfth of an inch; while in the cervical region the dura mater and the other membranes behind the cord were pressed together into a callous mass, which was firmly adherent both to the arch of the atlas and to the posterior columns of the cord itself.

Symptoms of Compression Paraplegia.—At the bedside we recognize and distinguish it from primary affections of the spinal cord, not so much by peculiarities in the paralysis itself as by the fact that this is accompanied, and in most cases preceded, by symptoms due to interference with the roots of the nerves coming off from the cord at or just above the level of the lesion. By Charcot these are termed "extrinsic" symptoms, while he gives the name of "intrinsic" symptoms to the loss of movement in parts lower

down, to the anæsthesia, and to the other effects of pressure upon the cord itself. Erb describes the former as belonging to a "first stage," the latter as characterizing a "second stage" of the disease.

In attributing the earlier phenomena to the circumstance that some of the spinal nerves have their roots directly involved in the lesion, one is not expressing a merely speculative opinion. They have been observed on dissection to be reddened and greatly swollen, although not showing any very serious histological change (Charcot), while in advanced cases at a late period they have been found transparent, grayish, and atrophied, with their fibres in a state of fatty degeneration.

Chief among the *extrinsic* symptoms is *pain*. And few points in clinical medicine are more important than the fact that this is referred by the patient not to the seat of mischief, but chiefly or solely to those parts to which the affected nerves are distributed. Thus, there is always danger of mistaking cases of spinal disease for various other affections; now for occipital or brachial neuralgia, then for pleurodynia, and again for gastrodynia, for colic, or for sciatica. Very often the pain appears to be fixed in a single spot, or to occupy only a very small part of the whole surface supplied by a single nerve. In other cases it shoots through all the main trunks belonging to a limb. Charcot lays stress on its burning character, and on the absence of *points douloureux* as distinguishing it from a mere neuralgia; but as I believe with the late Dr. Anstie that the *points* are to be detected only in nerves which have already been the seat of pain for a considerable length of time, I doubt whether the distinction can be maintained.

Associated with the pain there is often an extreme degree of hyperæsthesia. The patient may be unable to bear even the slightest touch without crying out, and every movement may be excessively painful. An eruption of zoster has now and then been observed in the course of some of the nerves which are most seriously involved in the disease. On the other hand, there is sometimes impairment of sensibility or actual anæsthesia over a more or less extensive part of the surface to which the affected nerves are supplied. And the muscles which receive branches from them may show tonic or clonic spasms, or become paralyzed and even atrophied, with loss of faradic contractility and an inability to respond to reflex stimuli.

It is to be observed that in speaking of these phenomena as "extrinsic symptoms" of a compression paraplegia, we are, for the sake of convenience, adopting an expression which is, in reality, incorrect. Precisely similar pains may occur in any case in which spinal nerves or their roots are involved in disease, even though the cord may to the last remain intact. Thus lateral curvature of the spine, which perhaps never causes paralysis, is frequently accompanied by a fixed pain in one or more of the ribs or intercostal spaces, which seems to be due to pressure upon nerves as they are passing through the intervertebral foramina. And in the most remarkable case of medullary sarcoma of the vertebræ which I have ever seen there was at no time any well-marked paralysis. The patient was an errand boy, aged sixteen, who, five months before his death, began to experience pain first in the loins, then in the shoulders, and then in all his limbs. His sufferings became intense and hardly bearable. The whole surface of the body was excessively tender, but especially the lower part of the back. The pain was constant, but it varied in position. There was numbness and a sensation of tingling in each hand; this was first noticed in the fingers supplied by the ulnar nerve on either side. He became wasted to the most extreme degree. The only way in which he got any ease was by lying flat on his back, with his legs drawn up. Every movement of his neck, and of any part below it, caused him severe pain. One day he became insensible and had a series of epileptiform seizures, in which he died. A mass of white medullary new

growth (possessing microscopical characters such as are now known to belong to the sarcomata) occupied the lumbar glands, and spread from them to the intervertebral discs, eating also into the lumbar vertebræ themselves. It passed up in front of the spine into the neck, where it involved part of the brachial plexus on each side. It nowhere penetrated into the spinal canal or implicated the membranes of the cord.

There seems to be no doubt that pain is more marked as an early symptom in cases of malignant disease of the spine than in those of other forms of compression paraplegia. Perhaps one ought to be contented to accept this as an example of the rule that cancerous affections in general are apt to be very painful. There are many nervous filaments ramifying in the periosteal and other tissues connected with the vertebræ, and one can easily understand their leaving the patient no peace when involved in a new growth. Cruveilhier long ago associated together under the name of *paraplégie douloureux* certain cases attended with intense lancinating pains in the nerves of the sciatic and lumbar plexuses, the muscular branches of which are paralyzed. Recent writers have remarked that a large majority of the cases in question are instances of malignant disease of the spine, involving the roots of the nerves for the lower limbs in the cauda equina, or where they lie by the side of the lower end of the lumbar enlargement of the spinal marrow. But I do not think that the reason for this has been pointed out, namely, that whereas the massive lumbar and lowest dorsal vertebræ frequently become cancerous, they are comparatively seldom affected with caries. In a series of fourteen cases of paraplegia, due to Pott's disease of the vertebræ, which I have collected from our post-mortem records at Guy's Hospital, the affection was not once in this part of the spine. There were very few other cases in which it occupied this position; and then, as it happened, there was no paralysis. Thus, in one instance the third and fourth lumbar vertebræ were found "extensively diseased," and in another the eleventh and twelfth dorsal vertebræ had entirely disappeared, so that on straightening the spine a large space was seen in front, in which the sheath of the cord was exposed. But in each case the disease had been entirely latent during life, the patient had been kept in bed for a considerable time by some other malady, and this no doubt had enabled the spinal cord to escape being compressed. In the report of the second case it is expressly noted that repeated questions failed to elicit any complaint of pain, whether in the back or elsewhere.

Hitherto I have purposely deferred mentioning pain in the back—rachialgia—as an early symptom of compression paraplegia because, although it is not infrequently present, it is far less constant than one might have expected. It may assume different forms. Certain of the spinous processes may be tender on pressure, or over-sensitive to a slight blow or jar. Or the patient may be conscious of a feeling of stiffness in moving the back or the neck, according to the seat of the mischief. Sometimes—as in a case of cancer of the cervical vertebræ placed on record by Mr. Cæsar Hawkins—there is a great increase of pain in the neck when the face is turned over on the pillow to one side or the other, and the hands are used to steady the head in every change of posture.

After the "extrinsic" symptoms have lasted for months or even for years—having been perhaps regarded as neuralgic or rheumatic—they are succeeded by others of which the spinal origin is obvious to the most uninstructed observer. These "*intrinsic*" symptoms are by no means merely results of mechanical pressure upon the cord. As far back as 1856, Gull pointed out that granule masses were present in a case of this kind. And within the last few years the state of the spinal cord in compression paraplegia has been thoroughly investigated by Michaud and other French observers. They

found that the affected part becomes reduced in size (so that sometimes it is scarcely as thick as a quill) and flattened or distorted in shape. Its tissue may be pale, but otherwise natural, to the naked eye; or it may obviously have lost its normal structure. It is either softened or (in a more advanced stage) harder than usual. Under the microscope the neuroglia is seen to be thicker and more fibrous; the nerve tubes have no longer any medullary sheaths, but their axis cylinders persist and may even be increased in size. Granule masses are abundant. The ganglion cells are swollen, vacuolated, and pigmented, or sometimes they are degenerated and broken down. In other words, there is a chronic transverse myelitis, and it extends a little way above and below the spot which is actually compressed.

The characters of the paraplegia itself are such as might be inferred from the nature of the lesion causing it, according to the principles laid down in the last section. Sometimes its development takes place very rapidly, in the course of some days, or even within a few hours—much more often it occupies several weeks. The patient finds his legs more and more heavy, especially in going up and down stairs; he becomes unable to stand; at last he cannot even move his toes while lying in bed. Subjective sensations of numbness, pins and needles, etc., often precede the motor symptoms, but, later on, loss of voluntary power over the muscles generally preponderates over loss of sensation. The upper limit of the spinal lesion is commonly well marked, especially if there is much anæsthesia. It corresponds more or less exactly with the seat of the early "extrinsic" pains and tenderness, which, indeed, often persist after paralysis has set in. A point of great importance is that, as a rule, it shows no tendency to advance upward in the cord, but Erb quotes Michaud as having observed that in some very rare cases a morbid action ascends along the lateral columns, so that, for example, the upper limbs may after a time become paralyzed, although the part of the cord which is compressed is in the thoracic region. The lower limit of the lesion cannot be defined with similar exactness, but at least one can generally say that the lumbar enlargement of the cord is healthy, the only exceptions being those cases in which it is itself the seat of the pressure. Reflex contractions in the lower limbs are usually very readily excited, much more so than under normal conditions. Leyden, indeed, remarks that in old people the sensibility of the spinal centres to stimuli from below is comparatively little marked, so that compression paraplegia is generally more easily recognized in children than in persons advanced in years. The state of the bladder is very variable; it often continues to expel its contents quite naturally for some time after the legs have begun to be paralyzed, but whenever the paraplegia is complete it almost always becomes paralyzed.

Charcot lays stress on some modifications of sensibility which, he says, although not peculiar to compression paraplegia, are observed more frequently in these cases than in other affections of the cord. One is a retardation in the transmission of sensory impressions when there is not an absolute anæsthesia; fifteen, twenty, or even thirty seconds may elapse between the application of a stimulus to the cutaneous surface and the perception of it by the patient. I shall have to discuss this symptom more fully hereafter, when I am describing "locomotor ataxy." Another is a peculiar dysæsthesia, a very painful feeling, which is excited by slightly pinching a limb or touching it with anything cold; this lasts for several minutes, and apparently no more exact description of it can be given than that it is a sort of diffused vibration bearing no relation to the nature of the impression which calls it forth. Another is an "associated sensation" which is referred symmetrically to a spot upon the opposite limb corresponding exactly with that to which the stimulus itself is applied.

The *diagnosis* of compression paraplegia may be either very easy or very

difficult. The simplest cases of all are those in which there is spinal deformity, and in determining the fact that the cord is pressed on from without one can often at once define the nature of the lesion, for the short, round bend, or the sharp angle, produced by Pott's disease is quite unlike anything else. But often the natural configuration is preserved.

Leyden says that if caries occurs in either the lumbar or the cervical region there is rarely any projection of the spinous processes, the reason being that the natural curve of those parts of the column is in the opposite direction; but when a person stoops, both the cervical and the lumbar vertebræ are normally convex backward, and I think it is not at all uncommon for some one of the spines to appear unduly prominent when disease is present. At any rate, I remember several instances in which I was in doubt as to whether the projection was or was not excessive. In such cases it is important to observe whether the vertebral column retains its flexibility. In malignant disease, if there is anything to be detected on manipulation, it is the presence of a new growth and not a mere displacement of the bones. Cruveilhier, in his hydatid case (to which I have referred at p. 429), discovered at the bend of the twelfth dorsal and first lumbar vertebræ a spot of the size of a sixpence, which felt like a depression surrounded by a bony ring, and which he took for the remains of a spina bifida. As he says, with regret, if he had rightly interpreted what he observed, and had ventured upon making a puncture, the patient might perhaps have been cured.

On the other hand, meningeal tumors and gummata are, of course, altogether beyond the reach of the fingers. A careful analysis of the symptoms affords the only chance of distinguishing cases in which such affections are present from those of disease of the vertebræ unattended with deformity. I have already noticed the peculiar severity of the pain caused by malignant growths affecting the spinal column. But just in proportion as this enables a diagnosis to be made between that particular form of compression paraplegia and the rest, do difficulties arise in distinguishing cases of caries of the vertebræ or of meningeal tumor from those of simple transverse myelitis, or of any other affection of the interior of the cord limited to a particular segment of it. In describing myelitis I have mentioned that it is often accompanied by painful sensations referred to various parts of the body, and by the peculiar "girdle feeling"—phenomena which are strictly comparable with the "extrinsic" symptoms of a compression paraplegia, and which do not necessarily differ from them even in degree. I am disposed frankly to admit that in some cases a diagnosis between the two forms of disease is impossible.

It is chiefly at an early period that a case of compression paraplegia might be mistaken for one of rachialgia or *vice versâ*. The suggestion has, indeed, been made, that, considering the frequency with which this spinal neurosis is associated with hysteria, one ought to be prepared for its being now and then accompanied by hysterical paraplegia, so as very closely to simulate a grave disease of the vertebræ. But I do not know that a case in point has ever been recorded, and it is probable that a careful examination of the patient would show a want of correspondence between the apparent upper limit of the lesion in the cord and the seat of tenderness and pain in the spinal column. When unattended with paralysis, cases of rachialgia may generally be distinguished from those of incipient disease of the bones, without any great difficulty.

Prognosis.—Most forms of compression paraplegia terminate inevitably in the death of the patients, but their duration is very variable. According to Leyden, malignant disease of the vertebræ commonly runs a course of some months, or even of more than a year; it kills, he says, by a slow cachexia

and wasting, with dropsy, or directly by interfering with the cord. In some cases, when its seat is in the upper cervical vertebræ, the fatal issue is preceded by delirium and stupor, for which no explanation can be found in the autopsy. Meningeal tumors are described by Erb as slower in their effects; some cases end in eight or ten months, others in from two to five years, others last longer still, even to a period of fifteen years. It accords well with his statement that, in one of the two instances in which I found the cord interfered with by a growth on its exterior, the duration was sixteen months, and in the other, two years, if we reckon from the time when pain in the back was first complained of, and both patients may be said to have died accidentally, the one of pneumonia, the other of suppurative inflammation of the kidneys. But in a man, aged sixty-two, who had a malignant growth extending from the vertebræ into the spinal canal, death took place within a few days of the development of paraplegia, and not more than two or three months after the first complaint of pain in the front and sides of the chest. He was so florid and healthy looking that, until the paralytic symptoms showed themselves, I thought the case was one of hypochondriasis.

Treatment.—All that can be done in cases like the last is to relieve the pain and to prevent the development of cystitis and the formation of bed sores. The application of a belladonna or opium plaster to the back, or an occasional blister, may meet the former indication, but in most cases morphia has to be given internally or subcutaneously.

It is probable that mercury and iodide of potassium are capable of curing a paraplegia which is due to syphilitic gummata pressing on, or growing into, the cord; and I have already remarked that this treatment should never be omitted in any case which can possibly be of venereal origin.

The only other variety of compression paraplegia in which recovery of power over the limbs appears to be possible, is that due to Pott's disease of the spine. Of such cases a great many do well; among six which came under Erb's observation during the year before he wrote his article on this subject, only one ended fatally, the remaining five being cured or greatly relieved. Charcot refers to two patients, who had their lower limbs completely paralyzed for eighteen months and two years respectively, and who, nevertheless, recovered perfectly. He speaks in decided terms of the value of the application of the actual cautery along both sides of the projecting spinous processes. But it seems likely that all other plans of treatment will, in future, be superseded by Sayre's method of applying a plaster-of-Paris bandage, rolled round the body, while the patient is suspended from the head, chin and armpits, by a suitable apparatus. Cod-liver oil and other tonics should, however, also be administered.

One might well have doubted whether in these cases of caries of the vertebræ, in which recovery takes place from a compression paraplegia, an inflammation of the substance of the cord occurs, such as I have described at p. 398. But an observation of Michaud, which is cited by Charcot, shows that this really is so. A woman who had regained the use of her legs for more than two years, died of hip-joint disease. The cord, at the level of the spinal affection, was found reduced to the thickness of a goose quill, its sectional area being not more than one-third of that of a healthy cord in the same region. It was of firm consistence, and gray in color; in other words, it seemed to be affected with sclerosis in an advanced stage. The microscope, however, showed that a considerable number of nerve fibres, possessing medullary sheaths, were embedded in the thick, dense fibrous material which gave it these appearances. Only one of the gray cornua remained, and this displayed but a small number of uninjured nerve cells. I have already, at p. 402, cited this case as proving the possibility of

recovering from myelitis; and from that point of view I think that its importance can hardly be exaggerated.

Diffused Affections of the Spinal Membranes.—This appears to be the most convenient place for a brief mention of certain diseases which ought, perhaps, in strictness, to have been enumerated among the conditions giving rise to a "compression paraplegia," but which differ from the rest in not being limited to any one part of the cord.

Spinal Meningeal Hemorrhage.—Except as the result of severe injuries, which come under the care of the surgeon, this is a very rare lesion. The causes which I find assigned to it are strong bodily efforts, violent emotions, the suppression of a hemorrhoidal or a menstrual flux. If a diagnosis is to be made during life, it must be based upon the sudden development of the symptoms, and upon the admixture of the effects of irritation of the cord and spinal nerves with those of compression of these structures. Certain cases in which there have been severe pains in the back and limbs, rigidity of the spine, and partial paraplegia, have been attributed to the effusion of blood into the spinal membranes; and this explanation has not been regarded as invalidated by the circumstance that recovery has slowly taken place. But it would seem that in other cases in which meningeal hemorrhage has proved fatal, so that it has been demonstrated in the post-mortem room, the symptoms have not been equally characteristic.

Acute Spinal Meningitis.—I have already had occasion to mention the fact that inflammation of the spinal membranes is not a very uncommon consequence of the formation of a deep bed sore over the sacrum. In some cases the spinal canal is actually opened by sloughing of the fibrous membrane which closes its lower end; in others, perhaps, the morbid process extends inward along the canals for the sacral nerves. At least four instances of this kind have been observed in the post-mortem room of Guy's Hospital; and probably several have been overlooked. I do not find that any definite spinal symptoms have been noted in the cases in question. Pus has generally been diffused among the nerves of the cauda equina, and beneath the spinal arachnoid, up to the base of the brain. In one case it is stated that the inflammatory products lay outside the theca vertebralis.

Other forms of acute spinal meningitis are those which accompany the various kinds of inflammation of the membranes of the brain, and which will be fully described hereafter. A "hemorrhagic spinal pachymeningitis" is occasionally found in association with a similar affection of the cerebral dura mater. Of a primary acute inflammation of the spinal membranes, limited to them, and not involving the encephalic structures, I have no knowledge.

Chronic Spinal Meningitis.—This, again, is an affection about which very little seems to me to be accurately known. Certain local forms of it, occurring chiefly in association with syphilitic gummata, or with caries of the vertebræ, have been already mentioned. But it sometimes happens that the whole of the spinal membranes are found matted together and thickened round a cord which appears to be perfectly healthy. I once made an autopsy on a case of this kind, in an old woman of seventy, who had for about a year been suffering from a slowly advancing paraplegia. So far as I could learn, she had had no spasms of the legs, nor had they been rigid; she lay helpless in her bed, but the lower limbs still retained some power of sensation, so that she could distinguish heat from cold. She complained of pains in the legs (especially in the left one) and in the lower part of the abdomen, but it did not appear that they were severe. The visceral

arachnoid, when it had been stripped off the inner surface of the dura mater, was found to be as thick as a sheet of writing paper. In the lower part of the dorsal region, the adhesion of the membranes to the cord was particularly close for about an inch and a half of its length. The thickening extended upward around the pons, and to the under surface of the cerebellum, so that the cerebro-spinal aperture was closed. I thought, at the time, that the absence of any marked irritative symptoms might, perhaps, be due to the fact that the cord itself was also extensively affected; but Dr. Frederick Taylor, under whose care the case was during life, has since told me that he could detect in it no morbid change, except that the fibrous septa entering it from the surface appeared thicker than usual.

A similar case was recorded by Gull in his well-known paper in the "*Guy's Hospital Reports*" for 1856 (Case 7). In that instance, rigid flexion of the legs was a marked symptom, and they were also affected with frequent spasms. But it is not clear to me that the affection could have been satisfactorily diagnosed from a transverse myelitis in the upper dorsal region, with exalted susceptibility (or descending lateral sclerosis) of the part of the cord below.

Some modern German writers, however, describe spinal meningitis as of frequent occurrence, and as being often recognized during life without much difficulty. It is characterized, says Erb, at first by pain and stiffness in the back, a troublesome "girdle sensation," a feeling of weight in the legs, numbness, tingling, tearing, or dragging pains in them, and cutaneous hyperæsthesia. After a time partial paraplegia develops itself, which varies in degree from day to day. Erb supposes that such fluctuations depend upon alterations in the quantity of fluid effusion within the spinal canal, or perhaps in the amount of blood in the vessels about the cord; he even suggests that gravitation of fluid into the lower part of the theca vertebralis may make the patient unable to move the legs so well when he stands up as when he is lying down. There is generally but little impairment of sensibility; and, according to Braun, the sphincters retain their functions. Erb speaks of the morbid change in the membranes as being usually but little marked; they are more or less thickened, opaque, and adherent to one another; there is an excess of fluid round the cord. Cold is said to be the chief exciting cause of this affection; it has often been observed in officers after severe fatigue and exposure during a campaign. It then commonly begins acutely with fever, passing into a chronic form secondarily. In many cases it afterward becomes complicated with myelitis, and the peripheral part of the cord beneath the pia mater sometimes undergoes sclerosis in an annular form.

How far one is justified in maintaining an anatomical diagnosis of cases of this kind is, I think, very doubtful at present. But the clinical differentiation of them appears to be somewhat important, as regards prognosis and treatment. Erb, indeed, says that a good many prove fatal by bed sores, cystitis, or exhaustion. But he admits that others recover more or less completely; and Braun declares that the prognosis is very favorable, if the acute stage, in spite of the increasing paralysis, be treated at first with local bleedings and local cold applications, and then with warm baths. Afterward, when paralytic symptoms alone remain, the patient should begin a steady course of thermal baths, the brine baths of Rehme or Nauheim being preferable, for which places the English equivalent appears to be Droitwich. Erb endorses these statements, and declares that when there is no myelitis, baths at high temperatures (98°–108° Fahr.) are well borne, and that the reputation for curing spinal paralysis in general, which is possessed by hot springs, is really derived from their success in this particular class of cases. The cold-water system is also said to be useful;

and Hitzig and Erb have found benefit from passing galvanic currents along the spine. Iodide of potassium is the chief medicine which is given; but good food, wine, and tonics are important auxiliaries in the treatment.

Spastic Paraplegia.—As I have already more than once had occasion to point out, rigidity of the paralyzed limbs is no uncommon feature in cases of segmental disease of the cord, whether primarily affecting its substance or secondary to compression of its surface. It is only when the lowest lumbar centres are destroyed that the legs are of necessity flaccid. Thus in paraplegia from angular curvature they are often spasmodically flexed, whether in a child or in an adult; sometimes, on the other hand, they are as forcibly extended. These conditions are closely related to exaggerated reflex phenomena, such as are described at p. 390; and I think it is reasonable to refer them to an exalted susceptibility of the spinal centres for the lower limbs. Sir William Gull has recorded one case of chronic spinal meningitis in which there was rigid flexion of the legs; but I do not find any evidence that this symptom is particularly common when the membranes are diseased. On the Continent, since the publication of certain observations by M. Charcot, the tendency has been to attribute the supervention of rigidity in cases of paraplegia to descending sclerosis of the lateral columns. But it is admitted that the anatomical evidence in support of this doctrine is as yet inconclusive.

In the forms of disease of which I have been speaking, the contraction of the limbs is obviously secondary to paralysis of them, for it generally does not show itself until the patient has already lost power over his legs for a considerable time. But there are other cases in which rigidity is present from the first, and, indeed, constitutes the most marked symptom, so that it may seem to be the only thing that interferes with the patient's standing or walking. Thus a "tabes dorsalis spasmodica" is described by Charcot as an independent spinal affection, and Erb gives to the same complaint the name of "spastic spinal paralysis." These writers agree in thinking that its proper lesion is in all probability a symmetrical primary sclerosis of the lateral columns—or, perhaps, of the hinder parts of them—leaving the anterior gray cornua intact. Moreover, Dr. Gee has recorded in the "St. Bartholomew's Hospital Reports," a series of cases presenting somewhat different characters, and occurring in children, his designation for them being "spastic paraplegia."

The description given by Charcot and Erb is that the lower limbs generally assume a position of rigid extension and adduction. The contraction may vary indefinitely in degree. At first it is only occasionally present, and is generally less marked when the patient is lying down than when he stands upright. Thus one of the earliest symptoms of the complaint is a peculiar gait. In walking, the foot is lifted with difficulty; the toes are scraped along the floor and catch against every inequality in the surface; the step is short and hesitating; sometimes there is a peculiar hopping movement, the body being raised upon the toes at each step. Very often the attempt to walk causes a tremor in the foot and leg, which may extend to the trunk also. The back may be arched, and the head thrown backward, or the body may be bent forward over the toes, so that there is danger of falling, especially in descending stairs. The legs feel weak and heavy and are easily tired. Sometimes, if the patient sits upon a sofa, his lower limbs stick out before him, the feet not touching the ground. In more extreme cases he is confined to bed, and is perfectly helpless. His knees are tightly pressed together and cannot be bent. Yet there is generally no impairment of sensibility, and pain may be entirely absent. The bladder and rectum perform their functions naturally. Erb says that the galvanic and faradic

contractility of the muscles is slightly lowered. On the other hand, he lays stress on the constant presence of a greatly exalted susceptibility to "tendon reflexes." Not only are the common movements induced by tension of the *ligamentum patellæ* or *tendo-Achillis* exaggerated, but movements can be excited through such tendons as those of the *tibialis anticus* and *posticus*, the *biceps femoris*, and even through various aponeurotic structures. Yet there is not necessarily any corresponding increase of the superficial reflex movements which are excited by cutaneous impressions.

The complaint is said to occur chiefly in persons between thirty and fifty years of age, and in men more often than in women. Erb speaks of it as "very frequent," Charcot as "not very common." Its causes are unknown, unless one of them is the prolonged action of cold and wet. Its development is progressive, but very slow, occupying a period of eight, ten, or fifteen years; sometimes it remains stationary at the same point for a length of time. It may never extend above the hips, or it may at length affect the lumbar and the abdominal muscles or the upper limbs. The abdomen is then prominent and hard, and it is separated from the lower part of the chest by a furrow more or less deep. The fingers are from time to time, or even permanently, clinched within the palm; there is extension of the wrist and elbow joints, with pronation of the forearm; the arms may be fixed rigidly by the side of the trunk. One lower limb is often affected before the other, and the contraction may then spread to the arm of the same side before it involves the opposite leg. Sometimes the upper limbs are the first to be attacked. Erb says that it rarely happens—and only at the latest periods of the disease—that the lower limbs are rigidly flexed, instead of being extended.

According to Charcot and Erb the only real difficulty in *diagnosis* is from disseminated sclerosis in that form in which the symptoms of affection of the higher centres happen to be wanting. Charcot adds that in one of the cases with which he used to illustrate his lecture on "spasmodic tabes" scattered patches of sclerosis were found, at the autopsy, as high as the crura cerebri. The *prognosis* is said to be favorable, in so far as the complaint has no tendency to destroy life, except through the supervention of phthisis or some other intercurrent disease. As to *treatment*, Erb has seen two cases almost cured, and several much relieved by galvanic currents applied to the spine. He also speaks favorably of the "cold-water cure," and of the gaseous saline baths ("Sool baths") of Rehme and Nauheim. He has found the nitrate of silver sometimes useful as an internal medicine, and Charcot recommends the bromides. Strychnia is likely to do harm rather than good.

Dr. Gee's cases of "spastic paraplegia" occurred, all of them, in children, and the complaint was either congenital or began in early infancy. The legs were always affected and sometimes the arms. The contractions corresponded in character very closely with Erb's and Charcot's descriptions. Dr. Gee lays stress on the fact that handling the limbs increased the rigidity. Chloroform relaxed it in all cases but one; in that instance the muscles of one calf were a little wasted, the disease having lasted eleven years. To one patient the juice of conium was given, to another the extract of belladonna, but without any good results.

Now, on looking through the reports of cases that have been under observation at Guy's Hospital during the last few years, I fail to find any that answer well to the accounts given by these writers. There have been eight or nine instances in which a contraction of the lower limbs has been apparently the primary disease, but they have occurred in patients of all ages; and the knees and thighs have, without exception, been drawn up in a state of flexure instead of being stretched out. Any attempt to straighten

the limbs has generally been attended with great suffering; but in one case —that of a man aged thirty-three, under Dr. Wilks's care—the suspension of a weight of fourteen pounds to the right foot led to a marked diminution of the pain; the limb was bent, so that the heel almost touched the buttock, and it could not be put straight, even when chloroform had been inhaled. But the point to which I am most anxious to draw attention is that the extract of *Calabar bean* has, in three or four instances, appeared to remove the rigidity altogether.

The first case in which I observed this satisfactory result was that of a boy, aged six, who was admitted on August 19th, 1874. He had been well until three weeks previously; he was then found crying with pain, and it was thought that he must have had an injury, of which, however, there was no other evidence. He lay on his right side, with his legs drawn up, and unable to move them. They could be forcibly extended, but then became rigid. The expression of his face was indicative of pain, and he had complained of pain in the back of the head and over the spine. Sensation was unimpaired, but the fæces and urine were often passed into the bed. He could close his hands, but with a very feeble grasp, and sometimes they were noticed to be stiff. The diagnosis was chronic meningitis, and hydrarg. c. creta, iodide of potassium, bromide of potassium, and other medicines were given without any result up to the 8th of October. At that time he began to take the extract of physostigma in doses of one-sixth of a grain, gradually increased to half a grain, three times daily. Improvement quickly set in; the limbs became less rigid, and he regained the power of moving them. On November 21st the medicine was changed, cod-liver oil and steel wine being substituted for the Calabar bean. On January 9th, 1875, he left the hospital, being then able to walk fairly well without assistance.

In 1876 I was asked to see a boy, aged fourteen, who was under Mr. Cooper Forster's care, with contraction of the thighs and legs, which had been coming on for six months. He appeared to suffer intensely from pains in the affected limbs, and screamed when they were touched or when he made any movement, so that he allowed no rest to the other patients. The legs and thighs were much wasted. Remembering the former case, I prescribed the Calabar bean in doses at first moderate, but which were quickly increased. The symptoms at once began to subside. He was after a time transferred to my charge, and left the hospital cured.

On October 31st, 1876, a woman, aged forty-one, was admitted under my care with a spinal affection which had come on suddenly two months before. She first complained of pains in the leg muscles and then of stiffness in the joints. This was followed by great weakness and wasting of the lower limbs; and then they slowly began to contract. She lay in bed with her legs strongly flexed; when they were forcibly straightened it gave her great pain. Faradic contractility was found to be much diminished, and she had partial loss of sensation; she could not pass her water. She took at first half a grain of extract of physostigma three times daily; after a week this dose was doubled, and four days later it was trebled. By this time she could already extend her legs to some extent, of her own accord. The medicine was continued for a month and then some tincture of iron was prescribed. By December 23d she could straighten her lower limbs perfectly well. In the month of February, 1877, she became able to stand and to walk without help, and on March 1st she was discharged cured.

In 1878 a sailor, aged twenty-eight, came into the clinical ward under my care on May 29th. Fourteen months previously he had been in a river in South America loading timber, and had repeatedly got wet through and allowed his clothes to dry on him; he said he was well used to getting

wet with salt water but not with fresh water. Four or five days after he left off this work, he one morning, on waking, found that his left leg was stiff and painful in the ham. As the rigidity gradually got worse, he went into hospital at New Orleans, and after a time it passed off. He returned to work, but two months later, the weather having been bad, he again began to suffer from stiffness and pain in the left leg. Presently the right one also was attacked. They both became wéak, and for six weeks before admission he could not walk without a stick. At times he had short attacks of convulsive twitchings in the left leg. I found the thigh muscles decidedly rigid on both sides, especially the left. After leaving him for a few days without treatment, I prescribed the extract of Calabar bean in a dose of a quarter of a grain, afterward increased to one grain. Improvement was not at first very striking, but at the end of thirty-four days there was hardly any rigidity left.

The only case that I know of in which the extract of physostigma produced any unpleasant effects is one recorded in vol. xviii of the "*Guy's Hosp. Rep.*" A boy, aged ten, had been suffering for some months with paraplegia before rigidity of the legs set in. A quarter of a grain of the extract was ordered, and after a week it was increased to half a grain three times daily. By mistake he took a grain at one dose, and an hour afterward he was blue in the face but perfectly conscious, perspiring profusely, a clear froth coming from the mouth, the pupils of natural size, the hands cold, numb, and almost powerless. He had an emetic, and in three hours he was as well as before.

The last case seems to me to have been one of myelitis, apart from the tonic spasm, which was a mere complication; but the other cases just related seem to me to prove that there is a spinal affection, of which the principal symptom is a rigidly contracted state of the legs, but which yet fails to correspond with the rather narrow definitions of Charcot and of Erb already quoted. And I would propose that for the present, at any rate, they should all be placed in a single category under the name of *spastic paraplegia*. Whether a sclerosis of the lateral column occurs in any of these cases, or even in the whole group, may be left doubtful. The fact that recovery takes place under medical treatment is perhaps evidence to the contrary, although not amounting to proof. What weighs more with me is the circumstance that there is a wide class of neuroses, of which I shall hereafter have to speak—all of which are attended with spasms of one kind or another—while none of them are as yet traceable to anatomical lesions. So long as they are regarded as due to functional disturbance of the nervous centres, I should be inclined to associate spastic paraplegia with them, so far as its pathology is concerned.

SPINAL PARALYSIS ATTENDED WITH WASTING OF MUSCLES.

Acute Atrophic Paralysis—IN INFANTS—IN ADULTS—ITS COURSE, SEQUELÆ, ANATOMY, AND TREATMENT.
Chronic Diffused Atrophic Paralysis—ITS COURSE AND ANATOMY.
Progressive Muscular Atrophy—SYMPTOMS—TERMINATION—TREATMENT.
Bulbar Paralysis—PROGRESSIVE MUSCULAR ATROPHY IN CHILDREN.
Lead Palsy—DISTRIBUTION—TREMORS—TREATMENT.
Pseudo-Hypertrophic Paralysis.

ACUTE ATROPHIC PARALYSIS.—In the last century this affection was described by Underwood in his celebrated treatise on the "Diseases of Children," and Jacob von Heine published a monograph on it in 1840. Both this writer and afterward Duchenne, of Boulogne, expressed the opinion that the cause of infantile paralysis was a lesion of the spinal cord. But as autopsies failed to reveal any morbid change, MM. Rilliet and Barthez proposed the name of "paralysie essentielle." Since 1863, however, more refined histological methods have shown that the anterior cornua of the cord are diseased. Accordingly, German writers now designate the affection "poliomyelitis anterior acuta." We cannot employ the old name of "infantile paralysis," because it is now ascertained that the same form of disease occasionally occurs in adults, but since wasting of the affected muscles is one of the principal symptoms, we may call it *acute atrophic paralysis.*

A child who is about to suffer from acute atrophic paralysis, generally at first falls ill with some febrile disturbance, oppression, or drowsiness. Sometimes, however, the earliest symptom is an epileptiform convulsion, or there may be spasmodic twitchings of the face or limbs, grinding of the teeth, and rolling of the eyes. The fever usually lasts a day or two, but it may pass off in a few hours, or it may continue for a week or longer. The convulsions may be repeated during twenty-four or forty-eight hours. If the child is old enough, he may complain of pain in the back and limbs.

When the child seems to be getting better, and is being washed or dressed—or perhaps when it first attempts to stand—the mother or the nurse finds, to her horror, that one or more of the limbs is powerless. It may have gone to sleep at night with full use of its arms and legs, and in the morning one or more of them may hang flaccid and motionless. The affection may be a *monoplegia*, the paralyzed limb being generally a leg (Volkmann says the right leg), but sometimes an arm; or it may be a paraplegia, or a hemiplegia, or a crossed paralysis of one arm and the opposite leg. Not infrequently there is loss of power in all four limbs, and even of the trunk, and sometimes they are attacked in succession at intervals of a few hours.

Occasionally the initial fever is very slight; it may, perhaps, be absent, and the paralysis be the first symptom that the child is unwell. But, as might be anticipated, there is no thermometric evidence of this.

It is characteristic of acute atrophic paralysis that the affected parts are completely relaxed. No reflex movements can be elicited, whether by stimulations of the skin or of the tendons. On testing the muscles with faradic currents, one finds that their contractility becomes markedly diminished

within the first four or five days, and that it is entirely extinguished at the end of a week or a fortnight, but the susceptibility to galvanic currents is increased. In other words, "the reaction of degeneration" (see p. 350) is present. Rapid and early wasting of the muscles takes place. Until recently they were said to undergo fatty degeneration, but in reality the histological changes in them are of a different kind, corresponding exactly with those which I have already described at p. 351 as following some lesions of the nerve trunks. The surface of the paralyzed limbs is cold, and they are pale or of a livid bluish tint; the volume of the arterial pulse in them is said by Volkmann to be diminished, and Charcot states that after death the main blood vessels are found remarkably reduced in size.

As a rule, no impairment of sensation can be detected; the child cries lustily as soon as the poles of a galvanic battery are applied. The functions of the bladder and rectum are performed normally or nearly so, and bed sores never appear.

Unlike almost every other form of paralysis, that now under consideration shows no tendency whatever to a slow and progressive advance. The limbs are often attacked one after another, but the loss of power in each of them, although not absolutely sudden, becomes complete within a few hours. Erb says that the whole development of the disease, in successive outbreaks, may be protracted over a week; it generally occupies much less time.

In fact, no long period elapses before recovery begins to take place; sometimes this is observed within a few days, more often in the course of two or three weeks. Even muscles which have to some extent undergone wasting, regain their size and strength. If the arms and the upper part of the trunk are affected, they are particularly likely to get quickly well. In the course of a few months the disease may entirely disappear, especially when it was all along confined to a single limb. Some writers have disputed the claim of such a "temporary paralysis" to be classed with those forms in which there is permanent loss of power; but I agree with Volkmann in thinking that it is the same disease. It is, however, very exceptional. Almost always the recovery is incomplete. It goes on for a month or eight weeks, so that the parents of the child cherish the most pleasing hopes; but after this the progress becomes slower, and at the end of six or nine months it ceases forever.

The condition of the little patient at this time is very variable. One limb or two may be powerless and shrunken throughout, or the affection may have become limited to certain segments of an arm or of a leg. As a rule, the distal parts are more apt to suffer than those nearer the trunk. Volkmann, however, speaks of one patient as having a muscular forearm attached to a humerus like a stick, and Erb says that the deltoid sometimes suffers alone. Certain muscles exhibit a peculiar independence, now by remaining paralyzed when those near them get well, now by recovering while the rest are undergoing atrophy. Erb mentions that this is commonly observed in the *supinator longus* of the forearm, the *tensor fasciæ latæ* and *sartorius* of the thigh, and the *tibialis anticus* of the leg. When a muscle wastes, the destruction of its fibres is not necessarily made manifest by a corresponding diminution of its mass, for interstitial development of adipose tissue often takes place, which may entirely conceal it. Erb says that this is particularly apt to occur in young children, and that a muscle may actually appear bigger than in the opposite healthy limb, although it scarcely contains any contractile elements.

Another peculiarity of parts affected with this disease is, that they do not grow at the natural rate. Hence a paralyzed limb is commonly shortened to the extent of an inch or an inch and a half, and sometimes by as much as eight inches. Even the corresponding half of the pelvis may remain unde-

veloped. The bones are thinner and more spongy and their processes smaller than on the healthy side. Remembering Hilton's observations with regard to the arrest of growth in the feet of patients suffering from disuse of the hip or knee joint, one might be disposed to refer all these conditions to disuse, but Volkmann says that they occur even when the paralysis is very partial, so that the child halts very little and is on its legs all day. He has seen a persistent failure of development in four or five cases of "temporary paralysis," in which all the muscles recovered.

But the most important of the remote effects of the acute atrophic paralysis of children, are the contractions and deformities which are so commonly observed. I remember, twenty years ago, hearing Dr. Wilks insist on the fact that club foot, when not congenital, is the result of paralysis, and not primarily a spasmodic affection; and this view, which was opposed by "orthopædic" surgeons, is now generally accepted by pathologists. But it was reserved for Volkmann to show how the deformities are produced. Until recently, the explanation which was accepted was that contraction was generally the result of the unopposed action of the antagonists of the muscles paralyzed. But Hüter and Volkmann have shown that when there is loss of power in several groups of muscles, it not infrequently happens that those on which the paralysis is most marked lie *within* the open angle formed by the displacement of the bones at a joint. For example, a "genu recurvatum" of which the concavity is directed forward, may appear when the *quadriceps extensor* is powerless, or a "talipes equino varus," when no contractions can be obtained by galvanizing the calf of the leg. Volkmann explains these facts by showing that they are in part due to the influence of gravitation; thus the weight of the foot causes the toe to point downward, when the limb hangs in the air. But another important factor is the gradual strain on a joint, in efforts made by the patient to employ the weight of the body as a propelling force. Volkmann points out that when a person whose thigh muscles are paralyzed learns to walk without crutches he swings the trunk forward so as to extend the knee as fully as possible. The result is that after a time the ligaments behind the joint yield, and a genu recurvatum is produced. The articulations in such paralyzed limbs often become exceedingly loose. Lastly, a subordinate element in the causation of deformities is the contraction of the new connective tissue developed in the interstices of the wasted muscles.

Acute atrophic paralysis in an adult develops itself in most respects as in infants. Meyer, of Berlin, is said to have been the first who, in 1868, recorded cases of this kind: Duchenne, Erb, and many others have since written upon the subject. The disease seems never to set in with epileptiform convulsions; but there is fever of greater or less intensity, with headache, drowsiness, and even delirium. For a few hours, or after a single night, or at the end of a few days, one or more of the limbs becomes paralyzed; sometimes there is a transitory failure of the bladder; there is not the slightest impairment of sensibility, the muscles are perfectly relaxed and their susceptibility to reflex stimuli is lost. Recovery may either be complete or partial; in the latter case the affected parts become contracted and deformed, but not to the same extent as at an early period of life. Of course, there cannot afterward be any difference in the length of the limbs on the two sides of the body.

Histology.—Within the last few years the morbid anatomy of the disease has been very carefully studied, and probably all the most essential points have been ascertained. Cornil, in 1863, was the first to record an autopsy in which changes were found in the cord; but the body was that of a woman who had been paralyzed for nearly half a century. So far as is known this affection never destroys life directly. Erb, indeed, suggests that death

perhaps sometimes occurs before any special symptoms have appeared to indicate the real nature of the case; and therefore that a minute examination of the cord should be made whenever a child succumbs to convulsions or to febrile symptoms of unknown origin. The earliest post-mortem examination which has hitherto been made was by Royer and Damaschino, in the case of a boy who died of scarlet fever within two months of the onset of the paralysis; a few others have been at various periods up to twenty months. The results are summed up by Erb as follows: To the naked eye there is generally no perceptible alteration. Sometimes the cord seems rather tough, and its antero-lateral columns a little shrunken at the level of the lumbar or of the cervical enlargement. Its structure may be slightly blurred. The fore part of the gray matter may be whitish or pink and softened, or even reduced in size, the corresponding motor nerve roots may be atrophic, gray, and translucent. The microscope reveals an extensive area of morbid change, perhaps even one inch or more in length, in this region. It is sometimes confined to one anterior gray cornu, sometimes it involves both of them, according to the distribution of the paralytic symptoms during life. Granule masses are generally present in abundance; there is nuclear overgrowth, the vessels are dilated and full of blood, in other words, there is an *inflammatory softening*. But the most striking change is a more or less complete disappearance of the large multipolar nerve cells, many of them that still remain being in various stages of atrophy. The nerve fibres also have undergone destruction, even to their axis cylinders. At the periphery of the affected area the nuclei are often massed together in large numbers, so as to form a kind of capsule, but slight diffused lesions are discoverable throughout the gray matter of a large part of the cord, generally in the whole length of the dorsal region. In the antero-lateral white column there is but little to be seen; a slight degree of sclerosis sometimes, thickening of the trabeculæ, atrophy of a few of the nerve fibres, granule masses scattered sparingly. The motor roots display all the appearances of "degenerative atrophy."

Where many years have elapsed since the onset of the paralysis, the appearances are far more marked. The shrinking and atrophy of the anterior part of the cord is now evident on the cut surface, especially when the affection is one-sided, in which case the symmetry of the two halves is lost. The antero-lateral column may be obviously gray in tint, and translucent as compared with the posterior column. Under the microscope the diseased area in the lumbar or the cervical enlargement is seen to consist almost entirely of a delicate connective tissue, containing an immense number of corpora amylacea, but now no granule masses. No normal ganglion cells or nerve fibres are at this stage to be seen; any remnants of cells are shrunken and pigmented.

That a local affection of the spinal gray matter should set up such violent fever as we have seen to be often present in this disease is surprising: but we seem to have no alternative but to regard it as symptomatic. There may, indeed, be a question as to whether the myelitis is not at first diffused, and whether it does not clear up and subside throughout the greater part of the cord, while one particular region, to which it becomes limited, undergoes entire destruction.

There have been many discussions as to the starting point of the affection, whether it is in the multipolar cells or in the neuroglia. Charcot insists that the fact of its strict limitation to certain regions of the cord is strongly in favor of the former view; he also says that at some points the cells may be found diseased while the connective tissue remains all but normal. Erb thinks that perhaps both tissues become simultaneously diseased.

Ætiology.—With regard to the *causes* of acute atrophic paralysis very little

is as yet known. It is far more common in children between one and four years of age than in those who are older, but it not rarely develops itself in infants from six to twelve months old, and Duchenne has recorded cases occurring at the ages of twelve days and one month respectively. The two sexes appear to be equally liable to it. Whether teething plays any part in its ætiology is very doubtful. Wharton Sinkler, an American observer, found that it arose very much more often between the months of May and September than during the rest of the year, the proportion being as forty-seven cases to ten. The so-called neuropathic family tendency seems seldom to be present. Duchenne says that he has never seen this affection in two children belonging to the same family, and the few cases on the other side that have been published may probably be mere coincidences. Those who are attacked by it are commonly neither rachitic nor scrofulous, but robust and healthy. Sometimes it is directly set up by a chill; at least Erb says that this has been demonstrated beyond dispute. Sometimes it arises during convalescence from some acute disease, such as scarlet fever, mild smallpox, typhoid fever, or ague. This may also be the case in adults. Some years ago I saw a marked case in a young man, aged twenty-two, who was just getting better of pleurisy. In women it may occur during the puerperal periods.

The *diagnosis* is seldom difficult if one keeps in mind the clinical history and features of the disease. One must, of course, remember that other forms of paralysis may occur in childhood; perhaps that which is most likely to cause a mistake is a peripheral paralysis of the brachial plexus from sleeping on the side, or from pressure by a band fastened tightly around the arm. In the case of an infant brought by a person ignorant of the circumstances, it may be impossible to come to a positive conclusion.

The *prognosis* must always be guarded; complete recovery is rare. When a child first comes under observation at an advanced stage, with the muscles already wasted, one is apt to think that the issue would have been better had *treatment* been earlier. But if it is placed under treatment from the commencement, there is great difficulty in saying how far any improvement that occurs is attributable to the remedial measures that may have been adopted. The application of galvanism to the spine seems to be the most important thing. The poles must be large. Erb advises that one should be placed over the affected part of the cord, the other over the front of the body; a current of moderate strength may be passed for one or two minutes at a time. Afterward the paralyzed muscle may be galvanized in turn. Faradization may also sometimes be useful, if the muscles have not lost their susceptibility to induced currents. Even when several years have elapsed, one must never hastily conclude that the electrical treatment will necessarily be fruitless. It now and then happens that a very few applications produce extraordinary effects, and very often by persevering for several weeks, or even for months, one at length attains considerable success. In one case—of which I unfortunately have no notes—the internal administration of strychnia was, beyond dispute, the means of restoring to a child the power of standing. Shampooing and friction with stimulating liniments may sometimes be of service. Nourishing food, cod-liver oil, and tonics are matters of course. For the avoidance of the different forms of contracture, recourse must be had to mechanical apparatus. Volkmann says that drawing up of the limb may be prevented by fastening the feet every night on a splint with a flannel roller and carrying a strip of plaster across to the leg.

Chronic Diffused Atrophic Paralysis.—Within the last few years writers on spinal diseases have begun to recognize as a distinct affection a form of paralysis which was first described by Duchenne in 1849 and 1853,

under the name of "*paralysie générale spinale antérieure subaigue.*" He suggested that it was probably due to an atrophy of the cells of the anterior gray cornua; and as recent observations have shown that there is a chronic inflammatory change in this part of the cord, Erb proposes to call the disease "*poliomyelitis anterior subacuta et chronica.*" It seems to me that the most suitable designation is "chronic diffused atrophic paralysis;" this at once distinguishes it from the "*acute* atrophic paralysis" already described, and also from the *limited* forms of paralysis which are usually known by the title of "progressive muscular atrophy."

Course.—It generally begins in the lower limbs. The patient first experiences a sense of weakness in one or both of his legs, especially in going up stairs, or if he attempts to walk far. At the end of a few days, or in the course of some weeks, this develops itself into actual paralysis, so that he is obliged to keep his bed. Before long the upper limbs are affected, especially the fingers and wrists. The muscles of the trunk also become powerless, so that he cannot sit up, and has difficulty in coughing or sneezing, and even in passing his fæces.

The paralyzed muscles are absolutely lax and flaccid. No reflex movements can be excited in them by irritation either of the skin or of the tendons. They very rapidly waste, so that with a tape one can follow the loss of substance in the calves or in the thighs from week to week, until the bones seem to be covered only by the integuments. When electrical tests are applied to them they are found to present the "reaction of degeneration" (p. 350). Duchenne long ago pointed out that their susceptibility to faradic currents is extinguished. Erb and others have shown that they, nevertheless, at first react well to galvanic currents, but with the peculiarity that A.C.C. is more marked than C.C.C. (see p. 345), and that the contractions are slow and of a tonic character.

So far there is nothing in the symptoms of chronic diffused atrophic paralysis which is distinctive of this affection rather than of a myelitis spreading upward through the whole substance of the cord from its lower end (see p. 411). What shows that the lesion is limited to the anterior part of it is the absence of anæsthesia, the retention of power over the bladder and rectum, and the fact that there is no tendency to the formation of bed sores. Erb, indeed, says that pains in the back and slight paræsthesia in the limbs are not uncommonly present, and may even be complained of before the paralysis has developed itself; but on testing the cutaneous sensibility one finds it absolutely perfect, or if blunted at all it is so only in the smallest possible degree. At the commencement there may be a little febrile disturbance, with headache and digestive disorder, but these quickly pass off and the patient afterward feels quite well, and eats, drinks, and sleeps as usual.

The further progress of the disease varies in different cases. Generally it remains for a certain length of time stationary; that is, it ceases to spread upward, or to extend to fresh sets of muscles, although those already affected go on wasting. Erb says that sometimes at this period the joints become slightly contracted. But at last, after several weeks, a gradual improvement begins to be observed; the upper limbs regain some of their movements, and afterward the lower limbs some of theirs. All this takes place very slowly; months pass before the patient can feed himself, or write, or walk. Sometimes, however, he ultimately becomes as muscular and as active as before, but sometimes some particular muscles remain wasted for the rest of his life; according to Erb, this is especially apt to be the case with the peronei. As recovery goes on, the electric contractility of the muscles gradually reappears.

On the other hand, there are cases in which no change for the better

occurs, but in which the paralysis extends upward to affect the face and the tongue and to interfere with deglutition and with respiration. In this way the disease may terminate fatally from one to four years after its commencement. It does not appear that there are any special indications which might enable a correct prognosis to be given at an early period of the case.

Histology.—Up to the present time, only two autopsies appear to have been made in which the cord was examined by the modern methods. One was recorded by MM. Cornil and Lépine, the other by Dr. Webber.* In each instance the multipolar nerve cells in the anterior gray cornua had undergone a more or less complete destruction; there were evidences of inflammatory changes in the presence of compound granule masses, the proliferation of the cells of the neuroglia, the accumulation of nuclei round the blood vessels, and the like. In the antero-lateral columns there was a certain degree of sclerosis or degeneration of nerve fibres. The motor nerve roots and the muscles were in a state of extreme atrophy. In other words, the morbid anatomy of the disease appears to correspond exactly with what might have been anticipated on theoretical grounds.

With regard to the *ætiology* of chronic diffused atrophic paralysis, nothing is as yet known. It is especially apt to occur in persons between the ages of thirty and fifty, but in this it resembles several other spinal affections. It has sometimes appeared to be the result of cold or of an injury to the back, sometimes to be attributable to intemperance or to sexual excesses.

The *diagnosis* is, in well-marked cases, easy. The only affection that closely resembles it is the diffused form of paralysis, which sometimes occurs in persons poisoned by lead. There may, indeed, be a question whether this is not dependent on the very same change in the anterior gray cornua of the cord; whether, in fact, we ought not rather to enumerate chronic plumbism among the causes of the disease now under consideration. I believe that the only distinctions are the presence of the characteristic line upon the gums, and of other effects of the metal, such as colic, sallowness of complexion, and gout. Erb says that he has seen two cases of paralysis limited to the upper limbs, which, in the absence of all evidence of lead poisoning, he was obliged to regard as instances of a primary "polio-myelitis anterior," limited to the cervical part of the cord.

The *treatment* of the disease appears to be that of chronic myelitis in general; the chief remedies being iodide of potassium, ergot, and the application of galvanism to the spine.

Progressive Muscular Atrophy.—In 1850, a French physician, Aran, described, under the name of "*atrophie musculaire progressif*," an affection which he believed to have been altogether overlooked by previous writers, and of which, indeed, only a few instances had before been recorded, chiefly by Abercrombie, Sir Charles Bell, and Romberg. Three years later Cruveilhier wrote upon the same subject. Aran's designation has ceased to be strictly appropriate; but it is still almost always used, and I think it is decidedly better than those that have been proposed instead, among which may be mentioned "wasting palsy," "Cruveilhier's paralysis," and "amyotrophie."

Chief among the symptoms is, in fact, muscular *atrophy*, and this is the more conspicuous because it often affects a single fleshy mass, or even an isolated muscle or a part of a muscle, while others close by remain as well nourished as ever. In the large majority of instances, the upper limbs are first attacked. Aran, whose statement is generally taken as fairly representing the truth, found that this was the case in nine among eleven

* ["*Trans. American Neurological Assoc.*," vol. i, p. 55.—Ed.]

patients, and that in seven of them the right arm was that in which the disease began. The proportion cannot yet be fixed with absolute accuracy, because it is still doubtful whether all the cases in which the wasting is first observed in the legs, really belong to the same category. Thus, whereas Duchenne says that he met with only two such cases out of 159, Hammond, of New York, speaks of having had eight in a total of twenty-five. Duchenne himself saw one patient in whom the disease began in the *sacro-lumbales*, and one in whom the *pectorales*, the *trapezii*, and the *latissimi dorsi* disappeared before the arms became involved. The apparently capricious way in which the atrophy is sometimes distributed, is shown by another of this writer's cases, in which, even at an advanced stage, no muscles of the upper limbs, except the *supinatores longi*, were attacked. Conversely, Trousseau mentions a patient of Bretonneau's, an old lady, who retained power over none of her muscles save those of the right index finger. She could not speak, but with her finger she picked out of a heap of letters those which she required to form words and sentences, and in this way she made her will.

As a rule, however, progressive muscular atrophy begins in the muscles of one particular region, namely, in the small muscles of the hand, and especially in those which constitute the ball of the thumb. Before passing to the forearm, it commonly spreads from the thenar to the hypothenar muscles, and also to the *lumbricales* and *interossei*. Or, sometimes, it appears first of all in the *deltoid* and other scapular muscles. A curious circumstance is that when it attacks the *trapezius* it always leaves the anterior part of the muscle uninjured; and another peculiarity is that the *triceps* usually escapes, even when all the other fleshy masses in the upper limb have wasted away. The patient's arm then hangs helplessly by his side, he cannot raise it by any muscular effort; if he wishes to lay hold of anything he has to swing it forward with a jerk, until his fingers are brought into contact with it. His hand often presents the "griffin" or "bird-claw" deformity, described at p. 350. His power of prehension is greatly diminished, especially when the *opponens* is atrophied. Instead of being able to grasp an object between the fingers and the thumb, he has to fix it as well as he can within the hook-like concavity formed by the fingers alone.

Whatever may be the starting point of the disease, it in most cases soon afterward shows itself in the corresponding muscles on the other side of the body. But sometimes the symmetry of its distribution is very incomplete; so that, for instance, one forearm and the opposite upper arm may be the regions principally affected by it. Having spread more or less generally over the upper limbs, it at length passes to the neck and to the trunk; the lower limbs almost always remain intact until a very late period.

The loss of substance in the muscles is commonly conspicuously visible through the integuments. The ball of the thumb is flat, and there is an obvious hollowing of the metacarpal spaces, especially of that which lies between the thumb and the forefinger; or the shoulder has lost its fullness, the bones forming sharp, angular prominences; or the normal rounded curves of the forearm are replaced by shallow depressions. But in stout persons the wasting is not always thus easily to be detected by the eye. It is not merely concealed by the overlying fat; there is often an actual new growth of adipose tissue in the interstices of the affected muscles; and as at the same time a tough fibrous material is present, the part sometimes feels quite as fleshy and firm as in the natural state. Thus Trousseau relates a case in which he could not account for a loss of power in the arms and elsewhere, until Duchenne pointed out to him what he had altogether overlooked, that

the deltoid and several other muscles had undergone atrophy. The application of a faradic current may at once clear up all doubt, if the disease is in a sufficiently advanced stage; or a little portion of muscular tissue may be removed for microscopical examination by Middeldorpff's "harpoon," or by Duchenne's "emporte-pièce."

The functional weakness in the affected muscles is increased by fatigue, and, to a remarkable extent, by cold. More than one patient has told me that when warm in bed, after a night's rest, his hands felt as strong as ever. During the day the hands and fingers often look pale and blue, and they are cold to the touch. Jaccoud and others have taken thermometric observations, from which it appears that the temperature is notably lower on the side on which the muscles are the more wasted; it is said that a difference can sometimes be detected, even between the two axillæ. Sometimes there is an excessive secretion of sweat. All these symptoms are referred by Leyden to disturbance of the vaso-motor nerves. In several instances a contracted state of the pupils is recorded as having been noticed.

A symptom on which some writers have laid great stress is the occurrence of transient "fibrillary tremors," or slight quivering movements limited to particular fasciculi of the muscles involved in the disease. They may either be spontaneous, occurring when the part is perfectly at rest, or one may elicit them by giving the skin a smart tap with the finger. All observers now admit that, so far from being pathognomonic of any one affection, they may be seen under various circumstances, and even in healthy persons, if carefully looked for. Those which follow taps upon the surface of the muscle are particularly well marked in patients who are emaciated by pulmonary phthisis or any similar disease. But it would appear that in progressive muscular atrophy the spontaneous fibrillary contractions sometimes far exceed those which occur under other conditions, producing perceptible movements of the limbs and attracting the notice of the patient, although they are attended with no pain. It is said, too, that they sometimes afford the earliest indication of the extension of the disease to a fresh group of muscles. Duchenne, however, found them absent in about a fifth of the cases that came under his observation.

The cutaneous sensibility is altogether intact; there is no pain in the affected parts; nor are any subjective sensations of numbness experienced. This, at least, is what Charcot says of that form of the disease which he terms "protopathic," and which alone is under consideration now. Leyden and other writers, while pointing out that impairment of sensation is comparatively little marked in proportion to the muscular wasting, have yet described a partial anæsthesia as of rather frequent occurrence. But they doubtless included certain other affections which Charcot has recently shown to be both clinically and pathologically distinct, and of which I shall give some account further on.

It is not in every case that progressive muscular atrophy manifests a tendency to spread from one group of muscles to another. On the contrary, it may remain for many years limited to those muscles which are first attacked by it. But, as we have seen, it often extends from the limbs to the trunk; and then it is very apt to destroy life by interfering with the respiratory movements. One should always carefully observe the condition of the intercostals, and also that of the diaphragm. If the former muscles fail to contract, the upper two-thirds of the chest cease to expand, and the act of expiration is proportionately shortened. If the latter is atrophied, the epigastric and hypochondriac regions are drawn in during inspiration. In either case the patient has frequently to stop between his words, to take breath. He has difficulty in crying out and in singing, and the attempt to cough or to sneeze causes him great distress. An attack of

bronchitis is almost inevitably fatal, so that the greatest care must be taken to protect him from exposure to cold. Not infrequently the intercostal muscles and the diaphragm fail simultaneously; and then, as the disease advances, suffocation becomes inevitable. Sometimes, on the other hand, the disease proves fatal by spreading to the muscles of the throat and of the pharynx; the symptoms which are then observed will be presently described under the head of "progressive bulbar paralysis." In not a few instances death occurs by phthisis or pneumonia, or an intercurrent attack of enteric fever. Its duration is from two or three to as many as twenty years.

With regard to the *causes* of progressive muscular atrophy, there is still much uncertainty. It occurs chiefly in young adults; according to Leyden, the mean age is about thirty-two years. It is far more common in males than in females. This fact, however, may be regarded from two points of view. Charcot and many other writers think that the disease is frequently transmitted by inheritance, and especially in the male line. That such transmission sometimes occurs is, I think, indisputable; but it would seem that the majority of the supposed examples have really belonged to a different affection, which will presently be described under the name of pseudo-hypertrophic paralysis. On the other hand, over-use of the muscles, leading to exhaustion (of them or of the corresponding spinal centres), is often made chargeable with the disease, as when it occurs in a bank clerk, or in a cobbler, or in a saddler; and this would naturally be more common in men, although washerwomen are mentioned as furnishing examples of it among persons of the weaker sex. But here, again, confusion may have arisen from the admixture of cases of "writers' cramp" with those of the affection now under consideration, since until recently the two diseases were often included under the vague designation of "scriveners' palsy," at present fallen into disuse. I have often seen progressive muscular atrophy in persons who had in no way over-fatigued their muscles, and I doubt whether the relation between the supposed cause and its effect is not merely one of accidental coincidence. Dr. William Roberts and others speak of cold and of blows on the back of the neck as sometimes setting up the disease; but Charcot seems to be right in thinking that the cases in point which have hitherto been recorded have not really been instances of progressive muscular atrophy in a strict sense of the term.

The *pathology* of this affection has only lately been placed on a satisfactory footing. Both Aran and Cruveilhier originally supposed that it was primarily a disease of the muscles, but the latter modified his opinions when, in 1853, he found, in the celebrated case of a rope dancer named Lecomte, that the anterior roots of the spinal nerves were gray and atrophied. The preparation from this case is, I believe, one which I some years afterward saw preserved in the Musée Dupuytren. However, the occurrence of such a morbid change in a number of isolated nerve roots, independently of any other lesion, would be scarcely conceivable, whereas the relatively small number of motor fibres in the peripheral trunks renders it easy to understand why in their case a similar affection should escape notice, supposing it to be present. And I believe that those who, since the time of Cruveilhier, have doubted the "myopathic" origin of the disease have always thought that it would ultimately be proved to have its seat in the cord. So lately as 1873, Friedreich, of Heidelberg, devoted a quarto volume to the support of the former view, and proposed to term the affection a "Polymyositis chronica progressiva." His chief arguments were that the spinal lesions which had in certain cases been found were very variable in their nature and in their exact position, and that they might fairly be attributed to the extension of an inflammatory process upward along the

nerves. But about the same time Charcot devoted five of his lectures to this subject, and he has shown beyond dispute not only that the most typical form of progressive muscular atrophy in one or both upper limbs is due to a particular change in the corresponding anterior gray horn or horns, but also that other clinical varieties of the complaint can be assigned to separate and distinct affections of the spinal cord.

It seems to me that one thing which has introduced a large amount of obscurity and confusion into the study of this disease, has been the adoption by subsequent writers of a phrase of Duchenne's, according to which it is characterized specially by *atrophy without paralysis*. The great electro-therapeutist ultimately abandoned the myopathic theory of its origin which he had at first supported, but he nevertheless continued to maintain that there was no failure of "nervous *motor* action." He thought that the multipolar cells of the anterior cornua naturally possessed two independent functions, the one motor, the other trophic, and he supposed that the *trophic* function alone was interrupted by the lesion of progressive muscular atrophy. He asserted that the loss of power was always proportionate to the degree of wasting which was present, and that any muscular fibres remaining at a particular time would always obey the will, even though they might not succeed in overcoming the resistance opposed to them so as to effect a visible movement. In proof of this doctrine he appealed to the fact that faradization of the affected muscles would cause contractions in them even when their substance had undergone the most extreme wasting, provided that some fibres were still undestroyed. Now, it is certain that the maintenance of a normal electro-muscular contractility—which Duchenne formerly maintained to be the condition in progressive muscular atrophy—would be in flat contradiction with what is observed in all other affections of the multipolar cells of the anterior cornua. But I think that the way out of the difficulty is not far to seek. Erb has recently investigated very carefully the action of galvanic as well as of faradic currents, and he says (Ziemssen, "Rückenmark Krankh.," ii, p. 311) that he has always been able to detect in some of the affected muscles a modified form of the "reaction of degeneration:" the fibres contracted slowly under a powerful galvanic current when they were no longer sensitive to the faradic current, and A.C.C. was greater than C.C.C. (p. 345). The impossibility of verifying this change in other muscles, and the fact that it is easily overlooked, are attributed by him to the partial and gradual character of the wasting process. The fibres which have undergone degeneration have their true nature concealed by the still healthy fibres which lie side by side with them. So far as theory can go, this seems perfectly satisfactory, but Erb himself has overlooked the corollary that it does away with the necessity for supposing that paralysis is not present. It seems to me that those parts of the muscles which are affected by the disease are really as much paralyzed as in any other spinal affection whatever. Thus, the principal distinctive feature of progressive muscular atrophy appears to be the way in which the individual fibres of the muscles are, one by one, attacked and destroyed.

Histologically the morbid process in them is far from being as simple as it was at one time supposed to be. By the earlier writers on the subject they were described as being in a state of "fatty degeneration," having lost their transverse striation more or less completely, and being full of granules and small globules of fat. The accuracy of this statement was soon disputed by Robin, who pointed out that the granules were often dissolved by acetic acid, and therefore could not be of a fatty nature; and it is now known that many of the fibres become merely reduced in size and thickness while retaining their striation, that others pass into the condition known as cloudy swelling, and that others,

again, undergo a "waxy" degeneration, exactly like that which we have seen to occur during enteric fever (p. 208). Another part of the process consists in an increase in the number of the sarcolemma nuclei, and at the same time the interstices between the muscular fibres become the seat of a cell growth, from which are developed the adipose and connective tissues that sometimes give to the nuclei a deceptive appearance of being still well nourished and substantial. The fibres themselves undergo destruction, not simultaneously, but in succession. Thus there are all possible degrees of wasting, down to a point at which the microscope reveals not a vestige of the normal striation, nor even a single empty sarcolemma sheath. Muscles so affected, instead of red, are pale gray or yellowish, and at length become converted into mere bands of white fibrous tissue, or shapeless masses of fat, passing across from the bones to the tendons.

I have already alluded to the observations of Charcot, from which it appears that under the designation of progressive muscular atrophy there have been included several distinct lesions of the spinal cord. I must now briefly cite his descriptions of these, and it will be convenient to mention, at the same time, those peculiarities in their symptoms and in their course by which he thinks that they can be clinically distinguished from one another.

1. In the typical affection of Aran and Cruveilhier—Charcot proposes to call it *amyotrophie progressive protopathique*—the primary morbid change is an atrophy of the multipolar cells of one or both of the anterior gray cornua. They may either become pigmented, or shriveled without any change of color. At the same time the neuroglia undergoes proliferation, granule cells are formed in it, its vessels become enlarged and thickened, and in extreme cases the whole anterior cornu may be found shrunken in all its diameters. The resulting impairment of muscular power presents those characters of sharp and apparently capricious limitation which have already been fully described, and is accompanied neither by disturbances of sensation nor by spasmodic contractions of any muscles. Charcot says that his account of the pathology of this form of the disease is based upon six or seven autopsies. It is only in thin sections of the hardened cord that it can be recognized with any certainty.

2. Contrasting with that affection there are others which Charcot terms *amyotrophies deutéropathiques*, because in them the change in the anterior cornua is secondary to a lesion of some other part of the cord. Of these, he describes a *sclérose latérale amyotrophique*, consisting in a chronic inflammatory process which begins in the lateral white columns (occupying them symmetrically on each side), and then spreads into the gray matter, principally in the cervical region. Türck, in 1856, appears to have been the first to notice such a morbid change occurring independently of any disease in the brain. Charcot says that it is characterized clinically by the presence of paralysis with rigidity of the lower limbs, in addition to the atrophy of muscles in the upper limbs. This, of course, accords perfectly with the modern French doctrine about the effects of sclerosis of the lateral columns in general. The spasm in the legs is at first transitory, but afterward permanent; they generally assume a position of flexion; they lose but little of their substance. In the arms and hands the muscles undergo wasting *en masse*, and are not picked out one by one, as in the typical variety of progressive muscular atrophy; the elbow becomes somewhat fixed in a position of semiflexion and pronation; the wrist is semiflexed; the fingers are bent into the palm of the hand. Charcot further says that the morbid change in the muscles presents inflammatory or reactive characters in a more marked degree than in the "protopathic" form; the connective tissue between the fibres undergoes a still more decided overgrowth, and the

nuclei with which it is infiltrated are more numerous. Another peculiarity is that the course of the disease is more rapid; all four limbs become quickly involved; the patient is confined to his bed in a few months and does not live more than from one to three years, the usual cause of death being an extension of the morbid process to the medulla oblongata, with the symptoms of "bulbar paralysis." This form of progressive muscular atrophy is said by Charcot to be always incurable; those affected by it have been from twenty-six to fifty years old; exposure to cold and damp has sometimes been assigned as its cause. At the Sâlpetrière five cases have occurred in which autopsies have been made.

3. Yet another form of *amyotrophie deutéropathique* is an affection which Charcot calls a *pachyméningite cervical hypertrophique*. This, as its name implies, is a chronic thickening of the dura mater, which presents a number of concentric layers, and may fill up the whole vertebral canal. It and the arachnoid adhere firmly to the cord, so as to compress and flatten it; and necessarily surround and press upon those nerve roots which come off at the level of the lesion, generally belonging to the brachial plexus on each side. This affection, like that last mentioned, is attended with progressive wasting of the muscles of the upper limbs, and with rigidity of the lower limbs. Charcot, indeed, says that it is distinguished by the circumstance that the ulnar and median nerves are especially involved (the musculo-spiral nerve escaping), so that the wrist assumes a position of extension instead of being flexed. He also remarks that parts of the cutaneous surface often become anæsthetic, not only in the arms, but even in the upper part of the trunk; and that when the lower limbs become rigid they do not seem to waste. But what is especially characteristic of cervical pachymeningitis is the occurrence of an early stage, lasting two or three months, and accompanied by severe pains in the neck and back of the head, by a sort of rigidity of the cervical muscles, by sensations of numbness and tingling in the upper limbs, and sometimes by bullous eruptions. All of these symptoms are, of course, due to irritation of the nerve roots at the seat of the disease. The affection evidently stands toward the other diseases that are now being described in the same relation as does a "compression paraplegia" toward the common forms of myelitis. Perhaps the most important point of all is that Charcot describes it as not always incurable. A woman under his care, after an illness which lasted five or six years, and during which she was for a long time perfectly helpless and confined to her bed, ultimately became able to walk, and could also, to some extent, make use of her hands. The lesion does not tend to spread to the medulla oblongata, so that the symptoms are not apt to become complicated by the supervention of bulbar paralysis. On the other hand, there may at last be loss of power over the bladder and the rectum, and bed sores may develop themselves, symptoms which are not observed in amyotrophic lateral sclerosis.

In 1868 I recorded, in the "*Practitioner*," a series of cases of progressive muscular atrophy, one of which would seem to have been an example of hypertrophic cervical pachymeningitis, if reliance can be placed on the facts that rigidity of the lower limbs constituted a prominent symptom, and that the patient, a woman, aged thirty-two, recovered. At that time I spoke of the affection as altogether exceptional. It is interesting now to refer to the clinical report, and to find that at an early stage she had complained of severe pains about the elbows, which were doubtless due to irritation of nerve roots. Moreover, three years previously, she had for a time lost power in the second finger of the right hand. I have also reason to believe that a second example of the same affection is afforded by another of my cases, that of G. P. H—, aged twenty-seven, who is recorded as having

recovered from a well marked form of progressive muscular atrophy under treatment by the continuous current. This man was readmitted into the hospital in 1869, with severe cramps and numbness in the arms, rigid extension of the legs, rigidity of the recti muscles of the abdomen, and a sensation of tightness round the waist, as if constricted by a cord. On referring to the notes taken in 1867, I find that at that time he had tingling sensations in the arms and weakness of the lower limbs, so that even then the case deviated to some extent from the typical "Cruveilhier's paralysis."

4. Again, in some instances of what must still be called a deuteropathic form of the complaint there is a very obvious lesion, in the form of a hollow space occupying the centre of the cord. The first case in which an atrophic paralysis of the upper limbs was observed in connection with such a lesion was recorded by Gull in the "*Guy's Hospital Reports*" for 1862. The patient, a journeyman tailor, aged forty-four, had suffered for thirteen months from loss of power in the little and ring fingers of the right hand, and for a shorter time from a similar affection of the corresponding fingers of the left hand. The muscles of the hands had undergone extreme wasting. He caught typhus in the hospital, and died of that disease. In the cervical and upper dorsal regions of the cord the place of the gray matter was taken by a large quadrilateral cavity, which was supposed to be a dilated part of the central canal. But a *hydromyelus* having sometimes been met with in persons who had been altogether free from spinal symptoms, the doubt might fairly be entertained whether its presence was not after all a mere accident. In children suffering from spina bifida, at any rate, such an affection is of common occurrence. Since the publication of Gull's case, however, similar ones, attended with like symptoms, have been met with by Schüppel and Hallopeau and Westphal; and it now seems impossible to explain them away. Th. Simon has shown that in reality the cavity is often altogether unconnected with the central canal, which lies in front of it and is no larger than natural. He proposes to apply to such cases, instead of hydromyelus, the name of *syringomyelus*, originally invented by Ollivier, but not with the object of conveying this distinction. I have already mentioned that the development of a solid new growth in the cord is often attended with the formation of a cyst in its interior; but the cases now under observation do not seem to come under that head. Clinically, the progressive muscular atrophy due to syringomyelus does not appear to differ from the ordinary protopathic form of the disease.*

5. Lastly, a deuteropathic progressive muscular atrophy may occur as a complication in cases of *locomotor ataxy;* and also in those of *multiple sclerosis*, if the morbid change should happen to involve the anterior cornua and to destroy extensively the multipolar cells. A *tumor* growing in the substance of the cord, or in the membranes, may sometimes be attended with symptoms of a similar kind. Erb remarks that it is not always possible to distinguish between a meningeal new growth and cervical pachymeningitis. I had in 1878 a case which illustrated this very point. A woman was admitted with atrophic paralysis of certain muscles in the left hand; and she also was liable to exceedingly violent paroxysms of pain in the right shoulder and arm, attended with sudden redness and swelling of the tender parts, evidently due to disturbance of the vaso-motor nerve. She had had iritis, and on the whole I inclined to the diagnosis of a syphilitic gumma, growing from the membranes and pressing on the cord in the cervical region. She improved under iodide of potassium before she left the hospital.

Diagnosis.—From considering how the various spinal lesions that may give

* [See the remarks by Dr. Taylor on his ten cases, reported in the "*Pathological Transactions*," vols. xxix, p. 21, and xxxv, p. 36.—Ed.]

rise to progressive muscular atrophy are to be distinguished from one another, we naturally pass on to discuss the diagnosis between this disease and other diseases that are attended with wasting of muscles. And first among them should, perhaps, be mentioned various forms of *peripheral paralysis* due to neuritis of the several branches of the brachial plexus, such as have been described at p. 347, *et seq.* There is no doubt that cases of this kind have often been mistaken for those of Cruveilhier's palsy; but a careful observer will generally recognize at a glance the limitation of the affection to the area of distribution of particular nerves; and all doubt will be removed by the application of galvanic and faradic currents, revealing a far more marked perversion of the normal electrical reactions than is ever seen in progressive muscular atrophy. The same tests will at once distinguish ordinary cases of *lead paralysis*, even when the history of the patient, the line on the gums and the other poisonous effects of the metal are not of themselves sufficient to prevent any hesitation as to the diagnosis. I have seen a few instances in which, although it was known that the poison had been absorbed to some extent, opinions yet differed as to whether this fact would satisfactorily account for the symptoms, which seemed to be too severe, or too little amenable to treatment, to be caused by lead. Two such cases I published in the "*Practitioner*," under the name of progressive muscular atrophy, although with some hesitation. I am bound to say that now, on reading them over, I think that the view then taken was wrong: they were cases of plumbism. Another condition which has sometimes to be distinguished is a diffused wasting of the muscles, attended with great helplessness and loss of power, as a result of protracted *rheumatism*. The hollowing of the interosseous spaces and of the ball of the thumb may be very striking in cases of this kind. French writers also describe, under the name of *marasme essentiel*, an affection which they say occurs in hypochondriacal patients, who gradually assume the appearance of living skeletons.

The *prognosis* of progressive muscular atrophy must be based, in the main, upon the facts already stated with regard to the natural course and duration of the disease, varying as its different forms and individual cases do.

Treatment.—It only remains for us to consider to what extent cure is possible, and I think that the universal experience has been that drugs are altogether useless. Neither arsenic, nitrate of silver, phosphorus, nor iodide of potassium can restore the wasted muscles, or prevent further extension of the morbid process. But there is a large amount of testimony to the value of electricity. By Duchenne faradization of the affected parts was strongly recommended. He advised the application of currents of moderate intensity, with not too frequent intermissions, and for a few minutes only at a time, so as not to fatigue the fibres remaining undestroyed; he particularly insisted on the importance of including in the treatment any important muscles, such as the diaphragm, the intercostals, and the deltoids when they are first threatened by the disease, and before it actually invades them. In the case of a man named Bonnard, who had lost many of his trunk muscles, and who was beginning to suffer from dyspnœa, so that he could scarcely walk a few steps without stopping to take breath, faradization of the phrenic nerves, repeated three or four times a week, was of great service, enabling him to walk considerable distances, and to go up stairs without fatigue. A similar treatment, applied to certain muscles of the arm which were wasted, restored their functions, so that at the end of six months he was again able to support his family by his exertions. And he went on for some years without the disease advancing further.

On the other hand, Remak advocated the use of the continuous current. His method was to place the positive pole in front of one mastoid process,

and the negative pole on the opposite side of the neck, near the spinous processes of the vertebræ, not higher than the fifth cervical. This, he found, would often produce contractions, which he termed diplegic, in the fingers or other paralyzed parts.

All observers admit that in not a few cases each of these plans of treatment fails utterly. They should, however, always be tried in succession, or at the same time. And it is worth while to persevere with them, even when they seem at first to be doing no good, because it has sometimes happened after several months that favorable results have been attained. At the commencement of the disease the affected parts should be rested as much as possible. In one of my cases, the forearms were wrapped in cotton wool and placed in splints, and after a week the patient was found to have much more power in his hands. But in the more advanced stages, methodical exercise of the wasted muscles appears to be sometimes useful.

Progressive Bulbar Paralysis.—In 1860, Duchenne, of Boulogne, gave a clinical description of a form of progressive paralysis of the tongue, palate and lips, which had previously received no systematic recognition from writers in medicine, although Trousseau, as far back as 1841, had noted the peculiar symptoms presented by a well-marked case occurring in a personage of princely rank. The disease has now become well known in England, and is generally termed labio-glosso-laryngeal paralysis; but of late there has been a tendency to substitute for this name that of "progressive bulbar paralysis," which was originally suggested by Wachsmuth in 1864, and which certainly seems to be preferable. As Kussmaul has remarked, it might with still greater accuracy be called "progressive paralysis of the bulbar nuclei;" for the morbid change does not involve that part of the medulla oblongata which transmits the motor and sensory strands for the limbs and the body generally, and is confined to the gray centres for certain of the cranial nerves on the floor of the fourth ventricle.

As a rule, progressive bulbar paralysis begins very insidiously. The earliest symptoms are commonly subjective—a feeling of pressure, or even of pain, at the back of the neck and head, a little giddiness, a sense of constriction round the throat or chest, a slight discomfort in talking, as if the tongue were heavy and its movements labored, or a tired feeling after speaking for some time without intermission. Krishaber has stated that in two cases he discovered a loss of reflex irritability in the pharynx and larynx some months before any signs of paralysis made their appearance. Sometimes the palate is affected before the tongue, or the disease begins in the lips; in the former case the speech acquires a "nasal" quality, and swallowing seems to require an unusual effort; in the latter the expression of the face about the mouth becomes altered, or there may be a little difficulty in the utterance of certain letters, as though (to use a phrase of Duchenne's) the lips were half paralyzed by cold.

Sometimes, however, the commencement of the disease appears to be sudden. Kussmaul, in one of the clinical lectures published by Volkmann, relates that a patient of his, a Catholic priest, found one day, while preaching, that his mouth was distorted, and that he had a difficulty in speaking. He was able to finish his sermon, but from that time there remained a heaviness of the tongue. For a week previously he had suffered from pains in the back, but he had had no giddiness. Dysphagia soon set in, and within six months the case became one of confirmed bulbar paralysis, with loss of power in the arms and wasting of the small muscles of the hands. It was ascertained at the autopsy that there was no hemorrhage into the pons or the medulla oblongata; and this, as well as the progressive character of the complaint, distinguishes it from certain cases of Wilks, to which

I shall presently refer (p. 460). In a woman whose case is recorded by Leyden, the first symptom was a sudden attack of dyspnœa, lasting five minutes; a few days afterward she noticed a difficulty in moving the tongue when she spoke or ate.

In fully-developed cases, the tongue is generally the part in which the loss of power is most obvious. It lies flaccid in the floor of the mouth; the patient can neither bend it laterally, nor raise it against the palate, nor hollow its centre; he may even be unable to protrude it beyond the teeth. Scarcely less marked is the paralysis of the lips. The mouth remains open, and with its angles drawn wide apart; the naso-labial furrows are deepened, the lower lip hangs away from the gum. The patient cannot whistle or blow out a candle, or kiss. What is most remarkable is that although facial muscles supplied by the lower branches of the *portio dura* are thus affected on both sides in every case, those to which the upper branches of the same nerve are distributed as constantly escape. The orbicularis palpebrarum and the occipito frontalis act as well as ever. The countenance thus acquires a curious expression: the expression about the eyes is full of life, that of the mouth is fixed, sad and gloomy.

On the other hand, the paralysis of the palate is not indicated by any obvious change in its form. The uvula hangs in its ordinary place; and Duchenne says that he always found that irritation of it caused the usual reflex movements. With the laryngoscope the vocal cords may, in advanced cases, be seen to be more or less completely paralyzed.

Several important functions are impaired in ways which are somewhat complicated and demand careful study. Thus the defect of speech appears to vary with the part which becomes earliest paralyzed. According to Kussmaul, if the lips suffer first, O and U are the vowels which the patient experiences most difficulty in uttering; if the tongue, I is sooner lost. A is always retained longer than any of the others.* Among consonants, loss of power in the tongue renders the patient first unable to utter R and SH; next, S, L, K, G, T; afterward D and N. Paralysis of the lips prevents the formation of P and F, then of B and M, ultimately of V. Paralysis of the palate gives to the speech a nasal twang, and it specially prevents the formation of the lip sounds B and P, because it allows so much of the air to escape through the nose; the proof of this being, as Duchenne pointed out, that closing the nostrils may enable these letters to be sounded. So far, the affection of speech is one which merely concerns articulation, and may, as is now proposed, be called "alalia" or "anarthria," in contradistinction from the "aphasia" that depends upon lesions high up in the left side of the brain, and to the "aphonia" that is caused by loss of power in the larynx. But in bulbar paralysis, after a time, the vocal cords themselves lose their functions; the voice then becomes altogether extinguished, and the patient is able to utter nothing beyond a meaningless grunt.

Another set of movements which are interfered with in progressive bulbar paralysis, are those which are concerned in the reception of food into the mouth, and its transmission backward into the œsophagus. During mastication it collects inside the cheek, not only when the buccinator is paralyzed, but (even apart from this) because the tongue cannot properly dislodge it. Very often the patient helps himself with the fingers of both

* These vowels must be taken in the Italian pronunciation: U = OO (in *fool*), I = E (in *feel*), A = Ah (in *far*). To complete the list of the chief sounds which occur in English, those represented by the letters Th would have to be added; probably they are among the first to disappear in cases in which the tongue is early affected. I may remark that Kussmaul's statements correspond closely with what might have been anticipated from theoretical considerations, of which a very admirable account will be found in a paper by Dr. Bristowe, in the first volume of the "*St. Thomas's Hospital Reports.*"

hands, supporting the floor of the mouth or the cheeks, or pushing the half-chewed food into the proper position. Then, again, the tongue cannot roll up the softened pulpy material into morsels for swallowing, nor carry them into the pharynx. Loose fragments are constantly dropping out of the open mouth into the plate or upon the patient's clothes. Other pieces collect about the root of the tongue or in the grooves by the side of the epiglottis. The attempts to swallow them succeed very imperfectly—some pass up into the pharynx or through the nose, others enter the larynx and set up a choking cough; they may even accumulate in the fauces to such an extent as to interfere with the passage of air and to produce sudden death. It depends upon circumstances whether the patient finds most difficulty in dealing with solids or with liquids. If the principal defect is a weakness in the tongue and in the muscles of mastication, he requires to have all his food reduced to a semi-fluid state; but when there is paralysis of the parts concerned in closing the larynx during deglutition, he can often dispose of solid masses better than of liquids, being unable to prevent the latter from trickling down into the air passages. The inability to swallow gives to the disease another peculiarity in the circumstance that the saliva keeps running out of the mouth; such patients keep a handkerchief constantly held below the chin, and, as Wilks remarks, this often at the first glance enables one to guess what is the matter with them. The secretion itself is sometimes viscid, sometimes watery. It seems so abundant as naturally to suggest that it is formed in excess, and in one instance Schultz is said to have estimated that there was six or eight times as much of it as would have been poured out under normal conditions. Kussmaul, however, found no such increase in a case in which he determined its quantity. Sometimes the masticatory muscles become at length involved in the paralysis; the patient is then unable to move the lower jaw from side to side, nor can he close the mouth firmly.

Generally, the respiration is not obviously affected at an early stage of the disease. Later on dyspnœa often becomes a marked symptom, and on stripping the patient one may find that the respiratory movements of the chest walls are very shallow. In some cases, however, distress of breathing and a constant craving for air are said to have been present, although the diaphragm and the thoracic muscles were still vigorous. Kussmaul cites the written statement of a woman under his care who was in great dread of gaping, because "she was obliged to groan and strain in order to get rid of the air and then breathe more freely." He follows Duchenne in attributing such symptoms to paralysis of the bronchial muscles. There is generally an inability to sneeze, to cough, to hawk up phlegm, and to blow the nose; the patient sometimes complains bitterly that he is no longer able to smoke. Toward the last, paroxysms often occur, attended with a rapid pulse; these are supposed to depend upon paralysis of the vagi.

Among the symptoms which have been noticed in some exceptional cases, may be mentioned slight deafness, noises in the ears, ptosis, paralytic affections of the ocular muscles, numbness and anæsthesia of the face or tongue. Physiological considerations have led some observers to search for sugar and for albumen in the urine; but I believe that the results have always been negative.

The higher cerebral functions remain undisturbed, the patient sleeps well, his intelligence and memory are perfect. The movements of the body and limbs are generally free and active, except toward the last, when emaciation from want of food often causes extreme weakness.

But although the great motor paths through the bulb and the pons thus escape and are in no way interfered with, it nevertheless frequently happens that there is associated with progressive bulbar paralysis an affection of the

upper (or even of the lower) limbs, such as has already been described as "Progressive Muscular Atrophy." And sometimes one, sometimes the other of these two diseases is the first to develop itself. The fact that this connection exists is of considerable importance, for Duchenne drew a sharp distinction between them. In labio-glosso-laryngeal paralysis, he said there was paralysis without atrophy; in progressive muscular atrophy there was atrophy without paralysis; and certain cases in which he found the tongue wasted were regarded by him as examples of a separate malady, a true progressive muscular atrophy involving that organ. But as I have already observed, the tendency of recent investigations into "Cruveilhier's palsy" has been to discredit the earlier statements as to the non-occurrence of a real paralysis in that disease, and even as to the necessary presence of a trophic degeneration in all the muscles affected by it. Moreover, there seems to be no satisfactory proof that, after death from progressive bulbar paralysis, the muscles have ever been examined microscopically and found to be, all of them, in a healthy state. And, inasmuch as Charcot once discovered a tolerably advanced degenerative atrophy in a tongue which, during life, had appeared smooth and of natural size, it is clear that nothing but pathological evidence can be conclusive. In many cases the fleshy substance of the tongue, and even that of the lips and palate, is obviously pale, of a yellow or grayish-red color; and it is streaked with fat or more or less completely converted into a mass of fatty connective tissue.

Thus at the present time the opinion of all the most competent authorities is that progressive bulbar paralysis is, in part, identical with progressive muscular atrophy, differing merely in the circumstance that it affects a particular set of nerve nuclei in the bulb instead of those in the cord. It is admitted that in the earlier stages the tongue is often not obviously reduced in size, but in advanced cases it is described as being soft, small, wrinkled on the surface, and incessantly agitated by a fibrillary tremor. The lips also become thin and sharp edged; their muscular substance quivers, and the skin over them is marked with minute furrows. In the palate, as might be anticipated, wasting of the muscles is not discoverable, at least during life. It is allowed that there is no absolute correspondence between the degree of the paralysis and that of the atrophy.

The results which have been attained by testing the affected muscles with electricity appear to accord with these statements. The earlier investigators described the faradic contractility as normal; but several German observers have since found it lowered, or even extinguished; and with galvanic currents, Erb (as well as Kussmaul) has recently detected the "reaction of degeneration" in a perfectly characteristic form. The mistake seems to have arisen from the stimulus having been applied to the motor nerves rather than to the muscles themselves.

Histology.—Progressive bulbar paralysis is one of those diseases of which it has been possible to work out the morbid anatomy satisfactorily only within the last few years, since we have been in possession of the new methods of investigating the nervous tissues. Kussmaul cites eight cases, in each of which a complete microscopical examination was made. In general, no marked change is seen in the fresh medulla oblongata; but it is said that sometimes a little want of symmetry in the two halves of the floor of the fourth ventricle has been detected, or a slight shrinking; or that this part has appeared discolored and reddish-gray; or that its texture, when cut into, has looked blurred; or that its consistency has been greater or less than natural. The microscope shows at most some granule cells, a little increase of the connective tissue, a few atrophied or pigmented ganglion cells, or vessels with thickened walls. One thing, indeed, is very obvious, even to the naked eye, namely, an extreme degree of atrophy of the nerve roots arising

from this part of the cerebro-spinal axis. It is especially conspicuous in the hypoglossal and the facial nerves; but it is generally plainly visible also in the three divisions of the eighth nerve on each side, and sometimes in the sixth, and in the motor portion of the fifth. The roots in question are gray and transparent, and exceedingly reduced in size.

In stained sections of the hardened medulla oblongata, the multipolar cells in certain nuclei are uniformly found to have undergone degenerative changes. Their color is darker than natural, being deep yellow or yellowish-brown; they are often shrunken; their prolongations may be indistinct or may have quite disappeared. They seem to be reduced in number. In one case Charcot and Joffroy are said to have come to the conclusion that the hypoglossal nucleus contained only one-tenth or one-twelfth part of the cells which would have been present in it in a healthy bulb. French histologists appear not to have recognized any marked change in the neuroglia, but in Germany both Leyden and Maier have found it increased in quantity and filamentous, containing stellate cells; in other words, presenting appearances like those which characterize myelitis going on to sclerosis. The nerve fibres undergo atrophy. The structures in which these parts are most constantly seen are the nuclei of the hypoglossal, the accessory, the vagus, and the facial nerves. As regards the last-mentioned nerve, indeed, the symptoms of bulbar paralysis point to the conclusion that only a part of its nucleus is affected; and this accords well with Lockhart Clarke's statement that its roots arise in two separate masses of gray matter; but it does not appear that the obvious pathological application of his observations has been yet traced out in detail. The nucleus of the glosso-pharyngeal nerve escaped the morbid process in a case recorded by Duchenne and Joffroy. The sensory nucleus of the fifth and that of the auditory nerve have been constantly found intact. The olivary bodies have sometimes presented degenerative changes, but more often they have been in a normal state.

With regard to the *ætiology* of bulbar paralysis but little is known. It scarcely ever occurs in persons under thirty, and appears absolutely to increase in frequency as age advances up to the seventieth year. It is rather more common in men than in women. Erb speaks confidently of its being sometimes caused by cold, and Kussmaul relates a case in which the patient traced it to a cold caught while he was at work in the fields; he was attacked with headache and pain in the neck, his mouth became full of water, and a week later he was conscious of difficulty in swallowing and in speaking. Among the other conditions which have been supposed to give rise to it are excessive smoking, over-exertion in playing wind instruments, syphilis, and falls on the head, or even on the feet or the buttocks, causing concussion of the medulla oblongata. In one of Trousseau's cases it began during convalescence from a short febrile attack, attended with delirium.

The *diagnosis* of this disease at its commencement requires considerable acumen; one might easily make light of the early symptoms, and so justly forfeit the confidence of the patient. Even when they are fully developed, a hasty conclusion is dangerous. It is to be remembered that the lips and the tongue may be paralyzed by various affections, beside that which constitutes the disease described by Duchenne. What characterizes them is the presence of additional symptoms which do not belong to it. Most of the cases that have been recorded as occurring in young persons have presented distinctive features, and their pathology must be regarded as still undetermined. Again, Wilks has pointed out that a condition precisely like that which belongs to progressive bulbar paralysis may be suddenly developed as the result of a circumscribed effusion of blood

into the lower part of the pons. He relates in the "*Guy's Hospital Reports*" two cases, in one of which an old brownish cyst was found at that spot after death. It does not appear that their course was such as to justify one in giving a more favorable prognosis in this form of bulbar paralysis. On the other hand, he observes that he has seen the disease simulated fairly well by hysteria; and other observers have recorded cases, in which syphilis has been present, and which have been cured by iodide of potassium. All such cases must, for the present, be regarded as distinct. So also, tumors growing near the bulb may cause similar symptoms.

Prognosis.—It seems probable that all of the few recorded instances in which recovery has taken place from "bulbar paralysis" have been essentially different from the disease now under observation. The "*Clinical Society's Transactions*" contain a very remarkable case, observed by Dr. Dowse, of a young man who after a series of epileptic seizures is said to have been affected with paralysis of the whole body, with the exception of the muscles of the eyeballs. At the end of four years he had improved greatly, but the tongue still lay immovable in the floor of the mouth, and he was quite dumb, and had difficulty in deglutition. He was under observation in the institution at Highgate for nearly twelve months, and was making no further progress when a few applications of a continuous current to the tongue restored the power of articulation almost suddenly. He went on taking cod-liver oil, quinine, and phosphorus; and at length his cure was complete.

Very different is the prognosis in those cases which depend upon a progressive change in the bulbar nuclei. They appear always to end fatally. Their duration is generally from one to three years; but it may be as long as five years. Sometimes the patient is choked unexpectedly by a mass of food which cannot be propelled beyond the entrance into the larynx; of this an instance occurred at Guy's Hospital in 1865. Sometimes death is brought about by an attack of syncope, sometimes by a paroxysm of dyspnœa. Kussmaul remarks that such seizures are particularly apt to happen after exertion of some kind, but that they not infrequently occur at night when the patient is in bed. In other cases exhaustion and emaciation gradually lead to a fatal termination; or pulmonary phthisis may develop itself; or a pneumonia, consequent upon the admission of food into the air passages during the act of deglutition.

I do not think I have ever seen any benefit result from *treatment* of this disease. Kussmaul recommends dry cupping at the nape of the neck in early cases, and the use of shower baths. Among drugs he thinks the nitrate of silver most likely to be serviceable. He has seen transitory benefit from faradization of the palate and tongue; and he mentions two cases in which striking results were for a time attained by the use of strong galvanic currents passed through the neck and spine. One patient, a priest, who had been unable to get his tongue out between his teeth, could after being galvanized protrude it beyond his lips; his speech became more distinct and his deglutition easier. After four or five weeks, however, the improvement ceased. Specially applicable to progressive bulbar paralysis is, perhaps, Schulz's method of inducing the act of deglutition by galvanism. It consists in fixing the positive pole upon the nape of the neck, and then rapidly moving the negative pole downward over the side of the larynx. This procedure may be repeated at short intervals several times during four or five minutes. In the first instance a current from six or eight cells should be tried; but to produce the desired result a large number of elements are often required. When the patient cannot swallow food at all, or seems likely to be choked in the attempt, a tube must, of course, be passed into the stomach; but this procedure often has to be abandoned, on account of the

irritation excited by it. Nutrient enemata then afford the only means of sustaining life for a time, unless recourse is had to gastrostomy. In one case the trachea was opened when danger was apprehended from frequent attacks of dyspnœa; and the result was that they ceased, although of course the other symptoms remained.

Acute Bulbar Paralysis.—This appears to be the most convenient place for a brief notice of an affection which on clinical grounds requires a separate description, although to the pathologist it is merely the representative of a circumscribed myelitis which happens to be seated in the bulb instead of in the spinal cord. Three examples of it are cited by Erb, all of which were observed by Leyden. In one case a patch of softening with numerous capillary extravasations of blood was plainly visible in the pyramids and in the olivary bodies at the autopsy; in the others no lesion was discovered until the parts had been hardened, when the microscope revealed inflammatory changes of the most marked kind. Each patient had been taken ill rather suddenly, and had died in from four to ten days. The symptoms varied considerably, being, of course, dependent upon the exact seat of the morbid process. Chief among those which were present in one case or another were headache, giddiness, vomiting or severe hiccough, difficulty of deglutition, or even a complete inability to swallow, a more or less profound impairment of speech, irregularity or great rapidity, or occasional interruption of the breathing, a quick and feeble and irregular intermittent pulse, a partial or complete paralysis of the tongue, of some or all of the limbs, and perhaps of the face, formication and pains in the limbs. There was no failure of consciousness, nothing at all resembling an apoplectic seizure. Depression and collapse quickly set in, and the fatal issue was brought about by paralysis of the respiratory muscles, with distress of breathing and lividity. As may be supposed, the diagnosis of the seat of the disease was not difficult; but it was not easy to exclude the possibility of embolism of the basilar artery or of the vertebral arteries, or their branches, or of a minute spot of hemorrhage; or even of a rapidly developed compression of the medulla oblongata by some disease in its neighborhood. The most important consideration of all is that, perhaps, such cases need not invariably terminate fatally, since ordinary myelitis is not infrequently recovered from. The treatment must no doubt be such as is found useful in that affection, with the addition of measures adapted to preserve the patient from the effects of his being unable to swallow, and of the impairment of the cardiac and respiratory movements. A case which seems to have been of this kind occurred in Guy's Hospital in 1874, in the person of a man aged forty-five. It proved fatal in about ten days. The central part of the pons appeared softened; but after preparation in chromic acid no decided morbid changes were made out.

Progressive Muscular Atrophy of Childhood.—According to Duchenne, progressive muscular atrophy, when it occurs in children, presents certain peculiarities in its symptoms and course. It begins in the lips, which become thick and hanging and cannot be brought together. If the child smiles, the angles of the mouth are drawn far apart, and the cheeks are flattened by the action of the *buccinator* muscles. The articulation of labial letters and of the vowel *o* is impaired. It is almost always between the fifth and the seventh year that this form of the affection first appears. But at that time it often attracts very little notice from the parents, although the reality of the morbid change is said to be at once made apparent if the orbicularis oris and the other muscles are tested by faradic currents, when they are found to have lost their contractility. About the eleventh or

twelfth year, however, the muscles of the shoulders and arms begin to waste capriciously and irregularly; and it is now that medical advice is first sought. Later still, the muscles of the trunk and those of the lower limbs are attacked in their turn. I have no recollection of having ever seen a case of this kind; but Duchenne, whose first cases were recorded in 1855, said in 1872 that he had observed no fewer than twenty. One thing which would rather tend to show that the progressive muscular atrophy of childhood has no very close relation to the most common forms of wasting paralysis in adults, is that it very often occurred in two or more brothers and sisters; but a curious circumstance is that the father of the first two children in whom Duchenne recognized its peculiar characters, was afterward, at the age of forty-eight, attacked with a typical "Cruveilhier's paralysis," affecting first the shoulders and arms, but ultimately the lower limbs; he also stated that his father had died of a similar complaint. Whether this was anything more than an accidental coincidence it is difficult to say.

Paralysis from Chronic Plumbism.—Beside causing a form of colic and inducing gout, lead may also affect the nervous centres and give rise to a variety of symptoms.

Of these, the most common is a form of paralysis affecting the upper limbs, which, from its peculiar characters, is often called "the dropped wrist." I believe that this always attacks certain groups of muscles much more than others. Generally the extensors of the hand and the supinators are the chief ones to suffer; the patient's hand hangs powerless from his wrist, and his fingers are more or less forcibly flexed. When the forearm is laid prone upon a table, he is unable to turn it round so as to bring the palm uppermost. The muscles which form the ball of the thumb are affected very frequently, and sometimes before any others; those of the little finger also, as well as the lumbricales and interossei. In some cases the deltoid muscles are first attacked, and with them the lower part of the trapezii and the muscles which cover the dorsal surface of the scapulæ. The affected muscles always become greatly wasted. Thus the ball of the thumb, instead of being rounded, is sunken; the loss of substance in the interossei and lumbricales causes the flexor tendons to be visible on the palm of the hand; the neighborhood of the external condyle of the humerus and the back of the forearm are hollowed out and flaccid. If the shoulder is attacked it loses its roundness, and the outlines of the bones can be felt much more plainly than is natural. In some cases the whole upper limb may be weak, and all its muscles more or less wasted. But I have never seen a case in which the biceps and triceps and the flexor muscles of the hand were affected to the same extent as the extensor, still less of one in which any of these were the principal ones to suffer from the action of the poison.

Most frequently both upper limbs are attacked, but one much more than the other. Sometimes the paralysis is limited to one arm and hand. Certain writers have supposed that this is because they alone happened to absorb the poison, as, for instance, when a painter makes his colors with one hand, and little or not at all with the other. But such a view is altogether untenable, and I believe that any difference between the two upper extremities in this respect is to be attributed to the fact that the arm most affected is employed more than the other in the patient's daily work.

It is, however, a question whether lead paralysis is really due to an affection of the nervous centres, or whether it may not be caused by the local action of lead circulating in the blood upon the muscles themselves. John Hunter long ago took advantage of a fatal accident which had happened to a painter to examine the tissues of his hand and arm, which had been paralyzed. He found that the muscles were cream colored. Some years ago

a man died in Guy's Hospital, who had been attacked with lead paralysis seven years before, and had never completely recovered from it. Dr. Moxon found that the affected muscles—especially the deep extensors of the forearm —were represented only by loose, watery, connective tissue.

Sometimes, however, the diagnosis between lead paralysis and progressive muscular atrophy is so uncertain that the results of treatment have to be called in evidence upon it, and I am myself inclined to think that the latter affection, in its most typical form, is sometimes really the result of plumbism. I have met with at least one well-marked instance in which a paralysis of the forearm, presenting all the characters of progressive muscular atrophy, including its resistance to treatment, occurred in a man who had worked with lead for a length of time, and who had a well-marked blue lead line.

The paralysis which occurs in chronic plumbism is not always limited to the upper limbs. I have seen two instances in which the whole body was affected. The patient could not raise himself in bed, nor use any of his limbs.

The only disease for which lead paralysis is likely to be mistaken is progressive muscular atrophy. One distinction between them—on which Sir William Gull used to lay stress—is that in chronic plumbism the patient complains of pain when one grasps the affected muscles with one's hand, but I do not find that this is constantly, or even generally, the case. On the other hand, it is not attended with the fibrillary tremors which accompany progressive muscular atrophy. But the most remarkable distinction is afforded by the application of galvanism to the skin over the affected muscles. In lead paralysis, a faradic current causes but slight contraction or even none at all, whereas a continuous current gives rise to movements more readily than in health, that is, a smaller number of cells is required to excite the muscles to contract.

The general condition of the patient must also be carefully inquired into in all cases in which any doubt exists as to the cause of a paralytic affection of the upper limbs attended with wasting. The gums must be examined for the "blue line." One must ask whether the patient has suffered from any painful affection of the abdomen which could possibly be "lead colic;" but it is to be borne in mind that when a person has been slowly absorbing the metal in minute quantities for a length of time, paralysis often occurs without having been preceded by any pain in the bowels, and these are precisely the cases which are difficult of diagnosis. Sometimes the recognition of chronic plumbism is materially facilitated by the aspect of the patient, the countenance being in such cases peculiarly sallow and anæmic.

Another nervous affection which is said to be an occasional result of lead poisoning is a tremor resembling that caused by mercury. It has hitherto only been observed, by Brockmann, among the miners on the Hartz. It is generally limited to the arms and hands, but it very often attacks also the lips and the angles of the mouth. Very rarely it attacks the legs and the muscles of the head and of the trunk.

The medicine most useful in lead paralysis is iodide of potassium. Dr. Anstie lays stress on the importance of a nutritious diet, and on the value of cod-liver oil in such cases. Sulphur baths have been supposed to be serviceable. I believe that there is no doubt that they lead to the excretion of some of the lead which has been accumulated in the body; a blackish discoloration of the skin, and still more of the nails, is observed, which does not at once rub off, and which evidently is due to the conversion of some of the lead present on the body into a sulphide. Locally, the application of a continuous current, just sufficiently powerful to excite contraction of the

affected muscles is recommended. When its special action ceases faradization may be used instead, but not before.

Pseudo-Hypertrophic Paralysis.—In 1861, Duchenne, of Boulogne, recorded in the second edition of his "Électrisation Localisée" the case of a boy who, with legs so weak that he could scarcely walk or even stand, had the muscles of his calves and of his loins as large as those of an athlete. Seven years later Duchenne wrote a detailed paper on the disease in question. In the meantime cases had been noticed by a few German observers. I saw one in 1863, in Oppolzer's wards at Vienna; and another, which occurred to Griesinger, in 1864, gave Billroth the opportunity of excising a little piece of the deltoid muscle, and of proving that there was no real but only an apparent hypertrophy. It was found to consist almost entirely of adipose and fibrous tissue, separating from one another the muscular fibres, which themselves were unaltered. Duchenne, in 1865, made similar observations in one of his cases, with the aid of his "emporte-pièce." He therefore proposed to term the disease *paralysie myosclérosique*. Among other names which have been suggested for it may be mentioned "*lipomatosis musculorum luxurians*," and *sclérose musculaire progressif*. But, on the whole, I prefer to call it *pseudo-hypertrophic paralysis*.

Histology.—The substance of the enlarged muscles has a whitish-yellow color, with perhaps a faint reddish tint. There is still a linear arrangement of the fibres, and in extreme cases this may be the only thing which distinguishes it from the subcutaneous adipose tissue. The fat sometimes extends into the tendons, giving them during life an appearance of having been encroached upon by the fleshy bellies of the muscles. On the other hand, there has occasionally been found no fat, but fibrous tissue only, between the muscular fibres; and in the earlier stages of the disease, this new tissue has been found full of nuclei or containing actual spindle cells. The muscular fibres seem to become greatly reduced in number by a process of simple atrophy, which at last leaves only the collapsed sarcolemma sheaths. Those fibres which remain are not always completely unaltered, as described by Griesinger. Duchenne speaks of their transverse striation as unusually faint. Other observers have seen some which were striated longitudinally, some which were translucent or waxy, and some which were in a state of granular or fatty degeneration. Some fibres are even hypertrophied, having two or three times their normal thickness, as was first observed by Cohnheim.

Course.—The enlargement of the muscles, however, is not present at all stages of the affection. There is an early period during which the only symptom is an impairment of power in the lower limbs. The child—for pseudo-hypertrophic paralysis almost always begins in childhood—is noticed to totter in walking, and to be apt to fall; it has difficulty in getting on to its feet; it is particularly awkward in going up stairs; and when it tries to sit down, it falls into the chair. It may previously have been able to run about like other children. But in many cases the commencement of the disease occurs before the little patient has ever learned to walk; the proper age is passed without this being accomplished. At first the parents think that the child is only backward, as so many rachitic children are, but at length they see that there is something more seriously wrong.

Even when, at the end of a few months or a year from the beginning of the paralysis, the change in the muscles is discoverable, its extent and degree vary widely in different cases. The calves are often affected alone; next in liability to undergo enlargement are the masses of the *erector spinæ* in the loins, the *glutæi*, the *deltoids*, and the *infra spinati*. The muscles of the thigh, and the *pectorales* and *serrati magni*, are more often reduced in size, so as to

afford a strange contrast. But sometimes the whole of the trunk and all parts of the limbs display an exaggeration of contour which leaves the Farnese Hercules a long way behind, as Duchenne shows by comparing a sketch of that ancient statue with drawings of a patient of his, a boy, ten years old. The enlarged muscles are generally firm and elastic, and when they are brought into action they harden so that it is difficult to believe that they do not consist wholly of contractile elements. Leyden, indeed, speaks of them as doughy, and even as feeling semi-fluid when they contract; but I think that this is incorrect, at least in the majority of instances.

When a child affected with pseudo-hypertrophic paralysis stands up, its attitude is very peculiar. The abdomen is pushed forward, and the hollow of the lumbar vertebræ is greatly exaggerated. The nates project behind; but the shoulders are thrown much further backward still, so that a line carried downward from the upper dorsal spines falls behind the sacrum. The legs are separated widely from one another. If an attempt is made to straighten the back, the child at once falls down. When it walks, it balances the body from side to side at every step. It cannot rise from the sitting posture without the use of its hands.

The susceptibility of the affected muscles to faradic currents is sometimes normal, but in other cases it is considerably lowered. Fibrillary tremors, such as are seen in progressive muscular atrophy, are not generally to be noticed; but they have been observed in a few instances. The cutaneous sensibility is altogether unimpaired. The legs and feet are often cold and damp and bluish. The heels are usually drawn up, to a greater or less extent, by contraction of the *tendo-Achillis* on each side; and there may be a well-marked club foot. Sometimes the knees are rigidly flexed. The sphincters act naturally throughout the whole course of the disease. The intelligence may be perfect; but it is not rarely very defective, and in some of the cases recorded the patients have been idiots.

When the disease has developed itself to a certain point, it is described by Duchenne as remaining stationary for two or three years, or even longer. But at length a further advance takes place. If the legs only were affected, the arms are now involved. The muscles, which at this time lose their functions, however, never show even an apparent hypertrophy, but are always reduced in size, although the histological processes in them are of exactly the same kind as in those which were earlier affected. According to Duchenne, even those muscles which were at first enlarged ultimately shrink, until they too are obviously atrophied. The patient, who has generally now reached adolescence, becomes altogether unable to stand, or even to sit up; he is a prisoner upon the couch or the bed. Ultimately he dies of exhaustion, or is carried off by phthisis or some other intercurrent complaint. Friedreich, however, remarks that a large majority of the cases hitherto recorded have been lost sight of, so that the end of them is not known.

Very little has been learned as to the *ætiology* of pseudo-hypertrophic paralysis. The most conspicuous point in its history is its tendency to appear in succession in two, or three, or even four children of the same family, especially in the boys, and at the same age in all of them. As might be expected, the parents themselves have always been free from it; but its hereditary origin has often been traceable by its having occurred in brothers or other relations of the father, or even of the mother. Males are altogether far more subject to it than females, the proportion, among seventy-seven cases collected by Friedreich, being as sixty-four to thirteen. In a solitary series of cases recorded by Lutz it appeared in two successive generations in the female line only, affecting five individuals. On the other

hand, it has often attacked several sons of the same parents, and spared all the daughters. Out of 75 cases in which the date of its commencement was ascertained, Friedreich found 45 in which the children were under five years of age, 17 between six and ten, 8 between eleven and sixteen. In the remaining 5 cases the patients were adults; and 2 of them are said to have been aged forty and forty-one respectively when the disease began. As occasional exciting causes are mentioned unhealthy conditions of life, chills, over-exertion of the muscles, and the occurrence of convulsions or of an exanthem such as measles.

The *diagnosis* of the disease is very easy in well-marked cases. One morbid state which must be thought of in connection with it is that which Brodie and Hilton both believed to depend on the hip joints being congenitally placed too far backward; the drawings given in Hilton's work show a compensatory curvature of the spine very like that which is seen in pseudo-hypertrophic paralysis, so that one is inclined to wonder whether his cases could possibly, after all, have been examples of that disease in a slight form. That enlargement of the muscles is sometimes not a conspicuous symptom is, I think, certain; at least, unless Duchenne and Friedreich are wrong in including in the present category the cases which Dr. Meryon described in 1852, under the name of "granular and fatty degeneration of the voluntary muscles," and which occurred at about the right age in several boys belonging to two families. The objection may, indeed, be made, that Meryon found a different morbid change in the muscles; but it must be remembered that his investigations were made after death had occurred at a very advanced period of the disease, and not upon portions of muscle removed during life. Charcot and William Roberts, however, regard the cases in question as examples of progressive muscular atrophy. And it is to be noted that Friedreich himself relates as instances of the latter disease several cases which seem to belong to the same class as Meryon's. They occurred in three families residing in or near Heidelberg; and what is very curious is that Hemptenbacher, a pupil of Friedreich's, succeeded in tracing all three families to a single pair of ancestors a century and a half back. A similar complaint is said by Eichhorst to have appeared in six successive generations in a family at Königsberg.

It is in reference to the *pathology* of pseudo-hypertrophic paralysis that this question has its chief importance. Charcot and most other writers at the present time think that this disease is primarily an affection of the muscles themselves, and thus that it differs absolutely from progressive muscular atrophy, which has been traced to a lesion in the gray matter of the cord. Now, we have seen that in progressive muscular atrophy, and even in the atrophic paralysis of children, the wasted muscles often have their bulk made up to the natural standard by an interstitial development of adipose and fibrous tissues. But if, on the other hand, an apparent overgrowth of the muscles is not an essential feature of pseudo-hypertrophic paralysis, one cannot help suspecting that its relation to the other diseases must, after all, be very intimate. Indeed, although Pierret and Charcot studied with great care the histology of the cord in a fatal case of M. Bergeron's, and detected no morbid change in it, Dr. Lockhart Clarke and Dr. Gowers have since recorded a case in which they discovered extensive areas of disintegration in various parts, in some of which the nerve-cells were in a state of atrophy. Further observations are very much needed; but I strongly incline to the opinion that pseudo-hypertrophic paralysis will ultimately be found to be a spinal affection.

With regard to *treatment* there is unfortunately very little to be said. Duchenne claims the cure of two cases (the details of which are given in the "*Archives Générales*" for 1868) by faradization of the affected muscles,

with the aid of hydro-therapeutics and *massage;* they were both in an early stage, with but slight enlargement of the *gastrocnemii.* At a later period of the disease he found no treatment of any use. Benedikt has recorded three cases in which he obtained results by "galvanizing the sympathetic." But in the hands of Erb this procedure has since signally failed. Meryon considered that the administration of arsenic retarded the progress of his cases.

LOCOMOTOR ATAXY.

HISTORY AND NOMENCLATURE—SYMPTOMS AFFECTING MOVEMENT AND SENSATION—ITS RELATION TO PARAPLEGIA—ITS HISTOLOGY AND ANATOMICAL SEAT—PRODROMATA—LIGHTNING PAINS—ATROPHY OF JOINTS—VISCERAL ATTACKS—MYOSIS AND OTHER OCULAR SYMPTOMS—DISCUSSION OF THE RELATION BETWEEN THE ATAXY, DYSÆSTHESIA, AND OTHER SYMPTOMS—ÆTIOLOGY AND DIAGNOSIS: HEREDITARY CASES—TREATMENT.

Among the chronic affections of the spinal cord, there is one which is characterized essentially by an impairment of the power of co-ordinating or combining the actions of muscles in the execution of movements. That it is not an ordinary paralysis is evident from the fact that in some cases the contractile force of the muscles is undiminished. In England it is now commonly known as "locomotor ataxy," the name being derived from that of *ataxie locomotrice progressive*, given to it in 1858 by M. Duchenne, of Boulogne. But in Germany the most usual designation for it appears to be *tabes dorsalis*, and this title, being as old as Hippocrates, would be far preferable, provided that its claim to represent the disease in question could be satisfactorily established. Now, there is no doubt that Romberg and others had described under *tabes dorsalis* most of the principal symptoms of ataxy long before Duchenne wrote on the subject, and the French observer seems to have somewhat underrated the value of the work of those who had preceded him, although it is incorrect to say that he altogether ignored it. But, on the other hand, it is certain that *tabes dorsalis* had generally been used in a vague way for almost any very chronic and incomplete form of spinal paralysis, especially if it seemed to have been caused by sexual excesses and was attended with wasting of the muscles. Indeed, I cannot find that in this country the term ever had any precise signification. Moreover, it is only since the publication of Duchenne's observations that the attention of the profession has been kept fixed upon the disease, and its peculiar features were depicted by him far more accurately than by any previous writer. Consequently, although locomotor ataxy is a very clumsy name, I think we had better keep to it. To substitute tabes dorsalis is, at the present time, quite impracticable. It is worthy of notice that Todd—who in 1847 ("Cyclop. of Anat. and Phys.," iii, p. 721 R.) pointed out the distinction between paralysis and loss of the power of co-ordinating movements, and who mentioned the difficulty of walking and the tottering, uncertain gait, while considerable voluntary power remained—made no attempt to give any name to the latter affection. In one respect his knowledge went beyond the point reached by Duchenne, namely, in assigning the seat of the complaint to the posterior columns of the cord. It might be thought that if we could find a convenient English equivalent for the German *Hinterstrangsclerose*, there would be an advantage in adopting it; but such a change of name would, in reality, be very unfortunate, for, as we shall presently see, the lesion in question is very far from being so distinctive of locomotor ataxy as it was at one time supposed to be.

Symptoms.—Locomotor ataxy almost invariably begins in the lower limbs, and one of its most marked symptoms is an alteration in the gait. In walking, the patient lifts his foot high in the air and throws it outward, and then he brings the heel to the ground with a stamp. If the case is a slight one, it is especially in starting that he experiences a difficulty. Wilks relates that a gentleman whom he knew, if he stopped to look in at a shop window,

had to ask some one near him to give him a push before he could set off again. Once fairly started, such a patient may do pretty well, but perhaps he finds himself unable to turn round sharply without stumbling. Another of Wilks's patients, when he had once walked some distance along a road, wanted to go back, and was obliged to guide himself up against the bank, when he fell down. And yet another, an out-patient, one day apologized for being late at the hospital by saying that his friends had sent him off in a wrong direction, and that he went on until he fortunately met an acquaintance who turned him round! Most writers describe ataxic patients as rolling and staggering, like persons who are intoxicated, but Wilks says that the gait rather resembles that of a man on a ledge, who is anxiously balancing himself. It is, however, certain that many persons in the early stage of the disease have been condemned by their neighbors as drunkards. The attempt to run, or to hop, or even to ascend stairs, makes the patient's incapacity still more manifest, and he may be unable to stand on one foot, or with the two feet close together, without tottering and falling down. If told to walk in a straight line—as along one particular board in a floor—his course is most sinuous and irregular. The use of a stick, or leaning upon the arm of a friend, has an extraordinary effect in steadying his movements. In more advanced cases, however, as soon as he attempts to stand, his legs are violently jerked in all directions, and this, even though he may be held up on each side by an attendant. He may actually be unable to sit in an arm chair, the limbs being tossed about, so that he slides off on to the ground. When lying on a couch he cannot carry the foot straight toward an object so as to touch it, nor raise the leg up in the air with an even movement.

The complaint may last for years without extending beyond the lower limbs, but in many cases it at length affects the arms. The patient then becomes incapacitated for actions requiring delicate manipulation, such as writing or playing the piano; he cannot fasten his necktie or button his clothes. If he is told to bring the two forefingers into contact from a distance, they are carried about in the most irregular manner before they meet. He finds a difficulty in taking hold of anything held up before him, and when he attempts to draw or write in the air with his finger the figures are zigzagged in all directions.

Sensation is impaired to a greater or less extent in the vast majority of cases of locomotor ataxy. Subjective feelings of numbness, or of formication, are very often complained of. The patient says that when he stands his feet seem to be covered with thick woolen stockings, or to be treading upon a water-bed, or upon india-rubber. The cutaneous sensibility may be found diminished in the feet, the legs, or even the thighs, when carefully tested after the methods described at p. 353; but it very rarely happens that there is any approach to complete anæsthesia. Ability to feel pain is often absent, while tactile impressions are readily perceived. On the other hand, French writers point out that patients generally retain the power of appreciating differences of temperature long after they have lost common sensation. That curious symptom, the retarded transmission of impressions, is almost peculiar to locomotor ataxy. It especially concerns the sense of pain, but Hertzberg is said to have shown that tactile impressions and those of heat and cold may also be delayed to some extent. If there is no anæsthesia, the prick of a needle may first be only felt, and then, after an interval, a sensation of pain may follow. Erb says that the interval may be one of some minutes; Cruveilhier, who seems to have been the first to draw attention to the symptom in question, noted it at twenty or thirty seconds. Another point is that the pain produced by a slight injury may last longer than under normal circumstances and gradually culminate,

and, as a consequence, that the patient becomes unable to count a series of similar impressions if they succeed one another at all quickly. The paræsthesiæ which accompany locomotor ataxy are by no means limited to the lower limbs. Several writers insist on the frequency with which sensations of numbness are felt in those parts of the hands supplied by the ulnar nerves, even at the commencement of the disease, and Trousseau has pointed out that the mucous membrane of the mouth may be anæsthetic, the patient not being able to feel the food between his lips, nor to appreciate its temperature, nor with his teeth to distinguish hard substances from those which can be easily broken down.

A symptom on which Trousseau laid great stress, as indicative of locomotor ataxy, is the failure to execute movements as well with the eyes shut as when they are open. And there is no doubt that this symptom is often exceedingly well marked. Many a patient who can stand quite steadily so long as he is looking down at his feet totters and falls when he is made to close his eyes. It may even be the first indication that anything is amiss; as in the case recorded by Dr. Bazire, of a man who seems to have believed himself to be quite well until he noticed that he could not wash his face in the morning unless he was supported by another person, or could lean against a wall; for, as soon as he shut his eyes, he lost his balance, and staggered. And patients often complain that they have to watch every movement that they attempt to perform. According to Jaccoud, the effect of keeping the eyes open is in part due to the influence of light; for he says that a person who has ataxy cannot walk so well when they are closed as when they are open, even though something may be held in front of his chest in such a way as to prevent his seeing his feet. But, as Dr. Bazire has remarked, this is no doubt due to the feeling of confidence inspired by looking about one; for nervousness has a marked tendency to augment the symptoms of the disease, so that a patient who is conscious of being observed by others cannot walk nearly so well as when he supposes himself to be unnoticed. A similar explanation must, I imagine, be brought to bear upon the fact, noticed by Benedikt and Friedreich, that some ataxic patients who are perfectly blind, nevertheless, totter more when they are made to shut their eyes.

At present, however, it is the opinion of those best qualified to form a judgment in the matter, that the inability to execute movements accurately with the eyes closed is, strictly speaking, a sign, not of locomotor ataxy, but of defective sensation in the lower limbs. Erb says that he has found this symptom wanting in certain cases, namely, those in which sensation is absolutely unimpaired. On the other hand, it is invariably present when there is much anæsthesia. He admits, indeed, that it may often be noticed to some extent in cases in which there is no objective evidence of loss of sensation in the skin. But he accounts for this by supposing that it is the earliest indication of a failure of the so-called muscular sense. The obvious conclusion is that a patient suffering from any spinal disease may be expected to totter in attempting to stand with his eyes shut, if he has imperfect sensation in his legs and feet. There is no doubt that the want of stability is exaggerated and made more manifest by a defect of co ordinating power. But it must be clearly understood that, however valuable this symptom may be in the diagnosis of locomotor ataxy from rheumatism, neuralgia, or other peripheral affections of the limbs, it does not afford a criterion by which any one lesion of the spinal cord can be distinguished from the rest.

The fact is that the boundary line between paraplegia and ataxy requires very careful consideration. I have stated that in some instances of the latter affection the contractile power of the muscle is undiminished. This was proved by Duchenne, who invented the dynamometer for the

purpose. In the case of certain muscles, a rough estimate of their strength can easily be made. For example, one can pretty accurately measure that of the *quadriceps extensor* muscle, by getting an assistant to fix the lower part of the thigh, and then telling the patient (who should be lying on the opposite side) to keep the knee straight, while by grasping the malleoli one forcibly flexes it. Or one can test the force of the psoas and iliacus muscles by placing the man in a chair in the sitting posture, and making him lift his knee toward his chest, while at the same time one resists this movement with the hands placed over the lower end of the femur. Or, following Trousseau's plan, one may determine what weight the patient is able to bear upon his shoulders while standing with the support of a friend's arm or leaning on a piece of furniture. A young man under his care could support a hundred and sixty pounds in this way; and another patient was able to carry on his back a medical man who had imagined him to be paraplegic. But, while the theoretical importance of these observations is very great, as showing that locomotor ataxy may be altogether independent of paralysis, it is a great mistake to suppose that all, or nearly all, of the patients who exhibit a want of muscular co-ordination have their muscular power undiminished. On the contrary, one often finds it much enfeebled, and some of the earliest symptoms in these cases are commonly an inability to continue standing for any length of time, a sense of fatigue in walking even a short distance, and the like. It is true that the suggestion has been made that such sensations are really muscular paræsthesia, and do not prove a loss of motor power; and Duchenne records their presence in his patients in whom he demonstrated that the force of the muscles was unimpaired. But my experience is quite in accordance with that of Erb, who found an evident paresis in at least one-half of those cases in which the characteristic symptoms of locomotor ataxy existed.

A view which may be taken of such cases is that they are merely examples of an incomplete paraplegia, presenting a certain amount of failure of co-ordinating power, as a complication. This, I think, is the opinion held by Dr. Wilks; and it was, until recently, mine also. If one is right in supposing that a chronic inflammatory process in a particular region of the cord is the cause of ataxy, it seems reasonable to admit that this may coincide with, or even be secondary to, a similar process occurring in other regions; and at first sight it seems that mixed cases may in this way be satisfactorily accounted for. And it is a fact that most ataxic patients exhibit some other symptoms which also belong to ordinary myelitis, besides those that have already been mentioned. One of them is the curious "girdle feeling," described at p. 401. Trousseau speaks of some of his patients as feeling as if the chest, the arms, or the legs were compressed by an india-rubber cuirass. Or the sensation may be that of a belt constricting the abdomen, of a garter tied below the knee, or of a tight shoe pressing on the foot. Again, if we inquire how the functions of the pelvic viscera are carried on in ataxy we shall find but little to distinguish it from the partial forms of paraplegia. Thus the bladder is apt to be irritable, and its muscular power may to some extent be diminished, so that the urine no longer flows in a good stream, or is from time to time passed into the bed. Erb notes that the fæces are sometimes discharged without the patient's knowledge, and attributes this to a loss of sensibility in the mucous membrane at the anus. The sexual functions are almost always impaired. At an early state the genital organs are often extremely irritable; it is said that this condition may be accompanied with an increased virile power, but much more frequently there is marked weakness. As the diseases advances, the patient generally becomes altogether impotent. I believe, however, that there is no doubt that some persons have been able

to procreate children after having been for years the victims of pronounced ataxy. The reflex excitability of the cord to cutaneous stimuli is almost always present, although in very variable degrees—as, indeed, is the case in healthy individuals. On the other hand, recent observations by Westphal and Erb appear to show that the susceptibility to tendon reflexes is from the earliest period of the disease invariably extinguished, which is far from being the case in paraplegia. The electrical irritability of the muscles is often perfect, but sometimes slightly augmented or impaired. They remain well nourished until the disease has reached an advanced period in its course; but this is likewise the case in paraplegia, unless the lumbar part of the cord is destroyed. Ultimately many ataxic patients pass into a condition of complete paralysis with atrophy of the muscles, bed sores, absolute failure of the bladder, and cystitis.

The leading French and German observers, however, seem to think that in the mixed cases of ataxy and partial paraplegia the disorder of co-ordination is the fundamental and essential affection, while the impairment of muscular force is secondary and accidental. Their reasons appear to be partly based upon morbid anatomy, partly upon clinical observation.

Anatomy.—I have already alluded to the fact that a sclerosis of the posterior columns of the cord is found after death from ataxy. The current description of this change is that it is visible as a gray or grayish-yellow streak on the surface on each side of the posterior median fissure; in transverse sections it is most marked in the upper lumbar and dorsal regions, and diminishes in extent both above and below. Sometimes it reaches upward as high as the restiform bodies. Its histological characters are those which have already been given at p. 398. In the more advanced stages most of the nerve fibres have disappeared, their axis cylinders being no longer discoverable; but even in the oldest cases a not inconsiderable number of them are found with their structure unaltered, scattered through the dense connective tissue. The posterior roots are thin, gray, and atrophic. The pia mater at the back of the cord is generally opaque, thickened, and more or less adherent to the other membranes. There have been many speculations as to the mode of origin and significance of this affection. Do the nervous elements first undergo degeneration from over-use or over-excitation, and is the growth of connective tissue the result of a secondary reactive process? Or is the original morbid change a chronic inflammation of the neuroglia, leading afterward to an atrophy of the nerve fibres? Or does the disease begin as a meningitis, and spread to the cord? Or is its starting point in the sensitive nerve roots? Remak and others have taught that the pathology of ataxy varies in different cases, and that there are corresponding variations in its causes, and even in its early symptoms. But some observers have doubted whether, after all, sclerosis of the posterior columns is constantly found. Trousseau refers to a case, of twelve years' duration, which had been seen by Duchenne and accepted as typical, and in which Gubler and Luys and Duchenne examined the cord and its posterior roots, and could detect no alteration in them, whereas many of the nerve fibres had disappeared from the anterior roots. The importance of such negative evidence seems to be greatly enhanced by the fact that the course of the disease is commonly so protracted as to give one scarcely any chance of tracing it to a fatal termination in ordinary hospital practice. On the other hand, a precisely similar change in the posterior columns is frequently seen as the secondary result of a local myelitis, without any symptoms of ataxy having been present. In cases of Pott's disease of the dorsal vertebræ, for example, such an "ascending degeneration" often exists in the cervical part of the cord, but the co-ordination of the movements of the arms and hands is in no way affected by it.

The solution of all these difficulties seems to have been recently discovered by Pierret. The change hitherto described is limited to the internal fasciculi of the posterior columns, the so-called tracts of Goll. Now he finds that in ataxy these parts of the cord are affected in the cervical region, when the upper limbs had escaped, as much as when they were involved in the disease. On the other hand, a similar morbid change in the external fasciculi of the cervical posterior columns, the *bandelettes externes*, or *fasciculi cuneati*, occurs only when there have been irregular movements of the hands and arms, and it is more marked on that side of the cord corresponding to the limb which was the more disordered in its actions. The conclusion is obvious, that in ataxy, as in other diseases of the cord, sclerosis of the tracts of Goll is, after all, an "ascending degeneration," and has nothing to do with the symptoms, and that the essential lesion has its seat in the outer parts of the posterior columns. Pierret even relates a case in which a sclerosis limited to the *fasciculi cuneati* was found in the dorso-lumbar region, the patient having died of hæmatamesis after displaying some of the early symptoms of ataxy. In transverse sections the sclerosis is seen as two narrow, gray streaks, one lying behind each of the posterior cornua of cineritious matter, and parallel with it. It is not obvious to the naked eye, nor until the cord has been hardened, and as the secondary change in the tracts of Goll is sometimes wanting even in advanced cases, one can understand how it has come to pass that some good observers should have declared the cord healthy, when locomotor ataxy had been present during life.

It is evident that to prove the constant presence of sclerosis of the *fasciculi cuneati* as the initial lesion of locomotor ataxy, present in every case, whether complicated or not with paralytic symptoms—an extended series of observations is needed, such as can be accumulated only in the course of a very long time. But in the meanwhile there are clinical grounds for placing under a single head all cases in which there is an impaired co-ordination of movements. One is the fact that, so far as I can ascertain, this symptom always shows itself as soon as there is any discoverable loss of power, if not still earlier. I do not know of any instance in which a patient has been admitted for partial paraplegia, unaccompanied with indications of ataxy, and in which such indications have subsequently been observed.

Prodromata.—Another clinical feature of the disease, which goes far toward establishing its claim to be admitted in its entirety as an independent member of the nosology, is the frequency with which it is attended with certain remarkable early symptoms, or, as they are often termed, "prodromata," belonging to no other spinal affection. Chief among these are *pains* in the lower limbs or elsewhere. These may either be of a stabbing or boring character, as though a sharp instrument were thrust into the tissues at the spot, which is generally near a joint; or they may be like flashes of lightning or electric shocks shooting down along the course of a nerve. Charcot says that they are worse at night. They last but an instant and return again and again during a period of from four to eight days, after which they may disappear altogether for a fortnight, or even for some months. Their occurrence seems to be favored by changes of weather. Erb remarks that he has often been struck by the unanimity with which different patients have complained of them on some particular day when there has been much wind, or a fall of snow or of rain. It is also said that they are especially liable to return at spring and autumn, and that they may be brought on by over-exertion of body, mental emotions, or the excitement of coitus. They are sometimes of the most agonizing character, sometimes so slight that careful inquiries are needed to elicit the fact of their presence. They are commonly set down as neuralgic or rheumatic. They frequently spread from the legs to the trunk, much more rarely to the arms. They may go on

for five, ten, or fifteen years, before any other symptoms show themselves. They often persist throughout the whole course of the disease. As to the frequency of their occurrence, Topinard found that they were wanting in only 22 cases out of 104; Erb in only 8 out of 60; Cyon that among 203 patients there were 138 in whom they were said to have been present, but no more than 8 in whom they were expressly stated to have been absent.

Another most curious early symptom of ataxy is a *joint affection*, which has been specially studied by Charcot. Its most common seat is the knee, next in frequency comes the shoulder, and then the elbow, the hip, the wrist. It is not traceable to any blow or other injury. It sets in suddenly with extreme swelling; there being not only effusion into the synovial cavity, but also a brawny infiltration of the parts around. Yet there is generally neither pain, nor heat, nor redness. In the course of a few months it may entirely subside, but sometimes it ends in the destruction of the articular cartilages, with erosion of the ends of the bones and a partial dislocation of their surfaces. Charcot says that it never occurs at an advanced period of the disease, except in the upper limbs, when they are beginning to be affected in their turn. He styles it "arthropathie ataxique" or "tabetique."

Yet other prodromata mentioned by Charcot are the following: a pain in the bladder or in the urethra, with a constant desire to micturate, a sudden pain in the rectum, and certain strange attacks of stomach disorder, which he terms *crises gastriques*, and which he says were first noticed by Delamare in 1866. He describes them as consisting of lightning-like pains, which start from the groins, and pass up both sides of the abdomen, toward the shoulders. They are generally accompanied by palpitation of the heart and a quick pulse, by vomiting (of a liquid at first clear but afterward stained by bile or blood), and by giddiness and malaise. Such an attack commonly lasts for two or three days at a time, and then passes off, leaving the stomach free to perform its functions normally.

But most remarkable of all are what Charcot terms the *cephalic* symptoms of locomotor ataxy, principally affections of some of the ocular muscles and of the eyesight. The functions of one or another of the branches of the third nerve on one side, or of the whole of the sixth nerve, may be more or less completely annulled; there being diplopia, strabismus, and various other phenomena, such as have been described (p. 332). Von Graefe is said to have remarked that in ataxic patients, when they have double vision, there is particularly little tendency to fusion of the images, and that this points to a central origin. The paralysis may be quite transitory, or lasting only a few days, or it may persist for weeks or months; it may return again and again; at an advanced period of the disease it may even be permanent. Another common symptom is an inequality of the pupils, one of them being constantly smaller than the other. Still more often both irides are symmetrically contracted to an extreme degree; and then the *myosis* is described as presenting "spinal" characters, that is, the pupils are insensible to the stimulus of light, but contract with accommodation for near objects. This condition is generally persistent. Its recognition is due to Mr. Argyll Robertson, of Edinburgh.

With or without some of the symptoms just enumerated, impairment of vision is frequently noticed. This is said to develop itself as a progressive narrowing of the visual field, generally from without inward, until the only part of the retina that remains sensitive to light may be a small patch to the inner end of the blind spots. It is accompanied by achromatopsy; the perception of green being generally the first to disappear, then that of red, and lastly that of yellow and of blue. There is often an increased sensitiveness to bright light, so that the patient sees better after sunset. The pupils are often contracted and motionless, even when there is nearly complete

blindness. The ophthalmoscope reveals atrophy of the disc, which in advanced stages is perfectly white, with sharply-outlined edge, and very small arteries, whereas at an earlier period it may be grayish in color. The two retinæ generally suffer together; the affection is a progressive one, and commonly goes on until there is complete amaurosis.

As to the significance of those cephalic symptoms, the occurrence of which in a spinal disease has, of course, attracted unusual attention, I shall have something to say further on. Their frequency appears to be very considerable. Erb estimates that, if even slight and transitory phenomena are reckoned up with the rest, affections of the ocular muscles are present in more than half of all the cases of ataxy; he thinks that permanent, or at least persistent, affections of them exist in one-third or one-fifth. How often the optic discs undergo atrophy cannot yet be stated accurately; ophthalmologists are apt to rate it too high, physicians below the truth. Erb observed it only 8 times in about 70 cases; Topinard, however, found visual disturbances in 49 of 102 cases; and Cyon, amblyopia or amaurosis in 60 of 203 cases. It often occurs at a very early period; according to Charcot it may precede all other symptoms of ataxy by ten years.

Nature of Ataxia.—If it be admitted that the facts brought forward in the last few paragraphs justify us in collecting together, under the name of locomotor ataxy, all those cases in which there is impairment of co-ordination, whether attended or not with loss of muscular power, we may next pass on to consider in what way the several symptoms of the disease are related to one another. Now, I have already remarked that an inability to stand, or to walk steadily with the eyes shut, is no longer to be enumerated among its distinctive features, being no more than an affect of anæsthesia of the lower limbs. A precisely similar doctrine, however, is held by some authorities with regard to the failure of co-ordination itself. Basing his views upon the acknowledged fact that guiding sensations contribute to the due execution of movements, Leyden maintains that ataxy is merely a consequence of interruption in the transmission of such sensations along the cord.

Now, it must be admitted that some plausible arguments can be brought forward in favor of the doctrine in question. One is that the most obvious lesion is seated in a part of the cord which is (or was) believed by physiologists to convey afferent impulses. Another is the frequency with which a more or less considerable degree of anæsthesia is present, even at an early period. And, if there are cases in which cutaneous sensibility appears to be perfect, it may be replied that in many of them the sensibility of the muscles and tendons and joints has not been tested, and that this is no less essential for the even and regular execution of muscular movements.

The recent discovery that it is impossible to elicit tendon reflexes, even at the commencement of the disease, may be pressed into the same argument, as being capable of interpretation on the view that there is a special impairment of the transmission upward of afferent impulses starting from the deeper tissues.

Nevertheless, it would seem that there are facts which make it impossible for us to accept Leyden's views. One is that some cases, at least, of ataxy are unattended with any discoverable anæsthesia or defect of muscular sensibility. Both Friedreich and Erb speak positively about this. Again, there is no close correspondence in individual patients between the degree of impairment of sensation and that of disorder of co-ordination. Lastly, there may be absolute anæsthesia without the slightest irregularity in the movements of the limbs. A case in point was recorded by Späth, in 1864, and was ten years later followed to an autopsy by Schüppel. The patient, whose name was Remigius Leins, had for more than twenty years lost

sensation in his arms and hands, and for six years in his feet. He was quite unconscious of the position of his limbs; and when they were moved by another person he knew nothing of it. Yet he could walk steadily, and without support, and tolerably fast. When asked to raise his foot to a certain height with his eyes shut, he did it with a perfectly regular and even movement. After death the whole length of the cord was found excavated in the centre; the posterior columns in the cervical region were completely destroyed; the anterior columns were everywhere normal. It is particularly to be noticed that in this instance the muscular sensibility was absent, as well as that of the skin.

If, then, ataxy is not due to any interruption of afferent or sensory impulses, how is it caused by a lesion situated in the posterior columns? Most writers seem to suppose that these columns may possess the special function of conveying impulses from a co-ordinating centre above, in the medulla oblongata or pons, to the motor nuclei in the cord. But I think it is clear that co-ordination, instead of being affected by a single centre, must require a very complicated apparatus, probably consisting of several parts situated in different regions of the cerebro-spinal axis. We have seen that the reflex movements which are performed by the spinal cord independently of the brain are never entirely inco-ordinated, and that sometimes they are co-ordinated in a very complex way. One can hardly doubt that the machinery which is brought into operation under such circumstances, is also made use of when similar movements are excited by the will. Again, I fail to conceive why there should be any channels for the conveyance of motor volitional impulses, apart from those which pass through, or are connected with, any centres for higher co-ordination that only exist in the medulla oblongata or elsewhere in the brain. To whatever extent the movements of the trunk and limbs are co-ordinated above the spinal cord, to that extent surely must the motor tracts of the cord itself transmit impulses themselves already co-ordinated.

Moreover, we must remember that what is observed in ataxy is by no means an entire absence of co-ordination, such as would occur if the volitional impulses passed straight down to an individual muscle or to a group of muscles, without relation to any others. On the contrary, it is probable that the failure is very often of the slightest possible kind. Scarcely appreciable irregularities in the force which the muscles severally exert, or even in the intervals of time at which they are increasingly brought into action, would account for all the phenomena presented by some well-marked instances of the disease. No wonder, then, that lesions of the proper motor tracts of the cord do not cause ataxy. We should rather expect it to be due to some affection just touching the outskirts of these tracts so as to injure them only very slightly. A sclerosis commencing in the fasciculi cuneati of the posterior columns seems to be just such a lesion as might produce this effect.

Again, it is certain that even in the earliest stage of the disease other parts besides the posterior columns must be involved. In no other way can one explain many of the so-called prodromata. What is to be said as to the origin of the atrophy of the optic discs? Pathologically, the morbid change in them is identical with that which is going on in the cord itself. But why should it occur in cases of locomotor ataxy, and not in those of diffused simple myelitis? And, further, why should amaurosis from this cause often precede all spinal symptoms by an interval of several years, so that Charcot actually finds grounds for believing that the majority of the female patients admitted into the Sâlpetrière for simple atrophy of the discs become sooner or later the victims of ataxy? The affections of the ocular muscles are supposed by Erb to be due to slight lesions of the nerve roots or of their nuclei within the medulla oblongata, but he does not attempt to show how it is

that any motor structures are attacked, nor why the nerves concerned in moving the eyeballs suffer more than any others. As regards the last point, indeed, I think I see an explanation in the delicate way in which the muscles in question are naturally balanced against one another. I have already pointed this out at p. 333, and it necessarily renders the slightest impairment of the power of any one of them at once obvious to both the physician and the patient. The same degree of paresis elsewhere would be altogether incapable of recognition, but it is said that an actual paralysis of the facial muscles, or of those concerned in mastication, has sometimes been observed.

The joint affection which is now and then seen among the prodromata of locomotor ataxy is believed to be generally dependent upon an atrophic change in the cells of the gray matter of the anterior cornua. Joffroy and Westphal are said to have made observations which directly confirmed this opinion.

On the other hand, the boring and flashing pains are doubtless dependent upon the morbid changes in the fasciculi cuneati of the posterior columns, or in the nerve roots immediately adjacent to them, but it seems to be doubtful whether the presence of a high degree of anæsthesia can be accounted for without the supposition that the posterior cornua are also involved. There is experimental evidence which suggests that a retardation in the transmission of sensory impulses would be especially likely to occur if this were the case; and, pathologically, a dark-gray shrunken condition of the posterior cornua has repeatedly been observed in cases of ataxy.

There is, indeed, abundant proof that in the more advanced stages of the disease, the morbid process may extend in all directions within the substance of the cord. Changes in the peripheral parts of the lateral columns were long ago noticed by German observers, and doubtless give rise to the paralytic symptoms which are so commonly present. Charcot even says that the nerve cells of the anterior gray cornua may be destroyed in such a way as to cause an atrophic change in the corresponding muscles. When there has been an absolute loss of power in the lower limbs, almost the whole thickness of the cord may be wasted, tough, gray and transparent looking.

The fact that even the prodromata of locomotor ataxy can be accounted for only on the supposition that changes exist in structures far distant from the posterior columns of the cord, makes it surprising that a definite boundary line should be traceable between it and other spinal affections. As in multiple sclerosis (*v. infra*), it seems that the clinical features of the disease are far more uniform than would *a priori* have been anticipated by the morbid anatomist. Charcot, indeed, admits that pains—which he says are precisely like those that occur in the early stage of ataxy, and which he believes to be really due to a sclerosis of the lateral columns—may be observed in disseminated sclerosis, in general paralysis, in chronic alcoholism, and even in simple myelitis and in compression paraplegia. But this is not going very far toward the recognition of a *secondary* locomotor ataxy. And I think we can now understand how it is that the lesions which give rise to even the partial form of paraplegia are too coarse and too diffused to be capable of producing the slight and delicate disturbance of function which seems to be characteristic of the complaint now under consideration.

Causes.—With regard to the ætiology of locomotor ataxy there is still much uncertainty. In many cases no cause is discoverable. Sometimes it has followed an attack of fever, acute rheumatism or pneumonia, sometimes it has taken the place of paraplegia in sequence of diphtheria, as in cases recorded by Jaccoud and Erb. Syphilis has been said by some writers to

give rise to it rather frequently, but this seems to be still a matter of doubt.* Erb quotes E. Schulze as having ascribed it to injuries, not merely to such as affect the spine, but even to a fracture of the thigh, or a fall on the abdomen. In some cases it appears to be pretty clearly traceable to a severe chill, as from falling into water, or sleeping on damp ground, to over-fatigue, as in soldiers after forced marching; to sexual excesses, or onanism; to prolonged mental anxiety or distress; perhaps even to excessive indulgence in tobacco. The various wars of the present century seem to have given rise to numerous cases of ataxy among those who have been engaged in them; it is difficult to say exactly how campaigning sets up the affection, but possibly by the combined operation of several of the causes already mentioned. The greater liability of men between the ages of thirty and fifty to these various morbid influences is doubtless the reason why such persons are especially apt to suffer from ataxy; as, indeed, from several other serious nervous diseases. The preponderance of males over females is very marked, but the proportion is variously stated by different observers at 8 to 1 (Erb), 3½ to 1 (Cyon), 2½ to 1 (Carré, Schulze). Ataxy very rarely occurs in persons under twenty years old, or begins in those who have passed the age of fifty. The case of an old gentleman of eighty is mentioned by Trousseau; but it is not stated how long he had been a sufferer. This writer gives some instances in which there was a marked family neuropathic disposition. One patient had an uncle and an aunt insane, a brother ataxic, and a brother hemiplegic; another patient was the son of a man who committed suicide, and two of his sons labored under certain peculiar nervous affections. Erb, however, says that he has seen many cases in which no predisposition to nervous diseases could be traced. Carré has recorded the direct transmission of ataxy by inheritance in a family of which the grandmother, the mother, her seven children, and eight others of her relations were all attacked. But such instances are very rare; and, as we shall presently see, it is possible that they may hereafter be found to constitute an independent malady.

From the account which has been given of the early symptoms of locomotor ataxy, it must be evident that its *diagnosis* at that period of the disease is often a matter of extreme delicacy. Probably there is scarcely a case in which the premonitory pains are not for a length of time dismissed as neuralgic or rheumatic. Every medical man is sure to be consulted at one time or another for pains in the limbs by patients who are to become the victims of ataxy. In many cases of this kind a careful investigation will elicit other indications of it. There may be a slight squint, a contracted state of the pupils, or a failure of tendon reflexes; or the patient may totter a little when told to walk, or to stand with his feet close together, while keeping his eyes shut. This last is a most valuable sign when the doubt is as to the presence of any spinal affection, notwithstanding that it is almost without significance as between ataxy and other diseases of the cord. If no corroborative evidence can be discovered, one can only wait before giving an opinion as to the nature of the case.

When the complaint is fully developed its diagnosis is generally easy. Pronounced paralytic symptoms, with wasting of muscles, may sometimes cause one to overlook the prior existence of an ataxy, and to concentrate one's attention on the lesions in the motor regions of the cord; but such a mistake can hardly be deemed very serious. Again, when there is "multiple sclerosis," and when the posterior columns are also attacked in a marked way, it may be impossible to assign the case to one affection rather than to the other. And in a future chapter I shall have to point out that locomotor ataxy not infrequently occurs in association with "general paralysis of the insane," in such a way as to have now and then caused

* See on this point "*Trans. Internat. Med. Congr.*" 1881, pp. 32–42. Also Dr. Gowers, in the "*Lancet*," Jan., 1881.

considerable confusion in the minds of those who have happened to be more familiar with one than with the other of these two diseases.

The failure of co-ordination which accompanies disease of the cerebellum does not seem to be likely to be mistaken for the complaint with which we are now concerned. It may generally be distinguished at once by the absence of other spinal symptoms, and by the presence of headache, giddiness, vomiting, optic neuritis, epileptiform attacks, and the various other signs of an encephalic lesion. And, so far as I can gather from the statements which have been published, the so-called cerebellar ataxy is not attended with the peculiar mode of walking described at p. 469, but merely with a rolling, tumbling gait, exactly that of a drunken man.

It is, however, doubtful whether at the present time the definition of locomotor ataxy does not include some cases which will hereafter be found to be distinct. Friedreich has described an *hereditary* form of the disease which presents several peculiar features. It has been recognized as yet in only three families; in each of them it has attacked in succession several children of the same parents, in all nine patients, of whom seven were girls. It has always begun at or near the age of puberty, between the thirteenth and the eighteenth years. It has been attended with remarkably little disorder of sensibility, the prodromal pains in the limbs and the later girdle sensation round the body have been absent or very little marked, and there has been scarcely any anæsthesia or none at all. The upper limbs have displayed a loss of co-ordination at an earlier period than usual. A peculiar form of nystagmus has been present in five cases; it is especially characterized by occurring only when the patient endeavors to fix his eyes upon an object before him; the movements are always bilateral, and are comparatively slow, being repeated about two or three times in a second. In every instance there has been a remarkable disorder of speech. The utterance is described as having been at first slightly lisping (Calloud), then as irregularly interrupted and stammering, and finally as almost unintelligible. It is altogether unlike the slow, accentuated articulation of a patient suffering from multiple sclerosis. The duration of these cases has been very long, in one instance it was more than thirty-two years; a curious circumstance is that no fewer than five of the patients died of enteric fever. Sclerosis of the posterior columns has been found in every case in which a post-mortem examination has been made. I have at present under observation a typical example of Friedreich's ataxy. The patient, a medical man, was first noticed to be uncertain in his gait when about sixteen years old. He was one day walking across a plank in a house that was being built, when he tottered; afterward he remembers that he had to be careful not to go near the edge of the path where it was raised above the road. But he came to the hospital as a student, passed his examinations, went into practice, and took a wife. At the age of about twenty-five he was obliged to give up his profession on account of an increasing inability to walk or ride; he became unable to write, and his speech became labored and thick. In 1878, when he was thirty, he could just walk from one room to another with assistance; there was even then but little impairment of sensation in his feet; when he moved his lips in speaking, a large number of the facial muscles were thrown into action, so that the mouth became drawn outward into a meaningless smile; the words were tolerably distinct, but some syllables were slurred over and others pronounced too strongly. The nystagmus was very obvious when he was told to direct his eyes to either side. He is a member of a highly neurotic family, and one sister suffers from some form of paralysis, but whether her case is like his I do not know.

Again, Wilks says that all the female patients, three in number, whom he has seen with symptoms of ataxy, in each of whom sexual excesses

or other causes of exhaustion had been in operation, recovered under tonics and the application of galvanism to the spine. He suggests that in such cases the disorder is functional or a neurosis, and I think that there can be no doubt of the correctness of this opinion. For when locomotor ataxy is dependent on sclerosis of the posterior columns, the prognosis is of a very grave character. Erb, indeed, says that it is not quite so bad as in cases of multiple sclerosis or of simple transverse myelitis; but this means little more than that the disease advances at a comparatively slow rate, for it very rarely subsides entirely. As a rule, its duration varies between six and twelve years, and it may last twenty or thirty years. Some writers have described an acute form, and I remember one case which was so diagnosed in a woman who died in hospital six weeks after admission, and about ten months from the beginning of her illness. But in this instance there was softening of the anterior columns in the lumbar region, and it now seems probable that the marked gray change found in the posterior columns was only a secondary degeneration. The course of the disease is not always steadily progressive; it often becomes quiescent at a certain stage, and remains so for a very long period. Generally, it is better in the summer than in the winter. Sometimes it scarcely seems to shorten the patient's life.

Treatment.—When ataxy is fully developed, it rarely yields to treatment of any kind. Erb, however, mentions two cases, in each of which almost complete recovery took place. One patient afterward held a Government appointment for several years; the other, who had been unable to get about without help, and had had incontinence of urine with cystitis, regained the power of walking for three or four hours at a time, and that of holding his water for five or six hours; he married, and he took the command of an ironclad frigate. The treatment which Erb recommends most strongly consists in galvanism applied as a continuous current to the spine. He uses a moderate number of cells, for three to six minutes at a time, once daily. Of 66 cases thus treated, 25 received no benefit, 41 were more or less improved. In most cases he also galvanizes the peripheral nerves.

The medicine which appears to be of most value is the nitrate of silver; it often fails entirely, but sometimes does a great deal of good. I have generally ordered about a quarter of a grain three times a day. Ergot, belladonna, phosphorus, arsenic, iodide of potassium, bromide of potassium, all seem to be useless. Erb says that it is wrong to prescribe strychnine.

The general management of the patient is of great importance. He must be carefully protected from cold and damp by flannel under clothing. He must avoid all bodily and mental exertion. Dr. Radcliffe thinks it advisable that crutches should be used in the early stages, so as to save the lower limbs as much as possible. It has been suggested that maintaining absolute rest in bed might favor the subsidence of the disease, but I do not know of any positive evidence in favor of this plan.

Locomotor ataxy is among those affections for which persons resort to the various spas. Almost all writers are agreed that hot baths should be avoided. Erb says that any temperature above 58·5° F. may be injurious, and that the patient should bathe only once in two or three days, and remain in the water not above fifteen or twenty minutes. Nauheim and Rehme are the places which seem at present to have the highest reputations. Peat-baths are recommended. But the figures given by Erb appear to show that the cold-water cure, with packings, frictions and douches, is more serviceable: of 19 patients treated in this way, no fewer than 16 were benefited.

Among the *symptoms* of ataxy the pains most need relief. Sinapisms, blisters, liniments of chloroform or belladonna, veratria ointment, opium or belladonna plasters, may be of some use; but often one cannot avoid frequent injection of morphia.

DISSEMINATED SCLEROSIS.

HISTORY—ANATOMY—SYMPTOMS AND COURSE—ÆTIOLOGY—DIAGNOSIS.

In the great illustrated work of Cruveilhier, published between 1835 and 1842, there are figures taken from four different patients, representing the spinal cord affected with what he termed "gray degeneration," in the form of patches scattered irregularly through its substance. Afterward various writers from time to time referred to similar cases, but it was not until about the year 1866 that a series of observations were made at the Sâlpetrière, from which it results that the disease in question is characterized clinically by a remarkable and definite group of symptoms, so that it can often be diagnosed with certainty. In these researches Charcot took the principal part. The subject was soon taken up in Germany, by Leo and others, but in England it seems to have been altogether neglected until, in 1873, Dr. Moxon published a case in the "*Lancet*," and in 1875 a paper containing eight cases in the "*Guy's Hospital Reports*."

Cruveilhier's drawings show that the bulb and the pons are affected as well as the cord, and the same morbid change has since been found in the hemispheres. I might, therefore, with equal justice have placed it among diseases of the brain; indeed, several symptoms belong to it as a cerebral affection. But, on the whole, this appears to be the best place for it. By French writers this disease is called "Sclérose en plaques disséminées;" the Germans term it "multiple Sclerose," "multiple Herd-Sclerose," "Inselförmige Sclerose;" in this country Dr. Moxon proposed the term "Insular Sclerosis," but that of "Disseminated Sclerosis" seems to be preferable.

As a rule, the morbid change is at once obvious when the nervous centres are examined in the post-mortem room. Bourneville has, however, recorded one case in which they appeared to be healthy until the microscope was used. Some writers have wondered that so remarkable a disease so long remained unknown, but its slow and hopeless course renders it little likely to go on to its fatal termination within the wards of ordinary hospitals. I do not find a single case which can be assigned to disseminated sclerosis in our pathological records between 1854 and 1873.

Histology.—The patches of sclerosis are rounded or elliptical or very irregular in form. In the cord they come to the surface, and are sometimes seen through the pia mater. In the brain they scarcely ever penetrate into the cineritious substance of the cortex, so that they are visible only at the base. However, in the cord, the pons and the basal ganglia, they show no tendency to spare the gray matter. They are often scattered in large numbers throughout the white matter of the hemispheres. The cerebellum seldom contains many of them. It has been said that they are generally more numerous in the antero-lateral columns of the cord than in the posterior columns, but this appears not to be the case. A similar change may be present in the olfactory bulbs and in the roots of the various cranial and spinal nerves. In the peripheral trunk it has not yet been recognized. Dr. Moxon found the brachial plexus in one case normal on each side.

The patches generally feel hard, but recent ones are said to be softer than the rest. These are also of a deeper gray tint, whereas the older ones are yellowish gray and less translucent. They become slightly pinkish when exposed to the air. They may either project slightly above the cut surface, or lie at the same level, or be slightly depressed. They vary from a microscopic minuteness up to the size of a hazel, or more. Histologically they present the appearances described at p. 398, as belonging to chronic myelitis generally. Charcot says that there are fewer corpora amylacea, and that a distinction is afforded by the way in which naked axis cylinders persist even when the neuroglia has become highly fibrillated; but the last point is contested by Erb. It is to be observed that in thin sections the affected parts are found not to be sharply defined at their edges as they appear to the naked eye; on the contrary, the sclerosis fades off very gradually into the healthy tissue. Dr. Moxon remarks that granule cells are more numerous at the circumference of the patches, and in the apparently normal brain substance beyond, than toward their centres.

Symptoms.—That the clinical features of disseminated sclerosis should vary in different cases, is no more than we should expect from the irregular distribution of the patches, which would account for the utmost possible irregularity in the symptoms. Charcot, indeed, speaks of it as, *par excellence*, polymorphous. He and other French writers describe, in addition to the "cerebro-spinal" form of the disease, separate "spinal" and "cerebral" forms, but it is admitted that such distinctions are scarcely ever needed at the bedside, and, as a matter of fact, the symptoms present a degree of uniformity which seems to me to be very remarkable. The only writer, so far as I know, who attempts an explanation of it is Dr. Moxon, and he regards it as "*a constant average result* of the numerous points of disease." But although this might account for the production of any common symptom —such as some diffused form of paralysis—I cannot see how it meets the present difficulty, for the affection is characterized by certain definite phenomena which are peculiar to it, and belong to no other complaint, and surely must, in some way, depend on a localization of the morbid change in particular areas within the nervous centres. I have already remarked that in being thus attended with symptoms more special than could *a priori* have been anticipated by the morbid anatomist, it resembles locomotor ataxy, although with a certain difference.

Chief among these symptoms is a sort of *tremor*, which, until recently, was confounded with paralysis agitans. There are, in fact, records of two cases diagnosed as examples of that affection by Skoda and by Hasse respectively, in which patches of sclerosis in the nervous centres were discovered after death. Careful observation, however, has shown that in paralysis agitans the trembling movement is less extensive, more regular, more rhythmical, more slow than in disseminated sclerosis. In the latter disease, when the hand is raised the oscillations increase in amplitude as it gets further from the side, until they greatly embarrass and disturb the patient. If he is asked to carry a cup to his lips it becomes more and more violently shaken, until at last it is dashed against the teeth, so as, perhaps, to spill its contents. In slight movements the tremor is not so marked; the handwriting, although shaky, long remains legible. Still more important is the fact that there is no agitation of the muscles as long as they are at rest and supported. When the patient is lying in bed one could not tell that anything is the matter, but as soon as he is asked to sit up, the arms, and then the head and the neck, begin to oscillate backward and forward. When he is resting in a chair the arms are quiet, but the head may still continue to show a slight tremulous movement. If he attempts to get on his feet the whole trunk and all the limbs become violently shaken, so that in severe cases standing is out

of the question. Mental emotion or excitement aggravates the tremor considerably. According to Erb, there are some cases in which it continues even when the patient is lying still, but this is altogether exceptional.

Charcot has suggested that this remarkable symptom depends upon the persistence of naked axis cylinders in the parts affected with the sclerosis. He conceives that the transmission of volitional impulses may become interrupted and, as it were, jerking; but such a view appears but little likely to be correct. Adenstine is disposed to think that the tremor depends in some way upon the presence of patches in the pons and still higher in the motor tracts, and Erb says that an analysis of twenty-two recent cases tends to support this notion.

Another indication of diminished sclerosis is a peculiar *affection of the speech*. The pronunciation is slow, accented, labored, as if the utterance of each syllable was a great effort. And yet it is not distinct; Charcot says that the letters, L, P, G, are especially apt to be slurred over; sometimes, but rarely, two or three syllables are run together. German writers describe the articulation as "scandirend" or scanning. In advanced stages it may be quite unintelligible. Erb further remarks that the voice is pitched in a monotone; or, as in one case that came under his observation, that there may be a rhythmical alternation of notes at a definite musical interval. Leube found with the laryngoscope that the vocal cords, although capable of closing the glottis, were yet apt to relax, and liable to changes of tension.

Thirdly, *nystagmus* is commonly present. It, however, differs somewhat from the ceaseless oscillation of the eyeballs with which ophthalmic surgeons are familiar. As Dr. Moxon has pointed out, it is absent so long as the eyes are at rest, showing itself only when they are directed upon one object. In other words, it is identical with the affection which is observed in Friedreich's hereditary ataxy (see p. 480), indeed, has already been suggested, both by him and by Erb, but both Friedreich and Erb have, indeed, already suggested that this is probably the case; but rather as a matter of speculation than as the result of direct observation.

In addition to these symptoms there are others which belong to disseminated sclerosis in common with other diseases. Subjective sensations of *numbness* and other paræsthesiæ are not uncommon; an actual anæsthesia, or impairment of tactile sensibility, can seldom be made out. The reflex susceptibility is generally normal, but sometimes it is much exalted. The gait at an early stage is often *spastic* in character. Afterward there is a marked tendency for the lower limbs to become rigidly extended and adducted; this condition being at first transitory, but afterward permanent. Dr. Moxon mentions that one of his patients was obliged to sit in an arm chair, so that when her legs stiffened she could hold on and save herself from sliding down on to the floor. Such early contractions are painless, but at a later period a painful flexion of the limbs is not infrequent. The characteristic symptoms of locomotor ataxy are now and then present in disseminated sclerosis; this is generally supposed to depend upon the existence of patches in the posterior columns of the cord. After a time complete *paraplegia* develops itself. The functions of the pelvic organs are at first but little interfered with. The urine may sometimes escape when the patient turns in bed, or coughs; or there may be slight retention. Constipation is often complained of. Dr. Moxon refers this, in part, to weakness of the abdominal muscles. The sexual organs retain their powers.

A transient diplopia is not infrequent at an early stage of the disease. Among other *cephalic* symptoms amblyopia often occurs, with narrowing of the field of vision and achromatopsy. Charcot insists on the fact that the impairment of sight rarely goes on to complete amaurosis, even when the

optic nerves have been found sclerosed in their whole thickness; he refers this to the persistence of naked axis cylinders in the midst of the diseased tissues. The optic discs are generally normal unless there is total blindness; in the latter case they have sometimes been found in a state of white atrophy. Headache, giddiness, and sleeplessness are not uncommonly present. The spirits are often depressed, and the memory and intelligence may be impaired. The patient often shows a tendency to burst into uncontrollable laughter, or to shed tears without any cause. Charcot insists on a peculiar *facies;* the expression is, he says, vague, uncertain; the lips droop and are half opened; the features have an air of dullness, even of stupidity. Sometimes there is an actual insanity. Charcot mentions two patients who exhibited the *délire des grandeurs* supposed to be characteristic of general paralysis; in others there has been melancholia, with refusal of food. A curious feature, which is observed in about one-fifth of the cases of disseminated sclerosis, is the occurrence of attacks of stupor, followed by transitory hemiplegia. They are attended with flushing of the face, a rapid pulse, and a temperature raised to 104° or even 106°. The coma lasts for a day or two, and then passes off into sleep. Sometimes, however, a seizure of this kind is directly fatal; one of Dr. Moxon's patients died so, in about twenty-four hours. Such an attack is said always to lead to an aggravation of the general symptoms of the disease, even when it is recovered from; in a case recorded by Leo severe "apoplectiform" fits occurred. It appears doubtful whether epileptiform seizures have hitherto been observed in disseminated sclerosis.

Lastly, in some exceptional cases atrophy of muscles is observed, affecting the upper or the lower limbs, the face, or the trunk. At the same time their electrical excitability, which ordinarily remains normal, may be lowered; the natural reaction is doubtless replaced by the "reaction of degeneration." Charcot says that the characteristic symptoms of bulbar paralysis sometimes show themselves, and dysphagia or paroxysmal dyspnœa may then be the immediate cause of death.

Course and Prognosis.—As a rule, the course of disseminated sclerosis is slowly but irregularly progressive. Remissions in the symptoms may occur spontaneously or under treatment. Very few remedies have been found of any service. Charcot says that the administration of strychnia or of the nitrate of silver has sometimes influenced the tremor and the weakness of the lower limbs very favorably, but only for a time. Some observers have seen transitory benefit result from the cold-water cure, from galvanism, or from the subcutaneous injection of arsenic. The average duration of the disease is said to be from five to ten years, the most rapidly fatal termination being in one year from the commencement, while the latest has been at the end of seventeen years. Something, however, depends upon the nature of the symptoms. Charcot says that cases in which "spinal" symptoms are alone present may go on for twenty years or longer. If death does not occur in one of the ways already mentioned, it is generally by the supervention of some intercurrent disease, such as pneumonia, pleurisy, dysentery, œdema of the glottis, or (above all) phthisis.

Ætiology.—With regard to the causes of disseminated sclerosis very little is as yet known. It appears to be equally frequent in either sex, although Charcot found twenty-five females to nine males among thirty-four cases which he collected. The age at which it most frequently develops itself is said to be between twenty and twenty-five years, but it has been several times observed at puberty, and a few instances of its occurrence in childhood have been recorded. It has not hitherto been known to begin in persons over forty or forty-five years old. Now and then it has appeared to be hereditary or has occurred in two individuals born from the same parents.

A chill, or a fall, or some emotional excitement, has sometimes appeared to give rise to it. Bauwinkel observed a case in which the patient was said to have fallen into water, and then to have allowed his clothes to dry upon him, three days before the first symptoms showed themselves. Charcot cites instances in which the disease began during convalescence from typhoid fever, cholera, or smallpox.

It must not be supposed that the *diagnosis* of disseminated sclerosis is invariably, or even generally, a very easy matter. Charcot says that trembling movements precisely like those which are present in that disease may occur not only in mercurial poisoning (when the history will in most cases remove all doubt), but also when the lesion is a "chronic cervical meningitis with cortical sclerosis," and even where it is a "primary or consecutive sclerosis of the lateral columns." The presence or absence of the other characteristic symptoms must guide the observer to a decision so far as these affections are concerned. But, again, great caution is required in women, to avoid mistaking hysteria for disseminated sclerosis. Dr. Moxon tells me that he has seen several patients about whom he has for a time been in doubt, but who have before long completely recovered. Moreover, the tremor may be absent, so that the case may appear to be an example of some common spinal affection. Charcot accounts for this by supposing that the symptom in question was present at an earlier period; but Erb refers to several cases in which it is said to have been wanting throughout the whole course of the disease. Thus he cites Westphal and Killian as having recorded instances in which the only symptoms were dementia and a more or less diffused paralysis with contractions of the limbs.

The *prognosis* of disseminated sclerosis is hopeless. Its progress is counted by years; but though very slow, it is very sure, and occasionally ends suddenly in apoplexy.

No efficient *treatment* has been suggested.

DISEASES OF THE BRAIN

(*DUE TO LOCAL LESIONS OF THE CIRCULATION*).

INTRODUCTORY REMARKS—LESIONS DEPENDENT ON OBSTRUCTIVE DISEASE OF THE ARTERIES—ON HEMORRHAGE—SYMPTOMS.—**Hemiplegia**—ITS CHARACTERS—DIAGNOSIS OF THE LOCAL LESION—COURSE AND SEQUELÆ.—**Aphasia.—Apoplexy.**—DIAGNOSIS FROM INJURY, POISON, ALCOHOL, PYÆMIA, URÆMIA, EPILEPSY—DIAGNOSIS OF THE CAUSES OF APOPLEXY—TREATMENT.

The brain differs from the spinal cord in its pathology in two important respects. On the one hand, its lateral halves, instead of being closely united and bound up so as to form a slender column within the firm sheath of the pia mater, are to a great extent isolated from one another, and are expanded each into a large, independent mass. On the other hand, its arteries, instead of penetrating its substance as fine twigs, which pass to both sides indifferently, are great trunks, with well-defined areas of distribution. Consequently, a large group of cerebral affections depend directly upon disease of the arteries of the brain, or upon an interruption of the supply of blood through some of these vessels. And in these very affections the symptoms are in a marked degree unilateral, any paralysis, or spasm, or loss of sensation in the body or limbs, being confined to parts on one side of the mesial line. *Hemiplegia*—or loss of power in one arm and the corresponding leg—is, in fact, generally present in such cases, unless, indeed, the lesion is so severe as to be attended with a rapid annihilation of all the chief functions of both sides of the brain—a condition which has long been known as *apoplexy*. Thus the affections in question contrast broadly in their features with those to which the cord is liable, and which (as we have seen) are commonly slow in endangering the vital processes, and are attended with a paraplegic form of paralysis. The boundary line is not, indeed, fixed at the foramen magnum; for the medulla oblongata and the pons—which the physiologist recognizes as continuations upward of the spinal axis—are subject to at least one of its diseases, that which I have already described under the name of bulbar paralysis. But the pons is also apt to be the seat of lesions due to changes in its arteries, and constituting a very fatal form of apoplexy. In fact, one may say that this part of the encephalon refuses to recognize the boundary line that separates the affections of the brain from those of the spinal cord. The paralyses which have their seat in it are not necessarily unilateral, but its pathology is to a great extent dominated by that of its blood vessels, exactly as is the case with the higher cerebral centres.

To avoid the necessity of going over the same ground more than once, I shall first give an account of the several lesions and of their respective causes and anatomical effects. Then I shall proceed to discuss the symptoms to which they may give rise; and finally, I shall endeavor to point out how we can diagnose one of them from the others, as well as how they are to be distinguished from affections of a different nature.

The lesions themselves fall into two groups: (1) Those in which there is simply an arrest of the flow of blood through one or more of the cerebral arteries, and (2) those in which an artery is ruptured, allowing the blood to escape into the brain or beneath its membranes. The former group includes chiefly *embolism*, *thrombosis*, and *syphilitic disease* of the cerebral arteries; the latter is made up of the various forms of *cerebral hemorrhage*.

I. *Arrest of Blood Supply through the Cerebral Arteries.*—This may occur in various ways, which require to be separately considered.

The obstruction may arise *outside the cranial cavity*. I have already alluded to a case of Sir William Gull's, in which arteritis deformans of the arch of the aorta led to complete obliteration of the innominate and left carotid arteries at their origins, so that the left subclavian artery alone was left to carry on the circulation in the brain. The patient, a woman, aged forty-one, died paralyzed and insensible; and certain parts of the cerebral centres were found to be in a state of softening. Some years ago, a woman affected with carotid aneurism was admitted into Guy's Hospital, who had nearly a year before been attacked with hemiplegia and loss of speech. Mr. Durham performed the operation of ligaturing the common carotid artery, whereupon she regained to some extent the power of speaking. However, she died soon afterward, and I made an autopsy and found that the aneurismal sac extended upward by the side of the internal carotid artery, and pressed upon it so that until the tension was lowered by ligature of the vessel below, the blood which reached the internal carotid from collateral sources had been unable to pass through it. Much more commonly, it is *after* ligature or compression of the carotid artery that hemiplegia sets in. I have notes of five cases of this kind. In most of them the paralysis was the direct result of the operation; but in one instance it did not occur until three days afterward, the patient becoming suddenly hemiplegic while the dressings were being changed. The suggestion has been thrown out that in those cases in which cerebral symptoms manifest themselves under such circumstances the communicating arteries which make up the circle of Willis may perhaps be abnormally small; but I am not aware that this has been demonstrated anatomically.

Embolism.—In the immense majority of cases the cause of arrested circulation in the cerebral arteries lies within the skull; and it is generally the presence of an embolism—a clot derived from some distant source, and washed into the vessel by the force of the blood stream. Most frequently, the starting point of the morbid process is some disease of the valves in the left side of the heart; the patient, perhaps, has ulcerative endocarditis, or there may be simple vegetations on either the mitral or the aortic valves. In one case, in which I made an inspection in 1877, a rough, friable mass of calcareous deposit was exposed upon the surface of the mitral valve, the lining membrane of the heart having undergone erosion, and a fragment of the same material was found wedged in one of the Sylvian arteries.

Sometimes stenosis of the mitral orifice is present, without any projections from its surface; the clot is then derived from the left auricle, which perhaps contains numerous other thrombi in the recesses between the fleshy columns of its appendix, or the formation of ante-mortem coagula in the heart's chambers may be independent of any valvular affection. In very rare instances the primary lesion is in the aorta itself.

We have had at Guy's Hospital two such cases; in one there was a patch of softening thrombus which adhered to a diseased part of the wall of the great artery; in the other ulceration existed. Altogether, I find in the records of post-mortem examinations for the last twenty-three years, forty-seven cases in which there was reason to believe that embolism of one of the

cerebral arteries had occurred. It is true that in a very large number of them (twenty-one) no clot was discovered in any of the vessels at the autopsy, the interpretation of the cerebral symptoms as due to this cause being based upon their occurrence in persons who had disease of the cardiac valves and upon the presence of such changes in the brain as are known to result from it. All writers admit that anatomical proof of plugging of the arteries is wanting in some fatal cases, especially when life has been prolonged for a considerable period. They suppose that the clot has in the meantime undergone absorption. That this may be the case is likely enough, but I would suggest that another possible explanation is that the spot at which the embolism was impacted may have been the bifurcation of the carotid artery in the neck, or one of its bends within the petrous bone or in the cavernous sinus; or, possibly, even where a vertebral artery is winding round the arch of the atlas—parts which, I believe, have generally escaped examination. The vessel into which the clot passes is, however, almost always one of the Sylvian arteries. I have notes of twenty-three cases in which embolism of these vessels was found at the autopsy, as against three cases in which its seat was the vertebral artery. The fact that emboli entering the carotid are almost always carried into the Sylvian branch is doubtless attributable to the circumstance that its course is more in a line with the trunk than that of the two other branches. Writers have stated that the left Sylvian artery is much more apt to be plugged than the right one, and an explanation for this supposed fact has been found in the difference of the angles at which the innominate artery and the left carotid artery respectively leave the aorta. But it is not confirmed by the observations which have been made at Guy's Hospital, for among twenty-one cases of embolism limited to the Sylvian artery of one side I find that there were eleven in which the left one was affected, and ten in which the clot entered the right artery, a difference too slight to be noticed. I am inclined to think that error has arisen from the statistical collection of miscellaneous cases from Journals and Transactions; the interest attaching to aphasia—a frequent result of embolism on the left side—must have often led to the publication of cases in which that symptom was present, whereas those in which it did not occur have been left unrecorded.

Another point in regard to which my cases are opposed to the statements of certain writers is as to the age at which cerebral embolism is most apt to occur. They speak of it as most frequent in very young adults; but I find that at the same hospital the largest number—12 cases—have occurred between the ages of thirty-one and forty years, as compared with 10 between twenty-one and thirty, 9 between forty-one and fifty, 8 between fifty-one and sixty, 6 between eleven and twenty, and 1 above the age of sixty. There have been rather more males than females among the patients; but this was probably accidental.

Thrombosis of the cerebral arteries—the formation of clots *in situ*, independently of any extraneous source—is far less frequent than embolism. Nothnagel says that it is generally secondary to disease of the arterial wall, either atheroma, or thickening with calcification. Sometimes it seems to be a direct result of feebleness of the heart's action. He is inclined to think that the vertebral arteries are more liable to be affected in this way than the carotids; and this accords with our experience at Guy's, where there have been three or four cases of thrombosis of the basilar artery, or of the posterior cerebrals, but (so far as I know) only one case of a similar affection limited to the middle or anterior cerebrals. In one remarkable instance, however, both the carotid arteries, the middle cerebrals, the anterior cerebrals, and (it is said) the "posterior cerebellar" arteries, were all obstructed by adherent clots. The patient was a man aged thirty-five; and our other

cases have been in persons aged thirty, thirty-five, forty-eight, and forty-nine years respectively. It would therefore appear doubtful whether Nothnagel is justified in saying that thrombosis, as a rule, occurs at a more advanced period of life than embolism.

Syphilitic disease of the cerebral arteries has only recently become known to pathologists; but it is already seen to be one of the most important effects of the *lues venerea*, although I doubt whether its full clinical significance has as yet ever been fully recognized. The first monograph devoted to it is that written by Heubner, of Leipzig, which appeared in 1874. He assigns to a Danish writer, Steinburg, the credit of having first attributed to an affection of the blood vessels many of the cerebral symptoms which arise as a result of syphilitic infection; and to Wilks that of having suggested that the change in the arterial coats is one of a special kind, and different from the ordinary atheromatous affection with which it had been confounded. Its histological characters have been determined by Heubner himself. He finds that it begins between the endothelium and the fenestrated membrane. Here a number of cells accumulate, which form a mass that encroaches on the calibre of the vessel. In one instance the fenestrated membrane was in part destroyed, the growth penetrating into the muscular coat. He speaks of this as exceptional; but we have at Guy's had two cases in which an affection that seemed evidently to have begun in the vertebral artery passed right through the walls of the vessel into the substance of the pons Varolii. Heubner himself points out that with the arterial affection there is often associated a gummatous growth in the loose tissue of the pia mater at the base of the brain; but he regards this as independent. The cells in the intima of the artery presently undergo development, the inner run into spindle cells arranged transversely, the outer into interlacing stellate cells. Heubner compares this process with the formation of a new wall to the vessel, in support of which view he states that a new fenestrated membrane may actually be generated immediately beneath the endothelium. The new tissue becomes vascular, and this perhaps saves it from undergoing retrograde changes. At any rate, it has hitherto been constantly found grayish white and semi-translucent, so that Heubner confirms the statement, which was originally made by Dr. Allbutt, that it has scarcely any tendency to caseate, differing in this respect from syphilitic affections of all other parts. I am disposed to think, however, that this may partly depend upon the fact that syphilitic disease of the cerebral arteries has hitherto been observed only in the large vessels at the base of the brain. One can hardly doubt that it must also occur in the smaller branches, where its effects would be less serious, so that it would be less likely to be seen in an early stage at a post-mortem examination. As the channel of the artery becomes narrowed the blood often coagulates in its interior, and thus the circulation through it is arrested. Heubner expresses doubts as to whether the growth ever by itself leads to complete obliteration, independently of thrombosis. Ultimately a process of cicatrization may take place, the cells developing into connective tissue, and the vessel undergoing conversion into a fibrous cord.

When a single vessel has been affected with syphilitic disease it has much more frequently been the carotid artery, or one of its branches, than the basilar; the proportion given by Heubner being as twelve to one. But in most instances several arteries become diseased simultaneously. In one of Heubner's cases the circulation at the base was interrupted at four distinct points; the left vertebral artery was obliterated, and so were also the basilar, the left middle cerebral, and the origin of the right anterior cerebral arteries. Still there is always a marked difference between the syphilitic affection and atheroma; the latter is commonly diffused pretty

uniformly over all the trunks in the immediate neighborhood of the circle of Willis; moreover, it tends to dilate the affected vessels, and to render them tortuous, rather than to narrow and occlude them.

As might be expected, it is generally at an advanced stage of syphilis that the cerebral arteries become affected. Among the cases collected by Heubner the oldest patient was fifty-one years old, the youngest twenty-two; and the numbers were pretty evenly distributed over the three decennial periods between twenty and fifty years of age. It is to be noted, however, that in all his cases, with scarcely an exception, there was a definite history of venereal infection. Now, syphilitic disease of the arteries of the brain is unlike every other remote effect of syphilis, if it is not apt to occur in persons who are ignorant of having the poison in their systems, and who are free from eruptions, nodes, and other obvious signs of its influence. At Guy's Hospital, since the first case recorded by Wilks in 1863, we have had in the post-mortem room five other well-marked instances of the affection, besides eight or ten of a more doubtful character. In comparatively few of them was there a clinical history of syphilis, the proof of their origin depending rather upon the presence of other internal syphilitic lesions, such as gummata in the liver or testes, lardaceous disease of the organs, etc.

Effects of Obstruction of Cerebral Arteries.—The state of the brain substance, in cases in which there has been arrest of the circulation in one or more of the cerebral arteries, corresponds fairly well with what might have been expected from our knowledge of the effects of interference with the arterial blood supply to other tissues. In general, it may be said that the affected parts are *softened;* and I may remark that the various affections that I am now describing include, perhaps, the majority of those cases which by the pathologists of the last generation were classified as examples of "softening of the brain." The anatomical relations of the vessel which happens to be obstructed, of course, determine the regional distribution and extent of the morbid change; and upon this, in its turn, depends the nature of the symptoms. In regard to the Sylvian artery, we shall hereafter find that the whole clinical aspect of a case differs, according as this vessel is plugged at its origin, or a little further on in its course.

Thus it is of great importance to the physician to be acquainted with certain investigations as to the exact mode of distribution of the cerebral arteries, recently worked out by two independent observers, Duret in France, and Heubner in Germany. As they point out, the small arteries that alone enter the tissue of the brain form two separate systems, which may be distinguished as those of the cortex and those of the central ganglia respectively. These systems are altogether independent of one another. No anastomoses take place between them; the zone at which they meet within the cerebral substance is situated about an inch and a half below the convolutions. Now, the "central" arteries arise directly from the trunks forming the circle of Willis; they are entirely unprovided with anastomoses. But the "cortical" arteries spring from a network in the pia mater, in which it is said that tolerably free communications exist between the different tertiary branches of the same trunk, and even (in some individuals) between the branches of different trunks. Let us now apply these facts to elucidate the effects of obstruction of the Sylvian artery at different points. That artery, close to its origin, gives off a number of small "central" twigs, which supply the whole corpus striatum (except the inner end of the caudate nucleus), and also the anterior part of the optic thalamus. It then divides into four terminal branches, of which one is distributed to the third frontal convolution, while the other three pass to the second frontal, the two central, the three parietal, and the three temporal convolutions (I

may note that the statements of Duret and Heubner are not precisely the same with regard to their exact destination). Thus, when the middle cerebral artery is obliterated beyond the point at which its "central" offshoots arise, the superficial parts of the brain are the only ones to suffer. And since the subdivisions of the vessel in the pia mater anastomose with those of the anterior and posterior arteries, it is not certain that any softening will result. There may be merely a temporary interference with the circulation in the area to which the Sylvian branches are distributed; or a limited part of this area toward its centre may be permanently deprived of its blood supply; or, lastly, almost the whole of the convolutions enumerated above may undergo destruction. Thus Charcot has recorded an instance in which an enormous superficial patch of softening involved the ascending frontal and the ascending parietal convolutions, as well as those of the insula, the central ganglia remaining healthy. The differences in the result in different individuals depend partly upon variations in the extent to which the vessels communicate with one another, partly upon whether the plugging takes place suddenly or gradually. It is, of course, quite possible for a single one of the four terminal branches, such as that to the third frontal convolution, to undergo obstructions apart from the rest. On the other hand, when the seat of the lesion is at the spot where the Sylvian artery arises from the internal carotid, the central ganglia are concerned. And in their case softening is almost inevitable, because their arteries have no anastomoses; it is only when the closure of the vessel takes place very slowly, as the result of chronic disease of its coats, that collateral channels sometimes seem to develop themselves, so that the blood supply is maintained. It is important to notice that when the main channel of the Sylvian artery is obliterated, extensive morbid changes in the corpus striatum and thalamus are not at all unlikely to occur in a case in which the convolutions entirely escape.

The appearances presented by the affected parts of the brain vary considerably under different conditions. When the patient dies rapidly, the cerebral substance may look perfectly healthy. This was the case, for example, in a man who was attacked with hemiplegia twenty-six hours after ligature of the internal carotid artery, and who lived only fifty-seven hours afterward. And I have already observed that where closure of an artery takes place gradually, the regions of the brain that are deprived of their normal blood supply often fail to suffer as much as might be expected. Thus Heubner remarks that in the syphilitic affection of the cerebral arteries the nutrition of the cortex is seldom seriously interfered with unless, out of the six main trunks that arise from the circle of Willis, two adjacent ones are completely obstructed. In the case already referred to, in which the circulation at the base of the brain was interrupted at four distinct points, there was no softening at all, although one Sylvian artery was obliterated.

When arrest of the flow of blood through a cerebral artery takes place gradually—as in thrombosis and in syphilitic disease—I believe that the morbid changes in the brain, if any occur, always take the form of *softening*. The affected parts are sometimes actually diffluent, being represented by a milky liquid, which occupies an ill-defined cavity, containing some loose shreds of connective tissue, and perhaps roofed in by the pia mater, or by the ependyma of the lateral ventricle. A very intermediate degree of consistence may occur, up to a point at which one can hardly perceive any difference from the healthy substance around, until one allows a gentle stream of water to play over the diseased surface, when it soon becomes ragged, and assumes a worm-eaten appearance. The color of softened parts of the brain is very variable—it may be white, or yellow, or even

brownish. Writers have been accustomed to describe separate varieties of the affection according to color. But (except in the case of "red softening," which will be described hereafter) I think it is of great importance to ignore such distinctions, for they have no real existence, and they have already led to great confusion. As a matter of fact, the condition now under discussion is that which most English pathologists call "white softening," but by Rindfleisch it is named "yellow softening;" and each of these two epithets is susceptible of an entirely different application; for the German writer uses the term "white softening" for a morbid appearance in the central parts of the brain in cases of hydrocephalus, and we have been accustomed to designate as "yellow softening" the œdematous state of the cerebral substance that is so often found in the neighborhood of tumors.

The differences in color in different cases of softening of the brain, indeed, depend simply upon the presence of extravasated blood in greater or less quantity, or upon its entire absence. The blood undergoes disintegration with the tissue elements among which it lies, and under the microscope it is early recognized in the form of yellow or red granules, or of hæmatoidin crystals. This instrument also reveals in softened brain substance "compound granule masses," or "corpuscles of Gluge," often in large numbers. These mulberry-like aggregations of minute granules of fat have already been mentioned as occurring in certain affections of the spinal cord, and, indeed, they are found in many degenerating tissues. In the brain, however, it is still uncertain how they are formed, for Huguénin, in a paper on the subject, admits no less than seven sources for them, namely, the nuclei of the neuroglia, the cells which make up the walls of the capillaries, those of the "adventitia" of the arteries, the nuclei of the smooth muscular fibres of the vessels, those of the perivascular lymph spaces, the *Spindeln der Cortex*, and very probably the ganglion cells of the cortex.

Again, in *embolism*, or after ligature of the carotid artery, softening is, as a rule, the only change presented by the parts of the brain deprived of their blood supply; but these affections have certain peculiarities that seem to modify the appearances which are met with in some cases. One thing is that the obliteration of the artery takes place suddenly. Even after ligature of the carotid artery the corresponding cerebral hemisphere is now and then found in a condition of vascular turgescence, precisely like the early stage of an infarctus. Thus, in one case in which I was present at an autopsy made by Dr. Wilks, all the vessels on the affected side of the brain —including the veins as far as the longitudinal sinus—were distended and filled with coagula, and the substance of the organ, which was pulpy, was of a dark red color. So, again, in those case of embolism that have occurred at Guy's Hospital, a condition of "red softening" is described as having been present, and in two other cases tough yellow masses were found which exactly resembled the wedge-shaped patches that are so common in the spleen and the kidney. "Red softening," indeed, is a change of which the significance is doubtful. It may be an ordinary softening, in which minute extravasations of blood have taken place in unusual numbers; or, perhaps, it may simply be an early stage of "white" or "yellow softening"—the reason for its not having hitherto been seen in cases of thrombosis or syphilis being that in these diseases death does not take place soon enough. But, on the other hand, it may be a local cerebritis, such as I shall have to describe in a future chapter. Now, it is well known that an embolism often sets up inflammatory processes in the parts around the vessel in which it becomes lodged, and which consequently is found imbedded in a mass of inflammatory thickening; and I have notes of one case in which a general meningitis was present, there being at the

same time suppurating infarctus in the spleen and kidneys. Again, the coats of the artery itself often become softened, and yield immediately below the seat of obstruction, so that an aneurism is formed. At least four examples of this have been observed at Guy's Hospital within the last few years; in one of them Dr. Goodhart found an aneurism on the Sylvian artery on each side. It was not, indeed, proved in every instance that plugging had occurred, but all the patients had disease of the cardiac valves, and I have already remarked at p. 489 that the pathological evidence of embolism sometimes fails us. Death was in most cases due to rupture of the aneurism, blood being extravasated in considerable quantity into the pia mater as well as into the substance of the corpus striatum, and even reaching the lateral ventricle. But, in addition, cerebral hemorrhage has been the cause of death of seven other persons who were affected with ulcerative endocarditis or had vegetations on their mitral or aortic valves; in all probability an aneurism, itself the result of embolism, was present in these cases likewise, although it escaped notice at the autopsy. Sir William Gull long ago suggested that it was chiefly in young subjects that intracranial aneurism should be suspected as a cause of cerebral hemorrhage; and Dr. Church, in tabulating a series of cases of this kind, confined his attention to individuals under twenty years of age. But I may note that among nine cases which have occurred at Guy's Hospital, and in which the ages of the patients were known, seven only were between fourteen and twenty-six, one patient being thirty-four and another fifty years old. I have already stated that embolism itself is by no means so strictly limited to an early period of life as has been generally supposed.

II. *Cerebral Hemorrhage.*—As a rule, however, effusion of blood into the substance of the brain takes place altogether independently of embolism, and as the result of an entirely different morbid process. Since the days of Morgagni it has been known that cerebral hemorrhage is far more apt to occur in or near one of the corpora striata than in any other part of the brain. But within the last few years certain anatomical facts have been made out, which have not only led to a more exact determination of its seat than had before been possible, but have also gone far toward explaining why this spot should be so generally selected for the rupture of an artery. Outside each lenticular nucleus there is a mass of white substance, which it is now the fashion to call the "external capsule." In ordinary sections the continuity of the two structures seems to be as close as between any other portions of the cerebral mass which happen to differ in their color. But when a brain is hardened, it is found that they can be separated from one another with very great ease, and apparently, although not really, without any nervous fibres being torn through. Thus the connection between them must be somewhat imperfect. Now, the central branches of the Sylvian artery, which supply the corpus striatum, run upward for some distance outside the lenticular nucleus before penetrating into its interior. Even on mechanical grounds, we can see why those vessels should be more liable to laceration than those of the hemispheres generally. They are much larger than the nutrient arteries of the cortex, they have no anastomosis with one another, and they arise directly from a large trunk, which may almost be said to come in a straight line from the heart. Indeed, Watson long ago pointed out that when injections are forced into the cerebral arteries of the dead body it is especially in the corpora striata that the vessels are apt to give way, and the wax or other material to be extravasated. Charcot goes so far as to speak of one particularly large branch as "the artery of cerebral hemorrhage. It is true that, as we shall presently see, the rupture of any artery in the brain is always preceded by the occurrence

of morbid changes in its walls, but I do not know that this fact deprives of their significance the considerations to which I have just alluded. The blood begins by making for itself a space between the lenticular nucleus and the external capsule. At first it forms a thin layer, but as it increases in quantity it gradually becomes a rounded mass. It now flattens out the convolutions of the island of Reil, which lie below and to the outer side of it, it pushes inward the corpus striatum, and also the thalamus opticus, if it extends far enough backward. When it accumulates slowly, it may press upon the parts around, so as to form a smooth-walled cavity for its reception. But more frequently it tears up irregularly the white matter of the hemisphere and the outer part of the corpus striatum. In some rare cases it reaches the superficial convolutions; far more often it ruptures into the lateral ventricle. Coagulation quickly occurs, and thus at an autopsy one finds a mass of clot weighing four ounces or more. Within the lateral ventricles there is sometimes a red liquid, consisting of blood mixed with the cerebro-spinal fluid; sometimes a clot occupies the side which was first reached by the blood, the contents of the other side being only blood stained with perhaps a little clot in the cornua. Dr. Broadbent has noticed that the middle cornu close to the seat of the hemorrhage is commonly empty, having been compressed by the extravasated blood before rupture into the lateral ventricle took place. Not infrequently the third ventricle, the *iter*, and the fourth ventricle are all filled with moulded coagula exactly fitting their cavities; the blood may even escape along the subarachnoid space so as to reach the exterior of the medulla oblongata and of the pons, and the parts at the base, as far as the opposite Sylvian fissure. The fissure on the side of the hemorrhage is too closely compressed to be able to receive any of it. Indeed, the convolutions of the vertex of that hemisphere are often greatly flattened, so that, as soon as the skull cap is taken off, one sees at once which side contains the blood.

I have before me notes of 96 consecutive fatal cases of cerebral hemorrhage that have occurred at Guy's Hospital. In 71, the seat of the affection was in the neighborhood of the cerebral ganglia. Now, there are only 4 of these cases in which I find it stated that the blood was confined to the substance of the hemisphere, not having found its way either to the surface or into the ventricles. In 57, it is expressly recorded that laceration into the lateral ventricle had taken place; and in 22 that even the fourth ventricle contained either a clot, or at least a blood-stained fluid. In 6 cases the superficial convolutions were reached. The right side of the brain was affected in 36 cases, the left side in 34. In 6 instances the part into which the blood was effused was behind the optic thalamus, or, in other words, beyond the limits of the region which I have described as the usual seat of cerebral hemorrhage. Twice the caudate nucleus was alone affected, the blood passing at once into the lateral ventricle through a superficial rent in the corpus striatum.

In twelve of the remaining twenty-five fatal cases the seat of the hemorrhage was in the pons Varolii; in one it was in the right half of the cerebellum. Among the cases of "apoplexy of the pons" there were five in which the blood had escaped into the fourth ventricle; two in which it had oozed out through the convex surface of the pons, so as to reach the base of the brain. I should add that in no less than eight of the cases in which there was hemorrhage in the neighborhood of the corpus striatum, blood was also effused into the pons Varolii; in one instance, as many as three independent hemorrhages were found in this part. I suppose that they had occurred secondarily, as a result of the obstructed respiration caused by the primary attack of apoplexy.

Lastly, there were twelve cases in which the extravasation was into the

membranes on the surface of the brain. "Meningeal Apoplexy" has been described by most English writers as a distinct affection; and this is, to a certain extent, justified by the fact that some instances of it are connected with purpura, anæmia, or blood poisoning, while others are caused by blows or falls on the head. But in the cases to which I am now referring it was associated with exactly the same changes in the heart and kidneys which we shall hereafter see to be commonly found in those of ordinary cerebral hemorrhage, and there is every reason to believe that the difference in seat was altogether accidental. The quantity of blood was often very considerable; the nerves and other structures at the base of the brain were buried in a thick clot, and the extravasation extended along the Sylvian fissures and in the meshes of the pia mater over the surface of the hemispheres to a greater or less extent, and also in some instances along the subarachnoid space around the spinal cord, and into the fourth ventricle.

When life has been prolonged for a few days after the occurrence of cerebral hemorrhage, the brain tissue round the clot commonly exhibits reactive changes. At first it is reddened, or even ecchymosed; then it becomes œdematous and of a yellow color, exactly as in a case of tumor of the brain. At a still later period it may be found in a state of softening. Whether suppuration ever takes place round a clot, as was previously supposed, is very doubtful. Sir Thomas Watson relates a case for which he adopted this explanation; but it is not unlikely that the clot, which was found lying in an abscess, was really of later date than the pus.

But cerebral hemorrhage does not necessarily destroy life, so that there are other morbid appearances, besides those that are seen in cases which prove directly fatal. I find in our records of post-mortem examinations at Guy's Hospital twenty-one cases in which the remains of effusions of blood of old date were discovered in one of the basal ganglia or in their immediate neighborhood. And, in addition, ten of the seventy-one cases of recent cerebral hemorrhage presented patches which had been of earlier origin. Sometimes all that was left was a soft, tawny, discolored spot, containing abundant granule masses and crystals of hæmatoidin; or a tough, flat, fibrous mass, an "*apoplectic cicatrix.*" Sometimes there was a well-defined membranous cyst with shreds of connective tissue and a clear or turbid fluid in its interior. In one instance the cavity was of the size of a walnut; in another it was as large as a bantam's egg. Such appearances have been plainly recognized thirteen or fourteen years after the attack, when the patient has died of some different disease. I do not find that any of our cases throw light upon the question within how short a time it is possible for a clot in the brain to undergo absorption, and for a cyst to take its place. It may be worth while to notice that in no single instance in which the remains of an apoplectic clot have been found in the brain after recovery has there been any indication that the blood had made its way into the lateral ventricle. But according to the statements of Rokitansky and Charcot it would seem that in some very rare cases even this is not inevitably fatal.

Ætiology.—With regard to the causes of cerebral hemorrhage, considerable differences of opinion still prevail. In this country the most obvious pathological change found in the bodies of those who have died of effusion of blood into the brain is undoubtedly *chronic renal disease.* I have made an analysis of 116 cases* which came under observation consecutively in the post-mortem

* This number is made up of 85 cases of hemorrhage into the basal ganglia or into the substance of the hemispheres (71 recent and 21 old), 12 cases of apoplexy of the pons, and 12 cases of meningeal apoplexy. I have excluded all those instances (referred to at p. 488) in which there was reason to believe that embolism was the starting point of the cerebral mischief, and also certain cases of meningeal hemorrhage, which accompanied purpura, or were due to injuries or other accidental causes.

room of Guy's Hospital; and in eighty-six of them some morbid condition of the kidneys is stated to have been present; in only fifteen out of the whole number it is reported that these organs were healthy. But, strange to say, continental pathologists seem not to have found the kidneys diseased in a similar proportion of cases. Charcot and Bouchard say that interstitial or parenchymatous disease was present in only three among forty-nine cases of theirs in which the state of these organs was noticed, there being, however, thirteen other cases in which the kidneys were "simply atrophied." Nothnagel, in Ziemssen's "Cyclopædia," appears to be equally ignorant of the close connection between renal affections and cerebral hemorrhage, for he quotes, almost without comment, the statement of Frerichs, that among 241 cases of Bright's disease there were only six in which effusion of blood into the brain occurred. This, indeed, is not so incompatible with the English observations as might at first sight appear. For none of our cases have been examples of the epithelial (parenchymatous or catarrhal) affection of the kidney; and comparatively few of them seem to have presented those conditions of mixed epithelial and interstitial changes which are so commonly the cause of dropsy and other symptoms in persons at the middle period of life. In the great majority the renal affection has been of the "granular" kind, the organs having shriveled up into mere relics of their former structure, but retaining their red color unaltered. In a few instances one kidney had undergone destruction from calculous affection of its pelvis, or the two organs had each become converted into a large tumor, made up of a congeries of cysts. But it is possible that in such cases the occurrence of cerebral hemorrhage was merely an accidental coincidence.

The fact that it is the "red-granular" form of Bright's disease which is commonly found when blood is effused into the brain, is of itself sufficient to prove that the hemorrhage is not a mere result of that increased tension in the arteries generally which is caused by the epithelial as well as by interstitial varieties of renal disease. And I think that all trustworthy observers are agreed that morbid changes in the coats of the vessels precede their rupture, although there are differences of opinion as to the character of such changes. Sometimes the arteries which arise from the circle of Willis are affected with an extreme degree of *atheroma*, or have their walls extensively calcified, so that one is inclined to suppose that this is the cause of the hemorrhage. In the case of a man, aged 40, who died in Guy's Hospital under my care, in 1869, and who had a hypertrophied heart and granular kidneys, there was a small aneurism on the left middle cerebral artery which had given way and poured blood into the Sylvian fissure, while there was also a large clot outside the corpus striatum, with laceration into the lateral ventricle, apparently of independent origin. But in most cases pathologists have until recently failed to find the ruptured artery, and the vessels at the base of the brain and their branches often seem to be fairly healthy. In 1866, however, Bouchard discovered, in a case of cerebral hemorrhage which had occurred in the wards of M. Charcot, two small aneurisms upon one of the vessels. And since that time these two French observers have collected no less than seventy-seven cases of the same kind, in every one of which they say they have demonstrated the presence of what they term *miliary aneurisms*. These are described as minute globular or fusiform swellings, from one-fifth of a millimètre to one millimètre ($\frac{1}{25}$th to $\frac{1}{125}$th of an inch) in diameter, so as to be plainly visible to the naked eye. They are sometimes very soft, sometimes firm and elastic, sometimes hard, like grains of sand. In color they are purple or reddish-brown, or grayish, according to the thickness of their walls and the state of their contents, which may be either fluid blood or clot of greater or less age. They are generally multiple, but sometimes only two or three can be dis-

covered in the whole of the brain after most careful search; sometimes as many as a hundred have been counted. They are most conspicuous upon the surface of the convolutions and in the sulci between them; a favorite seat for them is the deep layer of the gray matter or the line of junction between it and the white. But they may also be found in the thalami and corpora striata, in the pons and elsewhere. MM. Charcot and Bouchard themselves admit that, when an extensive effusion of blood into the brain has occurred, it is difficult to find the miliary aneurism, the rupture of which they suppose to have led to the hemorrhage. The best way is to place the affected hemisphere in a basin, and to change the water frequently by tilting, without shaking it. After a time the clots become loosened and float away, leaving a number of little bloody masses connected by vascular filaments with the brain tissue beneath. Most of these, when magnified, show only the open ends of vessels, torn across secondarily by the extravasated blood, and covered with little caps of fibrin that require to be carefully distinguished from actual aneurisms; but at length some are found which contain spherical ampullæ, continuous with small arteries and split on one side.

It is evident that the discovery of MM. Charcot and Bouchard affords only a partial explanation of the pathology of cerebral hemorrhage, since miliary aneurisms are not found with especial frequency in those regions of the brain which we have seen to be so generally the seats of effusions of blood. And, indeed, the French observers themselves point out that the lesion which they describe is, after all, but one of the effects of a diffused morbid change in the cerebral arteries—a chronic "sclerous periarteritis"—consisting in a thickening of the arterial sheath and of the adventitia, with an overgrowth of nuclei, while the muscular coat undergoes atrophy. At first they supposed that this condition was peculiar to the vessels of the brain and occurred only in old people. But another observer at the Sâlpetrière, M. Lionville, has since demonstrated the occurrence of a similar affection in the arteries of other parts, and even speaks of having found miliary aneurisms beneath the mucous membrane of the œsophagus and upon the surface of the heart. They have also been detected, although rarely, in young adults. In other words, we are brought by their investigations to almost precisely the same point as by the observations of Sir William Gull and Dr. Sutton on "arterio-capillary fibrosis." Indeed, it seems clear that the French and the English pathologists have respectively been studying the same morbid process, and their general agreement goes far to prove the accuracy of their conclusions. Only, it is strange that MM. Charcot and Bouchard should have failed to notice the affection of the kidneys on which Gull and Sutton have laid so much stress.

I think, too, that there can be no doubt that the diffused change in the blood vessels, to which reference has just been made, is often the cause of an impairment of the nutrition of the brain, attended with an obvious softening of its substance, which must greatly increase the risk of arterial rupture. Charcot and Bouchard quote Rochoux as having first promulgated the doctrine of a *ramollissement hemorrhagipare*, which was further developed by Todd; but they maintain that the only "softening" that bears any relation to cerebral hemorrhage is the secondary affection which we have seen to be of frequent occurrence in the tissue round a clot. But I find in the records of post-mortem examinations at Guy's Hospital no fewer than twenty cases in which localized patches of white softening were found in different parts of the brain—sometimes in the corpora striata or thalami, sometimes in the pons, sometimes in the hemispheres. Some of the patients had suffered from gout; a large majority of them had granular kidneys and hypertrophied hearts; there is not a single instance in which the kidneys are said

to have been healthy. In the ages at which they proved fatal, and in the circumstance that the large majority of the patients were males, these cases corresponded exactly with those of cerebral hemorrhage; and I can feel no doubt that it was a mere accident that death occurred before any of the arteries in the softened patches gave way.

I have just adverted incidentally to the fact that cerebral hemorrhage does not occur with equal frequency in the two sexes or at different periods of life. Men are much more liable to it than women. I find that of 113 consecutive fatal cases at Guy's Hospital, 82 occurred in males, only 31 in females. Among 110 of these cases, in the reports of which the ages of the patients are stated, there were 6 between twenty-one and thirty, 18 between thirty-one and forty, 37 between forty-one and fifty, 29 between fifty-one and sixty, 17 between sixty-one and seventy, and 3 above the age of seventy. These figures do not altogether correspond with the statements of writers in general, who make the frequency of apoplexy increase up to the age of seventy or even eighty years—not only relatively to the numbers of persons living at different ages, but also absolutely. It may be that men at a middle period of life are more likely than those who are older to be brought to the hospital when attacked with sudden and alarming symptoms, because their relatives are not so apt to suppose that their cases are hopeless.

Whether cerebral hemorrhage is of more common occurrence in men with short, thick necks and florid faces than in those who are of a different build, is perhaps doubtful. But there can be no question that gout is indirectly a very important factor in its causation, by setting up granular disease of the kidneys. And gout is notoriously infrequent in persons who are thin, as compared with those who are stout and plethoric.

Cerebral hemorrhage sometimes occurs first while the patient is making some violent effort, or subjecting his vascular system to an excess of pressure, so that one can hardly help regarding this as the exciting cause of the attack. Thus two of the cases that have recently occurred in Guy's Hospital were in gentlemen who had been running to catch a train; one was in a woman who had recently suffered from sea sickness in crossing from France; another, also, in a female patient, seemed to be the result of anxiety and disappointment at her husband having failed to return home when he had been expected. All writers, in fact, mention emotions, violent efforts, cold baths, straining at stool, prolonged laughing, coughing, or sneezing, indulgence in stimulants, as capable of bringing on the rupture of an artery in the brain, provided that the vessel is in the diseased condition which seems to be a necessary antecedent of hemorrhage. Among laboring men many are attacked while at work; but one must not forget that this takes up a great part of their daily lives; and that the affection develops itself in a large number of persons during sleep, when the pressure in the cerebral vessels is supposed to be particularly low. Intoxication by alcohol seems to be a frequent cause. A striking illustration of this (although the hemorrhage was not of the usual kind) came under my notice some years ago; a man who had a severe injury to the head six months before, got drunk two days in succession, and on the third day was taken in a fit, became comatose, and died thirteen days afterward. A quantity of blood was found effused in the arachnoid cavity; but the convolutions also showed several tawny patches of superficial softening, which were no doubt effects of bruising of the brain at the time of the accident; and it seemed to me clear that vessels in one or more of these patches had been the source of the recent hemorrhage, having given way in consequence of the disturbance in the cerebral circulation to which the alcohol had given rise. Sir Thomas Watson refers to two cases in which persons were attacked

by hemiplegia while in the act of sexual intercourse; and Dr. Wilks recently mentioned to me a similar instance.

The symptoms of these various affections of the cerebral arteries present many points of resemblance, but they also differ in several important respects. It is impossible for us to enter upon their study without having first considered the nature and significance of certain phenomena promising the highest clinical interest, each of which may occur as the result of any one of the arterial diseases in question. I refer to *Hemiplegia*, *Aphasia*, and *Apoplexy;* and I now propose to consider them separately, passing on afterward to consider the different ways in which they may be grouped together, and how far it is possible to distinguish from one another the various conditions which may give rise to them.

A. *Hemiplegia.*—It has already been mentioned that one of the leading effects of diseases of the brain—at least of such diseases as are confined to one lateral half of the organ—is *hemiplegia*, or loss of power in the arm and the leg and certain other parts of the body on one side. Now, this form of paralysis presents certain peculiarities which require careful study. In the first place, it is to be noted that the limbs which are affected are always those on the side opposite to the lesion in the brain. This, as is well known, depends upon the fact that the motor columns cross over in the anterior pyramids; those from under the right half of the pons Varolii passing down into the left half of the spinal cord, and *vice versâ*. Thus any unilateral affection of the nervous centres, situated above the decussation of the pyramids, if it causes paralysis at all, invariably causes paralysis of the opposite side. There are, indeed, some cases on record which have been supposed to be exceptions to this rule, and these have been recently marshalled forth by Dr. Brown-Séquard, who believes that they establish the possibility of the occurrence of hemiplegia on the same side as the lesion on which it depends. But, whatever the absolute number of cases of this kind that can be collected from medical journals and other sources, their relative frequency is altogether insignificant in comparison with that of those cases on which the rule of decussation is based. And I entirely agree with those critics who think that all the supposed exceptions are mistakes. In some of them it is most likely that the reporters have severally written "right" for "left," or "left" for "right," in making their notes. Every one who has done much case-taking will admit how very easy it is to fall into this error; and I may mention that our post-mortem records at Guy's Hospital contain at least two cases in which it is stated that the brain disease was on the same side as the paralysis. One of these is in the handwriting of Dr. Wilks, who certainly must have been greatly struck by such a fact, if he had observed it. But he leaves it entirely without comment, and I think there can be no doubt that it was a slip of the pen. In other cases, in which pathologists have deliberately thought that they had made a similar discovery, it is probable that there really were two lesions; one, the more conspicuous, on the same side as the hemiplegia; the other, on the opposite side—perhaps, a minute spot of softening—which may have really caused the paralysis, although altogether overlooked at the autopsy. I was once present at a post-mortem examination when such a mistake was very nearly committed.

I have spoken of this form of paralysis as affecting the arm and the leg; and it is important to note that there are definite limitations of the extent to which other parts of the body are capable of being paralyzed in what may be termed "common cerebral hemiplegia"—the *hémiplégie cérébrale vulgaire* of Charcot. The nature of these limitations can be most conveniently studied in the case of the facial muscles. Commonly all that is

noticeable is a little loss of expression about the mouth, which may appear to be drawn over to the opposite side. Todd ascribed this to impaired power of the buccinator muscle (which he believed to be supplied by the fifth nerve), and maintained that the facial nerve escaped altogether. Such a view, however, is quite untenable. The right doctrine appears to me to be that which Dr. Broadbent suggested, in 1866, in an article in the "*Medical and Chirurgical Review.*" It so happens that in 1860 or 1861 I had read before the Physical Society of Guy's Hospital a paper in which I had developed the same theory; and I may be allowed to quote from this communication, which is now before me, although, as I did not have it printed, the priority rests with Dr. Broadbent. The case on which it was founded was that of Henry W—, admitted into Guy's Hospital on Feb. 20th, 1860, under the care of Dr. Gull, for right hemiplegia, which was almost complete. The state of his face was very interesting. When told to shut his eyes, he could close them both. But whereas he could wink with the left eye alone, he could not do it with the right alone; and when I asked him to compress the eyelids firmly I found that the left orbicularis palpebrarum alone obeyed the will, the right one simply bringing the upper and lower lids into contact, without any wrinkling of the skin. This, and the further observations which I made on the same patient, led me to propound the law that "these movements, which are performed in harmony by the two sides of the face or body, remain unimpaired in hemiplegia." Dr. Broadbent afterward laid down the same law in very similar terms, and it has received general acceptance. The exemption of movements which are habitually bilateral (but not necessarily performed by corresponding muscles of the two sides) is seen in the case of the muscles of the eyeballs, of the masticating muscles, of those of the neck, chest, and abdomen, all of which retain their power.

It is, of course, to be understood that hemiplegia from a lesion of one side of the brain is not necessarily so complete as to present a maximum loss of power. Since the year 1860 I have not myself met with a second instance in which the affection of the facial muscles could be so accurately determined in accordance with Dr. Broadbent's rule. Not infrequently the face escapes entirely. Sometimes the leg can be moved perfectly, while the arm is completely paralyzed. Almost invariably, when recovery takes place, the patient regains power in the arm earlier than in the leg; he may, perhaps, be able to walk with a crutch at a time when he can scarcely move the fingers at all, and if any part remains permanently paralyzed it is said to be the upper limb. These facts are commonly attributed to the circumstance that the movements of the arm are more independent than those of the leg. It is regarded as a further illustration of the rule laid down in the last paragraph that the limb of which the movements are more highly specialized should be the more constantly and the more persistently affected. I must confess that this explanation seems to me unsatisfactory, and I have always been inclined to think that a certain part of the fan-shaped expansion of the crus cerebri may be uniformly more damaged than other parts, by the lesions that cause hemiplegia. It would certainly accord well with anatomical facts if the innermost fibres of the internal capsule and corona radiata should often escape, since they seem to lie outside the area to which the branches of the middle cerebral artery are distributed.

On the other hand, Dr. Broadbent's law must not be taken to mean that one can never make out any loss of power whatever in muscles which are used in association with muscles of the opposite side. All that we are justified in saying is that there is in no case anything like complete paralysis of such muscles. The tongue, for instance, is very commonly involved to some extent, so that when it is protruded from the mouth its tip is more

or less distinctly turned toward the side on which the limbs are paralyzed, and the patient often articulates very indistinctly and mumbles in his speech; so much that he may in some cases be unintelligible.

I may here remark that it was formerly considered difficult to understand why the ninth and the seventh nerves—arising, as they do, above the decussation of the anterior pyramids—should, nevertheless, resemble the nerves of the limbs in showing impairment of function on the side opposite the lesion in the brain. I believe that all physiologists are now agreed that this depends upon the fact that fibres which pass to the roots of those nerves undergo an independent decussation within the substance of the medulla oblongata or pons.

Again, it has been proved by several independent observers that the chest sometimes does not expand so fully on that side on which the limbs are paralyzed, at least when a deep breath is being taken. Dr. Walshe ("*Lancet*," 1849) determined this by actual measurement. Dr. Broadbent himself points out that when a hemiplegic patient is made to raise himself from the recumbent posture by his abdominal muscles alone, the rectus on the paralyzed side can be felt to act somewhat less powerfully, and a little later, than on the healthy side. So, also, if he is told to close the jaws firmly, the masseter and temporal muscles do not contract quite simultaneously or with equal force on the two sides.

Dr. Hughlings Jackson thinks that it is possible for a lesion of one hemisphere to produce a complete paralysis of all four limbs, but I am not aware on what evidence this opinion is based, or that any cases of universal loss of power have been recorded from an affection of such a nature that it could not possibly have compressed nor interfered with the opposite side of the brain. It is true that, as Dr. Jackson many years ago pointed out, patients suffering with aphasia, from disease situated above the corpus striatum on one side, are not rarely altogether unable to protrude the tongue from between the teeth. I have several times observed this, and it seems clearly to be an instance of double paralysis from a unilateral lesion; but I must confess I should be inclined to attribute it to an "inhibitory influence" transmitted downward upon the associated nuclei of the ninth nerves in the pons, rather than to believe that both sides are really "represented" in each cerebral hemisphere—which is the hypothesis suggested by Dr. Jackson ("*Brit. Med. Jour.*," 1874) to account for the supposed occurrence of a general paralysis of all the limbs as the result of an affection of a single hemisphere.

In some cases of hemiplegia, the eyes, instead of being directed straight forward, are turned to one side, and this side is always opposite to that of the paralysis, so that, for the purpose of artificial memory, one may say that the patient is *looking toward the lesion in his brain.* The "conjugate deviation" of the eyes is by some writers regarded as due to a paresis of certain ocular muscles, the external rectus on the side of the paralyzed arm and leg, the internal rectus on the other side. Others have supposed it to depend upon spasm of the antagonist muscles, and thus to be analogous to the state of "early rigidity," which will presently be described. But the face, also, is sometimes drawn over, away from the hemiplegic limbs, and then Prévost has found that the sterno-mastoid and the trapezius muscles are not necessarily in a state of contraction. Moreover, Dr. Jackson has pointed out that when "early rigidity" comes on in the arm and the leg, the "conjugate deviation" becomes reversed, the eyes being now directed toward the side of the paralysis; a case in point occurred at Guy's Hospital in 1867, in the practice of Dr. Rees. Thus it seems clear that this remarkable symptom is not of a spasmodic nature, and I think that Vulpian and Prévost are probably right in supposing that it represents, in a rudimentary form,

the rotatory movement (*mouvement de manège*) which is so commonly developed in animals after unilateral injuries to the head, for the circles in which most animals revolve always lie on that side on which the lesion is situated. Indeed, a woman under the care of Charcot, for left hemiplegia with conjugate deviation of the eyes, was several times found lying across her bed with her head to the right and her feet to the left; and another of the patients whose cases are related by Prévost showed a tendency to turn round like a top when placed upright. This writer has proved that the occurrence of conjugate deviation of the eyes is not limited to affections of any one particular region of the brain; among fifty-eight cases which he relates, there were four of meningeal hemorrhage, and four others in which the superficial convolutions were alone diseased. It is generally transitory —lasting only a few hours or a day or two—but sometimes it persists for several months.

We have seen that in "common cerebral hemiplegia" there is never a complete paralysis of any one of the cranial nerves, but it sometimes happens that such an affection of the third or of the seventh nerve is associated with hemiplegia, and then the seat of the disease can be determined with more than usual exactness. The facial nerve may be affected in two different ways. On the one hand, the lesion, if it is situated in the upper part of one lateral half of the pons, may destroy the facial nucleus, and so cause a total loss of power in the face on the same side on which the arm and the leg are paralyzed; and since the nucleus of the sixth nerve is very close, paralysis of the external rectus may, perhaps, be present in addition. On the other hand, an affection of the lower part of the pons on one side may cut the fibres of the facial nerve as they are passing across the motor tract, after they have decussated on their way outward from their nucleus. The paralysis of the face is then on the opposite side to that of the limbs. It might even happen that an extensive unilateral lesion of the pons should cause paralysis of both sides of the face by destroying the nucleus of one facial nerve, and the fibres of the other one.

Again, the association with hemiplegia of paralysis of the ocular muscles supplied by the third nerve shows that the seat of disease is the crus cerebri. The muscles in question are those affected on the side opposite to the paralyzed arm and leg. An instance of this has been placed on record by Dr. Hermann Weber ("*Med.-Chir. Trans.*," vol. xxviii). A man, aged 52, was attacked with paralysis of the right side of the body and of the muscles supplied by the left third nerve; he died after two months, and an oblong clot of blood was found in the left crus. Following Gubler, recent French writers have given a special name, that of "*Hémiplégie alterne*" to cases in which either the facial or the third nerve is affected on the side opposite to the limbs. This expression has been translated into English as "alternate hemiplegia," but I think it is not worthy of adoption, since it is likely to be misunderstood, and is not really necessary. "Crossed hemiplegia" is a better term.

There is still something to be said with regard to the *regional diagnosis* of hemiplegia due to disease situated in the parts of the brain which are higher than the crus cerebri. And, first, it has been a moot question whether paralysis of the arm and leg can be caused by a lesion limited to the white substance of the hemisphere, or to the convolutions, or affecting both structures to the exclusion of the basal ganglia. Mr. Hutchinson has insisted on the frequent occurrence of this symptom in cases in which meningitis appears to be limited to one lateral half of the brain, as so often happens after surgical injuries, but then it is difficult to say how far the inflammatory process may extend into the interior of the organ. Until quite recently, the numerous instances in which extensive destruction of the super-

ficial parts of the cerebrum has been followed by no definite signs of loss of function have been regarded by most physiologists as proofs that hemiplegia could not result from such an affection. And even when certain experiments made by Hitzig and by Ferrier (to which I shall more fully allude in the next chapter) had shown that irritation of particular convolutions caused movements in the opposite limbs, it was still held by many observers that no disease of these convolutions could produce a permanent paralysis in accordance with a distinction, laid down by Dr. Hughlings Jackson, between the effects of "discharging" and those of "destroying" lesions. These experiments, however, have led pathologists to reopen the question; and they have also greatly facilitated its investigation, by reducing to comparatively narrow limits the area within which cortical affections causing paralysis are to be sought. The result has been that certain French and German physicians have found that diseases of the upper parts of the ascending frontal and parietal convolutions, and of the adjacent parts of the frontal and of the superior parietal convolutions may, if sufficiently extensive, give rise to permanent hemiplegia, although the corpus striatum remains perfectly healthy. I have already, at p. 492, described softening of this particular region of the brain as an occasional result of obstruction of the Sylvian artery beyond the origin of its "central" branches, whether by thrombosis or embolism. Charcot speaks of having made a collection of cases of this kind. The paralysis seems to have been undistinguishable from that caused by a lesion of the basal ganglia; it is particularly mentioned that the consecutive degeneration of the spinal cord was present, which will be described a little further on. On the other hand, Charcot has found that similar affections of the sphenoidal and occipital lobes caused no permanent hemiplegia.

Indeed, it is possible that we may hereafter be able to carry the matter still further, and to localize in particular convolutions the lesions causing paralysis of different parts of the body. This question, however, will be more conveniently discussed in the next chapter.

As regards the possibility of diagnosing what part of the basal ganglia is the seat of disease, Charcot maintains that, whenever a lesion is limited to one of the gray nuclei of the corpus striatum, the hemiplegia is always transitory and very often incomplete; whereas, if the internal capsule is involved, it is commonly complete and persistent. But he destroys much of the force of this remark by adding the obvious statement that neither the nucleus caudatus nor the nucleus lenticularis is ever *wholly* destroyed without other parts being affected at the same time. According to this writer the limitation of paralysis to a single limb is never due to the fact that the morbid process is confined to a special seat within one of the ganglia. Dr. Jackson's way of expressing the same doctrine is by saying that "the whole corpus striatum is represented in miniature by every single part of it." And Dr. Ferrier found that irritation of this part of the brain by faradic currents caused a general contraction of the muscles on the opposite side of the body, without its being possible to differentiate individual movements. But in spite of these high authorities I cannot help thinking it is possible that, at least in the internal capsule, the fibres which belong to the upper limb may hereafter be distinguished from those that pass to the lower limb. We shall hereafter see that Hitzig and Ferrier state that the cortical centre for the leg muscles lies nearer the median plane than that for the arm muscles. It seems to me a fair inference that if there be a distinction of fibres within the internal capsule, those for the leg should be situated further inward (or forward) than those for the arm. Now, it is certain that in cases of cerebral hemorrhage the inner and fore part of the capsule often escapes laceration, when the outer and hinder part is torn through.

May not this be the reason why the upper limb so often remains powerless after the patient has regained the use of his lower limb? And may we not attribute to the same cause the fact that the arm sometimes becomes paralyzed alone, the leg escaping altogether? Charcot himself thinks that a broad line of distinction must be drawn between affections limited to the anterior two-thirds of the internal capsule and those which involve its posterior third. In the former case there is no loss of sensation; in the latter there is hemianæsthesia, affecting the same side as the paralysis. He even believes, in opposition to some other authorities, that a lesion limited to the posterior third of the internal capsule may cause loss of sensation without any loss of power. The anæsthesia affects not only the limbs of one side, but also the corresponding half of the body and of the head. It includes an insensibility to pain and to changes of temperature, as well as to tactile impressions. It extends to the deeper parts and also to mucous surfaces; the patient feels nothing when his muscles are made to contract by the application of a current of electricity. It affects the special senses of hearing and smell and taste. It also affects the sense of sight, but there is a difference of opinion with regard to the kind of imperfection of vision which results. According to Dr. Hughlings Jackson this is a "lateral hemiopia," corresponding halves of each retina being affected in such a way that the patient is unable to see toward the paralyzed side. Dr. Jackson, in 1875, had seen some thirteen cases of hemiplegia attended with the symptom in question. In one instance, in which the paralysis was on the left side, a post-mortem examination was made by Dr. Gowers, who found a single lesion of the right thalamus, the posterior half of which was softened and of a grayish-yellow tint ("*Lancet*," May, 1875). The hemiopia had been of the most marked description; the man had sometimes seen only half of a word, reading "land" for "midland," and remarking to his son that "Liver" was a queer name, when it really was "Oliver." In opposition to the recent statements of Mandelstamm, Michel and others, I am still inclined to adhere to the view that at the chiasma only the inner parts of the optic nerves decussate. This view, to which I shall refer in fuller detail elsewhere, seems to afford a satisfactory explanation of the occurrence of "homologous lateral hemiopia," as Charcot terms that symptom. But Charcot himself denies that such is the affection of sight which forms part of hemianæsthesia. On the contrary, he maintains that there is a "crossed amblyopia"—an impairment of vision in both halves of one eye—the one on that side of the body which is deprived of sensation. He says that the acuteness of vision is often diminished by one-half, or even in a still higher degree. The visual field is generally narrowed on all sides, but for some colors the range is far more limited than for others. The first to be lost is violet, then, in succession, green, red, orange; the colors for which vision is retained longest are yellow and blue, but even they may at last be indistinguishable, so that every object appears of a brown color, as if painted in sepia. If Charcot's statements are correct, the only possible explanation seems to be that which he gives, namely, that there is a second decussation of the optic tracts where those fibres cross over which failed to do so in the chiasma, and that this is situated beneath the corpora quadrigemina. The only way in which Dr. Jackson's observations can be reconciled with such a hypothesis, is to suppose that in his cases the disease involved one optic tract, but in the account of the autopsy made by Dr. Gowers it is expressly stated that the crus was unaffected. Charcot even quotes a case of Dr. Bastian's, in which a lesion limited to one of the anterior corpora quadrigemina caused crossed amblyopia and not lateral hemiopia.

I must not omit to mention that some writers have described anæsthesia as

only rarely accompanying "common hemiplegia." Such statements, however, appear to have been based upon observations made on cases which were already of long standing. There is every reason to believe that anæsthesia is generally present in recent cases, although its recognition may be difficult, or even impossible, in patients whose consciousness is much obscured. It is, however, far more transitory than motor paralysis, often passing off in a few days, and sometimes still earlier. In this respect lesions of the brain exactly resemble those of the spinal cord and of the peripheral nerves; and probably the explanation given at p. 353 is applicable to the question now under consideration. Subjective sensations of formication and numbness are frequently associated with partial anæsthesia, and sometimes there is a condition of hyperalgesia, or increased susceptibility to painful impressions. Sometimes, too, spontaneous pains are experienced in the joints on the affected side, and particularly in the shoulder joint.

Hemiplegic limbs are often reddened and warmer than those of the opposite side—a difference which is attributed to interference with the functions of the vaso-motor nerves. The inequality of temperature commonly amounts to something less than one degree Centigrade; after some months it ceases to exist. In some cases there is excessive sweating; sometimes a slight œdema is discoverable. I remember one case in which dropsy (of renal origin) was limited to the arm and leg that were paralyzed. Charcot lays stress on the frequency of rapid sloughing of the skin over the gluteal muscles—an acute bed sore, as he terms it. One can hardly suppose that, in cases of this kind, there is much difference in the amount of pressure upon the two sides of the buttocks; and he regards the affection as a more or less direct effect of an interference with the nutrition of the tissues, caused by irritation of a particular region of the brain.

When hemiplegia is recent, the electrical reactions of the muscles are generally quite normal. Sometimes contractions are less readily excited than in the healthy side; sometimes, but very seldom, more readily. In the latter case, some amount of tonic spasm is always present; the elbow is generally flexed, and resists extension; the fingers are bent in upon the palm of the hand. This condition was described by Dr. Todd under the name of "early rigidity," and was attributed by him to laceration of the surrounding healthy brain substance by effused blood; and this explanation has been adopted by most subsequent writers, although I doubt whether one could demonstrate that there is more injury to the nervous tissues than in cases in which the muscles remain flaccid. Durand-Fardel, indeed, ascribed the occurrence of tonic spasms in hemiplegic muscles to the escape of blood into the lateral ventricle; but it does not appear that any definite facts can be adduced in favor of such a view. So long as the muscles are in a state of early rigidity, they do not waste.

After Effects.—Hemiplegia is often recovered from, the patient gradually regaining the use of his limbs, until at length no difference can be made out between the two sides. I have already remarked that, as a rule—to which there are very few exceptions—the leg recovers before the arm; the patient may be able to walk quite well with a crutch at a time when the hand is still motionless. Trousseau, however, relates two instances in each of which power was restored in the arm earlier than in the leg, and he maintains that when this is the case the prognosis is particularly unfavorable.

But in many cases hemiplegia is permanent. The patient may for the rest of his life be utterly unable to move any part of the upper limb, or he may regain power in it to a greater or less extent. In almost every case of this kind the affected muscles pass into a state of contraction, which is

commonly known as "late rigidity." Bouchard found that it was absent in only one out of thirty-two cases that he examined. It generally consists in a flexion of the various joints; the elbow is bent at nearly a right angle, the wrist is pronated and folded on the forearm, and the fingers are drawn in upon the palm of the hand, so that the nails may penetrate the skin and produce painful ulcerations. Very much more rarely, a position of extension is assumed; the elbow may then be straight, the wrist may be thrown back, the fingers may assume the griffin's-claw attitude, which I have described at p. 350 as occurring in progressive muscular atrophy. The leg is less apt than the arm to become contracted, even when it remains paralyzed; if at all, the hip and the knee joints are flexed, and the heel drawn up. This form of rigidity sets in so gradually that the exact period at which it begins cannot be determined. Bouchard speaks of a case in which it was present in a marked degree two months after the apoplectic attack which had caused the hemiplegia. Sometimes one can overcome the spasm by a little traction upon the affected parts; sometimes it resists the application of an amount of force which causes the patient severe pain. The muscles are generally much wasted, and feel like tight cords beneath the skin.

Until recently it was supposed that late rigidity was caused either by the slow contraction of an apoplectic cyst, or by an inflammatory process in the surrounding brain substance. But in 1866 Bouchard suggested, in the "*Archives Générales*," that it was probably an indication of the supervention of certain changes in the lower parts of the cerebro-spinal axis, which had been pointed out by Türck in 1853, and had been previously noticed by Cruveilhier as occuring after unilateral lesions of the brain. These changes are to some extent visible to the naked eye; the crus cerebri is smaller than on the healthy side, and flattened, and presents a gray streak toward its inner edge; the corresponding half of the pons may be flattened; the anterior pyramid is of a grayish color and wasted, so that the decussation of the pyramid is more conspicuous than usual. Below this point there is no marked alteration on the surface, but transverse sections of the spinal cord may show, in the lateral column of the side *opposite* to the lesion in the brain, a triangular gray patch, which gradually diminishes in size, but is traceable down to the lumbar enlargement. Or it may be only after hardening, and with the aid of the microscope, that the change is discoverable. It is found to consist, not only in a degeneration of the nerve fibres which pass down the cord from the brain, but also in the formation of a delicate, new connective tissue. In other words, there is a sclerosis of a definite tract of the lateral column, which, however, also contains a number of nerve fibres which probably arise from the spinal nuclei, and therefore escape the degenerative process. Now, Bouchard's theory is that these unaltered nerve fibres become irritated and cause the contracted state of the muscles. In support of it, he adduced the analogous rigidity of the lower limbs which occurs when the upper part of the spinal cord is compressed (see p. 437). Charcot has since developed the same doctrine still further, and maintains that the sclerosis may spread as an independent affection to the anterior gray cornua, causing the muscles to become again flaccid and to undergo a rapid degenerative atrophy; or to the posterior cornua, inducing a partial anæsthesia; or, lastly, to the opposite lateral column, giving rise to a contraction of the other lower limb, as is supposed to have occurred in a case of Dr. Bastian's. More recently, however, Hitzig has proposed a very different explanation of "late rigidity." He points out ("*Arch. f. Psych.*" 1872) that prolonged rest is followed by a relaxation of the contracted muscles; that, for instance, after a night's sleep, paralyzed limbs are commonly flaccid and supple, and remain so until the patient

begins to exert the opposite limbs in getting out of bed, or in some other way. Again, he has observed that voluntary efforts with the unaffected arm often cause an increase in the rigidity of the affected arm. In one of his cases, as soon as the patient was made to lift a heavy weight with his left hand, the thumb and forefinger of the paralyzed right hand became quite stiff, although they had before been free from spasm. He supposes that the contractions of hemiplegic limbs represent an excess of those co-ordinated movements in distant parts which naturally accompany every action of the body. He assumes that the spinal centres are in a state of irritation, and that, as a consequence, the movements in question become far more marked than under normal conditions. In further illustration of his meaning, he instances the well-known fact (of which I remember to have seen an example) that patients who are altogether unable to move an arm by any voluntary effort sometimes have it thrown violently into the air when they yawn or stretch themselves.

To decide between the rival theories of Bouchard and of Hitzig would be very difficult. And I am the less disposed to attempt it, because I shall hereafter have to admit that there is a large number of other spasmodic affections of which the seat and nature are still altogether unknown. Moreover, I must draw attention to the fact that "late rigidity" bears a close relation to certain forms of mobile spasm which may also present themselves in hemiplegic limbs, but more rarely, and only when the loss of voluntary power is incomplete. They vary greatly in character in different cases. Sometimes a more or less violent tremor is observed, which may either continue even while the part is at rest, or occur only when it is made to execute some voluntary effort. Charcot, for example, mentions an instance in which the act of carrying a glass to the mouth was attended with rhythmical movements, so that the liquid in it would be thrown in all directions. In other cases spasms are seen, which are more or less choreiform in character, so that Dr. Weir Mitchell and Charcot have spoken of a "post-hemiplegic chorea." Lastly, some cases are attended with remarkably slow movements, principally affecting the thumb and fingers, and exactly like those described by Dr. Hammond under the term "athetosis." The name (ἄθετος, without a fixed position) is intended to signify that the parts concerned in the spasm cannot be kept still. They are constantly moving, without any voluntary effort on the part of the patient. The fingers are alternately flexed and extended, with varying degrees of adduction or abduction, so as to give the hand a very grotesque appearance; and the toes may present similar changes of position. Cases of this kind have been described in Great Britain by Dr. Allbutt and Dr. Gairdner; and Dr. Gowers has discussed "post-hemiplegic disorders of movement" very fully, in the "*Med.-Chir. Transactions*" for 1876.

B. *Aphasia.*—Another symptom which is frequently present in the diseases under consideration is loss of speech—the defect being of a special kind and presenting characters which merit careful study. It has within the last few years received several different names—alalia, aphemia, aphasia; but of these the last is now universally adopted, while the first (as I have already stated) is reserved for a different affection. It is altogether distinct from a mere impairment of articulation, such as I have described as occurring in bulbar paralysis, although this may itself form part of a common cerebral hemiplegia, being due to imperfection in the movements of the tongue and palate. In either of these conditions the speech is more or less thick and difficult to be understood. But in aphasia, if the patient can utter a word at all, he commonly pronounces it quite clearly and distinctly. He may be altogether mute. Or he may occasionally, when excited,

ejaculate some oath, which Dr. Jackson will hardly admit to be truly a part of language—particularly as it is often impossible to get him to repeat it deliberately. Or he may possess the power of saying two or three words or short sentences; these he uses sometimes correctly, but often in contradiction to his real meaning, and in answer to every question that is put to him. Thus Trousseau relates the case of a man named Paquet, who for four months said nothing but *cou-si-si*, *cousisi*, and who kept uttering these three syllables on all occasions, whether he was in a passion, or wished to express gratitude, or wanted to ask for or refuse something. Only, if he became very excited, he would sometimes say *sacon*, *sacon*,—probably an abbreviation of the oath *sacré nom de Dieu*. Dr. Broadbent had a patient who could scarcely say anything but *Oh, shameful! shameful!—Oh, pity! pity!* Of two celebrated cases that occurred to M. Broca, one was that of a man who said *tan*, *tan*, to every question for twenty-one years; another possessed four words, *oui*, *non*, *trois*, and *toujours*. If there is a more extensive vocabulary, the patient is very apt to use one word for another. There may then be a similarity in sound between the two, as when *purging* takes the place of *perjury*, *pamphlet* of *camphor*, *dispersion* of *dispensary*. Or the resemblance may be in meaning, as where a patient of Dr. William Ogle's said *boat* instead of *tub*, or (by an odd confusion of ideas) substituted the word *two-shilling piece* for *spectacles*. The writer to whom I have just referred draws special attention ("*St. George's Hosp. Rep.*," vol. ii) to the fact that grammatical form is always observed; substantives are used in the place of substantives, verbs for verbs, numerals for numerals. Thus M. Broca's patient employed *trois* to express any number, but corrected what he said by holding out the proper number of fingers at the same time. Dr. Broadbent's patient possessed only one name for a locality, namely, Burlington, where she had lived as a child; and she used this when she wished to name any place whatever. One curious circumstance is that a person who had known two or more languages may entirely lose the power of speaking one of them, while he retains that of conversing in the others. Trousseau gives an instance of this in a Russian who before his illness spoke French like a Parisian; and Bastian says that he has seen two similar instances, one being in a German who had long been resident in England. Sometimes, a patient who can utter only one or two words by himself is able to repeat a good many other words, if he is prompted. Thus Trousseau relates the case of a man named Marcon who could only say *Ma foi* and *'Cré nom d' un cœur*, but who, when asked, "Are you from the Haute Loire?" replied *Haute Loire*, and then to the query, "What's your name?" echoed again *Haute Loire*. "Your profession?" *Haute Loire*. "But your name is Marcon?" *Yes, sir*. "What department do you come from?" *Marcon*, and so forth. We shall hereafter see, however, that such a case as this is not to be considered as one of *mere* aphasia, although that affection was, of course, present. I should, for my own part, be disposed to refuse the name of aphasia altogether to such a case as that recorded by Dr. Bateman as having occurred at the Sâlpetrière, of a woman who, although she said nothing of her own accord, repeated everything that was said to her, and mimicked all the gestures of the students and of the other patients near her. We shall presently see that the view taken of such cases involves important questions as to the pathology and seat of the disease by which they are caused.

In some, but not in all, cases of aphasia the patient loses the power of expressing himself in other ways, as well as in speech. There is, for example, the act of writing, incapacity for which has recently been called "Agraphia." A person who is aphasic is sometimes able to make all his thoughts known with his pen. Trousseau relates the case of a carrier

belonging to the Paris Halles, who came into the consulting room making signs that he could not speak, and handed in a note in which the history of his illness was detailed. He had written it himself, and it was perfectly well worded. A few days previously he had suddenly lost his senses, and remained unconscious for nearly an hour. When he came to, he had no paralysis, but he could not articulate a single word. In the course of five or six weeks he completely recovered; but what was remarkable was that during the whole course of the disease he could manage all his affairs by substituting writing for speech. Such cases are very exceptional. As we shall presently see, paralysis of the right hand is very often present in those who have aphasia; and, of course, it is then difficult to test the writing. Trousseau, however, mentions one patient, who said *oui* to everything, and who was unable to write even with the left hand. One way of ascertaining whether the power of combining letters to form words is preserved, is to give such a person the loose wooden letters that children play with, asking him to spell his name or to put together a sentence. He may be quite incapable of doing so; indeed he may not even be able to pick out the letters that make up the one or two words that he is perhaps able to articulate. Trousseau's patient, for instance, whose one word was *oui*, could not point to the letters o, u, i, in the title page of a large quarto volume, containing those letters. Indeed, the impairment of the power to write is sometimes very marked, when the speech is comparatively little affected. Dr. Jackson records the case of a woman who could talk fairly well, although she frequently made mistakes in speaking, and called her children by many names; but when told to sign her name she wrote *Sunnil Siclaa Satreni*, in which there was not the slightest resemblance to the real name either in sound or spelling; and when told to write her address she put down *Sunesr met ts mer tina—lain.* We shall hereafter see that if there is much impairment of the intelligence the value of agraphia as a symptom is much diminished; but both Trousseau's patient and Jackson's appeared to have full possession of their mental faculties.

Again, it has been shown that in some cases of aphasia the memory of *gestures* is lost. Trousseau says that such a patient may be unable to *put on* the face of a person who is crying, although when he feels grief the expression of his countenance shows it clearly enough. The patient, Paquet, who said nothing but *cousisi*, was the subject of the following experiment: Trousseau first held out his two arms and hands, and moved his fingers, as though he were playing the clarionet, and asked this man to imitate him. He immediately executed the same movements with perfect precision. When asked whether he knew that the attitude was that of a man performing on the instrument in question, he would assent by nodding his head. Yet, when told, a few minutes afterward, to place himself in the very same attitude, he seemed to think, and was most times unable to do it. So, also, a patient whose case was carefully studied by Dr. Scoresby Jackson ("*Edin. Med. Jour.*," 1867) was found to be totally unable to play the piano. He put himself in the proper position, and placed his fingers on the keys, but he could not play a single bar, not even of a piece of music with which he had been familiar before his illness. With the left hand alone he managed a bar or two very slowly, but transposed several notes. Yet he could hum the same tune pretty well. On the other hand, games of skill are often practiced by aphasic persons with unimpaired accuracy. Dr. Scoresby Jackson draws special attention to the skill with which his patient played draughts; and Trousseau's man Paquet could play backgammon and dominoes perfectly, knowing all the tricks of those games, and cheating when he found himself losing.

It is important to notice that the very words which an aphasic patient is unable to utter are perfectly understood by him when spoken by another person. This, indeed, is but an illustration of a broad distinction which is traceable throughout the faculty of speech. A child learns the meaning of words addressed to it by others long before it can itself speak. A person partially acquainted with a foreign tongue recognizes many words, if spoken distinctly by any one else, which he would have been unable to call from his memory if he wanted them in conversation. Nay, we all of us are accustomed to a far wider English vocabulary in the daily speech of others than we ourselves make use of when we ourselves talk. Adopting terms used by Dr. Moxon, we may say that there is a great difference between "incoming" and "outgoing" language; or, following Dr. Broadbent, that words are to be considered in two distinct aspects, first as "intellectual symbols," and then as "motor processes." In aphasia it is the *outgoing language* and the *motor processes* that are interfered with.

Now, there are evidently two ways in which such a limitation of the aphasic condition can be accounted for. It may be that the perception of the meaning of the words spoken by others, and the uttering of words for one's self, are respectively functions of different parts of the brain. Or, again, it may be that both "incoming" and "outgoing" language have their seat in the same locality, or are both of them functions of the cerebral hemispheres as a whole, but that the latter requires for its execution the intervention of some subordinate nervous centres which have nothing to do with the former. The second of these hypotheses seems to me to be the correct one; it has been defended with great ability by Dr. Broadbent in the "*Med.-Chir. Transactions*" for 1872.

Within the last few years it has been established that aphasia—including agraphia when that is also present—is almost invariably dependent upon a lesion of the left side of the brain. This is not, perhaps, a new discovery, for Dr. Marc Dax, of Sommières, read a paper on the subject before a Medical Congress at Montpellier in 1836, but the paper is now lost. However, I do not suppose that any physician, either in Paris or in London, was acquainted with the views of M. Dax when, in 1861, M. Broca published in the "*Bulletin de la Société Anatomique*" a case which has formed the starting point of all our modern knowledge. It may seem strange that the association of right hemiplegia with loss of speech escaped the notice of any one of the acute clinical observers of the first half of the present century; but it was doubtless incongruous with the views which then prevailed as to the cerebral functions, and, indeed, it still remains an isolated fact, and one that needs to be specially investigated and explained. The existence of such an association is beyond question, and can at any time be determined by clinical observation. If a number of hemiplegic patients be taken, it will always be found that many of those in whom the paralysis is on the right side are speechless, whereas, probably, every one among them whose left arm and leg are paralyzed, will be able to articulate as distinctly as is compatible with the condition of the tongue and lips. Moreover, in a certain proportion of cases aphasia will be found to exist without any paralysis, the lesion being then almost invariably in the left side of the brain. The exceptions just hinted at are themselves based upon a rule, namely, this, that in left-handed persons loss of speech goes with paralysis of the left limbs, and not with that of the right limbs; or, in other words, that it depends upon disease of the right hemisphere instead of the left. Examples of such an association have been recorded by Dr. Jackson ("*Med. Times and Gaz.*," 1866), Dr. John Ogle ("*Lancet*," 1868), and Dr. Wadham ("*St. George's Hospital Reports*," 1869).

The next fact in the pathology of aphasia is that it is caused by lesions of a particular part of the surface of the brain on the left side, and can never be the result of disease limited to the basil ganglia. Now, Bouillaud was long ago led by clinical observation to locate the faculty of language in the anterior lobes of the brain, and, indeed, Gall had previously suggested the same seat for it. But it was not until a second case of M. Broca's was published, in November, 1861, that anything like precision of localization was arrived at in regard to loss of speech. The case in question is that of a man named Lelong, aged 84, who after an attack of apoplexy lost the power of uttering all words, with the exception of four, although he knew all that was said to him, and could make himself understood by gestures. He died at the end of a year, of a fracture of the femur. At the post-mortem examination the posterior third of the second and that of the third left frontal convolutions were found to have been destroyed by softening, their place being occupied by a collection of fluid in a cavity, the walls of which were of an orange-yellow color, from the presence of altered blood.

Ever since the third left frontal has been known as Broca's convolution, and the majority of pathologists have maintained that aphasia is always dependent upon a lesion involving it, or at least affecting some closely adjacent part of the surface of the same hemisphere. Now, this doctrine involves two distinct statements. In the first place, it asserts that no disease of the corpus striatum, or even of the white substance outside it, is capable of causing loss of speech. The general accuracy of this assertion is sufficiently indicated by the fact that cerebral hemorrhage, in the position in which it usually occurs, constantly leaves the speech unimpaired. And in 1876 Dr. Broadbent was able to say ("*Brit. Med. Journ.*," i, p. 436) that he knew of no case on record in which lesion of the corpus striatum not extending higher had given rise to this symptom. At first sight such a fact might seem inexplicable, since it is difficult to see how any effect can be produced by destruction of a convolution which may not equally follow the division of those conducting fibres which connect it with the basal ganglia, or even with the spinal centres. I do not know of any writer who has grappled with this difficulty, except Dr. Broadbent himself. He believes that there are two distinct routes by which impulses pass from Broca's convolution to the nerve nuclei in the pons, medulla oblongata, and medulla spinalis. One is the straight path through the left corpus striatum, the other is by commissural fibres which go to the third frontal convolution on the opposite side, and thence down through the right corpus striatum. So long as either of these routes remains open, speech is possible; it being, of course, assumed that the nuclei on opposite sides are so closely associated together by cross connections as to be set in action simultaneously. It may be noted, as one inference from Dr. Broadbent's hypothesis, that, if hemiplegia and aphasia occur together, the lesion must be one which either involves simultaneously Broca's convolution and the corpus striatum, or else affects an extensive area of the hemisphere in the neighborhood of the fissure of Rolando. Whichever is the case, the cause of the disease must almost always be obstruction of the Sylvian artery, whether by embolism, thrombosis, or syphilis. Now, I have already pointed out at p. 492 that whereas the central ganglia inevitably undergo softening when their blood supply from that artery is cut off, the convolutions may fail to present any obvious morbid change, although the interference with their circulation may, nevertheless, have been quite sufficient to impair their functions. In other words, if in making an autopsy one should overlook plugging of the Sylvian artery—and until lately it was not usual for pathologists to devote special attention to the condition of the cerebral vessels—one might easily suppose that the only disease was situated in the corpus striatum, and that this had caused the

aphasia. In July, 1877, I made an autopsy in which it would have been quite possible to commit this mistake; the patient was aphasic, and the only obvious lesion of the brain was in the corpus striatum, but the Sylvian artery contained an embolism at its very commencement.

In the second place, the doctrine referred to in the commencement of the last paragraph involves the assertion that aphasia cannot be caused by a lesion of any part of the surface, even of the left hemisphere, with the exception of the third frontal, or at least of some immediately adjacent convolution. Now, it may be that this position is not as yet completely established. Cases are very rare in which a morbid change is limited to a single spot on the convexity of the brain with sufficient accuracy to throw any light upon the question. If it be softening, it must be due to obstruction of a single branch of the Sylvian artery. We shall hereafter see that the effects produced by tumors are in some respects fallacious. I shall, however, have to mention a case in which the occurrence of transitory attacks of aphasia, in a woman who had cancer of the breast, led to the suggestion that she had a secondary nodule in the third frontal convolution of the left hemisphere of her brain, and in which this diagnosis was afterward verified by an autopsy. On the other hand, the facts cited by Trousseau, in opposition to the localization of aphasia in Broca's convolution, or in its close neighborhood, seem to me of very little value; and this is the opinion of Dr. Broadbent likewise, who said, in 1872, that he examined into all the apparently exceptional cases of which he had been able to find the records; and that it was remarkable how large a proportion of them broke down under careful scrutiny.

Reverting now to the theory of the causation of aphasia, we have to ask how it can be that loss of speech is always dependent on a lesion of the surface of the left hemisphere, except in left-handed persons, in whom the association is with disease of the corresponding part of the right hemisphere. By Trousseau this supposition was treated as an absurdity, opposed alike to analogy and to common sense. And I believe Dr. Moxon was the first to throw any light upon it, and to suggest an explanation which has virtually been accepted by almost every subsequent writer. His view ("*Med.-Chir. Rev.*," 1866) is that the two halves of the brain are originally symmetrical, and resemble one another in their functional capacities, but that in the course of education one side only becomes stored with those ideas of associated movements which are required for the performance of bilateral actions, such as are concerned in speech. Or, as he elsewhere puts it, one side of the tongue *guides* the other; just as the right hand guides the left one when they are made to execute similar motions, it being notorious that this requires infinitely less attention than the execution of opposed motions by the two hands respectively. Dr. Moxon does not more fully explain why the process of education should take place on one side of the brain rather than the other; and this has been regarded as a difficulty by some later writers. But, as we have seen, the associated movements which make up the act of writing, and in which only one hand is concerned, are so closely connected with those that constitute speech, that agraphia very commonly accompanies aphasia. Now, it is true that all men do not learn to write; but there is, perhaps, no savage who does not, from his earliest infancy, become accustomed to employ one hand rather than the other in gestures by which he supplements speech in communicating with others. And it was a suggestion made by Broca himself that this preference for the use of the left hemisphere for so many other purposes led to the education of convolutions of that hemisphere for the "motor processes" concerned in speech.

I have hitherto confined the subject of aphasia entirely to the loss of

"outgoing" language, as Dr. Moxon calls it; we have still to ask what are the relations of this affection to that other part of the faculty of speech—"incoming" language—which consists in the recognition of words spoken by others. Now, many modern writers, following Dr. Sanders and Dr. William Ogle, admit a separate variety of aphasia, which they term *amnesic* or *amnemonic*. The characteristic symptom of it is, they say, that the patient is able to utter words, provided that he has first heard them spoken by another person. In fact, one may be able, by prompting, to make him speak pretty freely, although he may be incapable of saying anything in reply to a mere question. Now, it is, in the first place, to be observed that the value of this test is limited to those cases in which it yields a positive result. If the man continues altogether speechless, after one has repeated again and again the word or the sentence which he is asked to utter, one gains nothing by the experiment. That the memory for "incoming" language is not altogether lost may, indeed, sometimes be obvious from the gestures of intelligence which such a person makes if the right word is suggested to him, and from his strongly-marked dissent when other words are substituted for it. Moreover, in those cases in which an attack of aphasia rapidly passes off, the patient may be able to give a complete account of all that occurred during his illness, and to state that his power of thinking was altogether unaffected. Prof. Lordat, for example, who once suffered in this way, said that he was able to combine abstract ideas, and to distinguish them accurately. Being accustomed to teach, he thought over the subject matter of a lecture, and found that he could dispose in his mind the chief points without difficulty, and introduce any changes that he pleased in their order. He thought of the Doxology, but he was not able to recollect a single word of it. I must leave it to the metaphysicians to discuss how far it is possible for the mental processes to be carried on without the revival of words, as symbols, in the consciousness. But I think it is clear that in M. Lordat's case the fault lay in the machinery of expression alone.

Again, when aphasia is incomplete, so that the patient can utter one or two words, and uses them in answer to every question that may be put to him, he is often perfectly conscious of the mistakes that he commits. Every physician has seen such cases, and will remember the shake of the head, the puzzled look, the smile—half amusement, half vexation—the repeated attempts to find the right word, the beam of satisfaction if by chance it at last comes off the tongue.

These facts seem to me to prove beyond dispute that the memory of language may be unimpaired even when there is complete aphasia. Now let us look at the other side of the question. Many patients affected with partial aphasia go on uttering the same word for months or years, without seeming to know that anything is amiss with them. Trousseau studied very carefully the case of an artist who, according to his own account, suffered from nothing but failure of speech, being able to understand perfectly all that he read, and being in full possession of his intellect. It turned out that he made the grossest mistakes in reading aloud, that he wrote one word for another without being aware of it, and that when asked to sketch a human figure, he drew like a child who had never been taught. The same writer, referring to another case, lays stress on the fact that the patient who read the newspaper, and expressed by signs that he understood it perfectly, was nevertheless in the habit of reading the sheet over and over again in the same day. And a girl who was under his care had for a year one book in her hands—a religious work, the "*Month of Mary*"—and almost always read the same page. He suggests that a very good test of the understanding of an aphasic patient is to take up a book, and to read a

few lines, telling the patient to follow with his eyes, and to turn over at the proper moment. The artist, whose case has already been referred to, could never do this correctly. From these observations Trousseau concludes, in spite of the facts mentioned in the previous paragraph, that in aphasia there is not merely loss of speech, but also impairment of the intelligence. But it seems to me that this writer did not sufficiently bear in mind the fact that the lesions which cause aphasia are in most cases such as interfere with the supply of blood to a large part of one hemisphere of the brain. We have seen that when this symptom is associated with hemiplegia it is almost invariably dependent upon obstruction of the trunk of the Sylvian artery. A good illustration of the principle that in such cases other symptoms may be present which have no necessary connection with the aphasia, is afforded by the fact that there is often anosmia—loss or impairment of smell—in the corresponding nostril. In the "*Med.-Chir. Transactions*" for 1870, Dr. William Ogle refers to seven instances of this: he supposes that the so-called external root of the olfactory bulb becomes "implicated when it is passing to the floor of the fissure of Sylvius." Dr. Ferrier's suggestion that softening extends to the *subiculum cornu Ammonis* (which he imagines to be the centre of smell) appears to be inadmissible, since that structure lies beyond the area of distribution of the Sylvian artery.

The question, therefore, is, not what degree of effect of understanding *may* be found in aphasic patients, but what amount of intelligence they are capable of retaining. Instances of pure aphasia, unattended with paralysis of the limbs, are especially worthy of study in reference to this question. I think it will be found, that while the loss of speech is often absolute, the memory may in such cases be unimpaired.

On the other hand, one is constantly seeing patients with right hemiplegia who go on for years, unable to utter a single word, or to communicate in any way with their dearest relations. It would be a sad thing to suppose that such persons are really in possession of all their mental faculties, and that they are living, as it were, imprisoned within an iron mask. We may, I think, dismiss such a notion. Indeed, it is certain that those who suffer from left hemiplegia, dependent upon any extensive lesion of the right hemisphere, manifest an equally marked impairment of intelligence, although they are not deprived of speech.

These considerations have led me to the conclusion that, although loss of speech is often associated with the inability to understand "incoming" language or to recognize mistakes made in "outgoing" language—and I think it would be convenient to transfer the use of the term *amnesia*, so as to make it correspond with this condition—yet the two things are essentially independent of one another, and the latter indicates the existence of a lesion extending far beyond the limits of Broca's convolution.

I have recently found that Dr. Broadbent has arrived at the same conclusion ("*Med.-Chir. Trans.*," 1872, p. 174). He, like myself, is firmly convinced of the truth of Broca's theory, so far as concerns the seat of the lesions which are attended with aphasia, when it occurs independently of any failure of intelligence. This theory, however, is very far from implying that the whole of the faculty of language is localized in any one part of the hemispheres. It only asserts that a certain spot in the left hemisphere contains machinery, without the use of which a person cannot utter words, nor, indeed, convey his thoughts to the pen in writing.

There is yet another point of view from which it is necessary to consider the pathology of aphasia; namely, as to whether this symptom, when occurring in persons of unsound mind, is necessarily dependent upon a lesion in, or even upon functional disturbance of, Broca's convolution. It seems to me that such a conclusion is very doubtful. Dr. Bastian mentions the

case of a lunatic who did not utter a single word for several years, except during two brief intervals, once when he had an attack of pleurisy that lasted some days, and once when he was suffering from toothache. And Dr. Forbes Winslow relates a similar instance: a person who had been insane for fifty-two years did not speak during thirty years, but recovered speech and answered questions perfectly well during the last fifteen years of his life. In such cases there is surely no reason to suppose that any local lesion was present.

The same observation may be made in reference to some of the cases which Trousseau gives as examples of aphasia; for instance, that of the lady who was wont to welcome her visitors by exclaiming, *pig, animal, stupid fool,* while she, nevertheless, seemed not to understand the meaning of the insulting expressions which she used. And, again, it is applicable to an example of supposed agraphia, occurring in a lunatic at Broadmoor, which has been recorded by Dr. Bastian. The inability to write was of an "amnesic" type, and seems to me to have been merely one of the manifestations of his insanity. ("The Brain as an Organ of Mind," 3d ed., p. 660.)

c. Apoplexy.—Remarkable changes have occurred in the meaning of the term apoplexy. From having originally signified a "stroke," in which the patient falls like an ox struck down by the butcher, it became applied to the effusion of blood upon the brain, which was found to be the most common cause of such attacks, and this use of the word was afterward extended to hemorrhages in other parts, so that pulmonary apoplexy, retinal apoplexy, and apoplexy of the suprarenal capsules were spoken of. There is, however, no advantage in employing apoplexy as a mere pathological synonym for "cerebral hemorrhage," a term which is itself both convenient and devoid of any second meaning. But in clinical medicine it is extremely desirable to have a name for that form of coma which is due to disturbance of the cerebral circulation from some local cause acting within the cranial cavity—as distinguished, I mean, from mere failure of the heart's action on the one hand, and, on the other hand, from narcotic or alcoholic poisoning, uræmia, epilepsy, or external injury; and for this purpose the word apoplexy appears to be altogether suitable and convenient. A long chain of authorities, including some of the most distinguished names in medical literature, might be quoted in favor of a somewhat similar application of it.

Symptoms.—A patient in an apoplectic fit lies "deprived of sense and motion." He cannot be roused, but there may be varying degrees of insensibility. Sometimes the well-known voice of a wife or son may elicit an unintelligible muttering or growling sound in reply, or the application of a spoon to the lips may cause them to be closed and the teeth to be clinched in automatic refusal of food; or one hand may be used to rub or scratch the side of the face or body. More often no such indications of cerebral activity present themselves; the limbs remain in whatever position they may happen to fall into; the respiratory movements and the beatings of the heart alone show that life remains. The pupils are generally torpid, and sometimes altogether insensitive to light; they are sometimes equal and of normal size, sometimes both dilated or both minutely contracted, sometimes unequal, one being dilated and the other contracted. The conjunctivæ can often be touched without any reflex movements being exerted in the eyelids. Pinching or pricking the skin seldom leads to any manifestation of consciousness, but sometimes the hand or the foot is drawn away, and so one may be able to make out that one side of the body is withdrawn from nervous influence more completely than the other side. Or it may be found that when the upper limbs are lifted and allowed to drop upon the bed, one is a dead

weight; or, on the other hand, the arm and the leg on one side may be rigid, those on the opposite side may be relaxed. We shall presently see that such indications of unilateral mischief are of considerable diagnostic importance, and another similar sign, which may sometimes be noticed when it is impossible to make out the presence of hemiplegia, is the conjugate deviation of the eyes described at p. 502.

In the foregoing description of an apoplectic seizure I have not attempted to arrange the various symptoms in a systematic way, but have rather endeavored to follow such an order as would be likely to be adopted by a medical man when called to examine an actual patient. The appearance of the countenance varies greatly in different cases; sometimes it is pale, sometimes congested and of a purple color, with lividity of the lips and tongue; the features are often turgid and swollen; the forehead and cheeks, and, indeed, the whole surface of the body, may be bathed in perspiration, which saturates the linen and stands in large drops upon exposed parts of the skin. The temperature, as measured by the thermometer, has been carefully investigated by Bourneville. He finds that there is at first a slight fall, amounting to about 2° Fahrenheit. In rapidly fatal cases this continues until death, but if life is prolonged the temperature rises, and for several days it may remain at about 100°. When death occurs at an interval of more than ten hours from the commencement of the attack it may be preceded by a rapid elevation of temperature. I believe that this was first noticed by Dr. Hughlings Jackson; it is always to be regarded as a very unfavorable sign. I have notes of one case in which the thermometer registered 107°. When recovery is to take place, the temperature commonly returns to the normal point two or three days after the commencement of the attack.

The pulse may be either increased or diminished in frequency; the prognosis is bad if it is greatly above or greatly below the average, say below 60 or above 120, but the converse is not necessarily true. Dr. Jackson mentions an instance in which the pulse was 72 within five hours of the death of the patient, the rate of the breathing being 14 in the minute. As a rule, the pulse becomes more rapid as the case goes on toward a fatal termination; but the worst sign of all is irregularity of the pulsations of the heart, a succession of rapid beats being followed by a series of beats at long intervals, or the heart, perhaps, stopping altogether for a brief space. Formerly, great stress was laid upon the "full" and "laboring" character of the radial pulse in apoplexy, and it was supposed to be a proof of the necessity for venesection. We now know that the high arterial tension which causes it depends upon the presence of chronic renal disease.

The breathing is sometimes infrequent; there may be as few as three or four respirations in the minute. Toward the last it often happens that the patient ceases to breathe for perhaps a minute, and a purple flush diffuses itself over the countenance, but afterward a deep breath is again drawn, the face resumes its natural color, and the respiration goes on as before. This may be repeated several times, until at length a final pause occurs; the heart may then go on beating for a considerable length of time, but at length its pulsations cease and the patient is dead. In some cases one may observe a remarkable modification of the ratio between the breathing and the pulse, which is known as the Cheyne-Stokes respiration.

But in many cases the way in which apoplexy destroys life is by a gradual increase of obstruction to the breathing, which seems generally to depend upon a concurrence of several distinct causes. One of these is very often the supervention of an œdematous pneumonia, beginning in the bases of the lungs and spreading upward through the back parts of those organs. Another is the accumulation of a thin secretion in the air passages, which

become beaten up into a froth by the inhaled air, and may completely fill the main bronchi, and even the trachea. It is probable that the pouring out of this fluid is often the result, in part, of an irritation set up by the entrance of fluid nourishment; for when a patient is comatose, milk and beef tea and brandy are very apt to run down into the larynx without giving rise to any warning cough or sensation of choking. But I think that there can be no doubt that another important factor in its production is the impediment to the act of respiration which is due to paralysis of the tongue and fauces. Every one who has made post-mortem examinations on the bodies of those who have been suffocated knows how large a quantity of frothy fluid is found in the air passages, even when death has been rapid; but an apoplectic patient may lie for days in a condition in which there is considerable interference with the breathing, as is shown not only by the lividity of his countenance, but also by the fact that the entrance and exit of air are each accompanied by a noise, which is commonly called *stertor*. The causes that lead to the occurrence of this sound were, I think, first clearly pointed out by Dr. Bowles, of Folkestone, in the "*Med.-Chir. Trans.*" for 1860. He admits that when the mouth is partially open the soft palate sometimes drops upon the tongue and vibrates as the air rushes in beneath it. But he ascribes far more importance to a change in the position of the tongue itself; he shows that when the mouth falls open, the point of attachment of the lingual muscles to the symphysis is carried backward, and he thinks that the tongue then comes into contact with the posterior wall of the pharynx. As might be expected, this is especially apt to occur while the patient lies supine. Dr. Bowles finds that turning him over upon his side, with the mouth inclined so that the saliva and other secretions can drain away from it, often causes the entire disappearance of stertor, and may be followed by a decided improvement in some of the other symptoms. When the breathing is noisy, the cheeks are often puffed out at each expiration, in consequence of paralysis of the buccinator muscle; this is of no significance, however, unless it be as suggesting an unfavorable prognosis.

The *duration* of an apoplectic seizure is very variable. In some exceedingly rare instances death takes place a few minutes after the commencement of the cerebral symptoms. Thus Dr. Jackson mentions the case of a woman who was sitting at the tea-table, when she stopped in the middle of a laugh, cried out, "Oh, my head!" fell back in her chair, and died within five minutes, at the most. Abercrombie relates a similar occurrence in a woman who was one evening attending a crowded meeting, and who seemed to be in perfect health. Toward the conclusion she uttered a loud and convulsive scream, and fell down insensible. She was immediately carried out, and was seen by Dr. Macaulay, who happened to be present. He found her pale and altogether unconscious, and within five minutes she was dead. In each of these cases the blood was found effused under the arachnoid. In 1864, there was brought to Guy's Hospital the body of a woman who had died almost instantaneously as she was returning home from the theatre with her children; in that instance, also, a large quantity of blood had been poured out over the sides and base of the brain. But some years ago I made an autopsy in a case in which death had taken place nearly as quickly, and in which there was a large clot within the left hemisphere, bursting into the lateral ventricle. The patient, a man, aged forty-one, was in a surgical ward for some laryngeal affection, and sent down to the dispensary to fetch the medicines; on his way he had a fit, became comatose, and died within ten minutes. One would have expected that hemorrhage into the pons should often cause instantaneous death. But Dr. Jackson remarks that he has never seen such a case, although he has known a woman lie deeply comatose for some hours, in whom this part

of the brain was hollowed out into a mere shell. In the most rapidly fatal case of a clot in the pons that I find recorded at Guy's Hospital, death occurred in forty minutes; two patients lived each for two hours, one nine hours and a half, one thirteen hours, one sixteen hours, and one two days. Meningeal hemorrhage also seems constantly to prove fatal within forty-eight hours. But in the ordinary form of apoplexy, in which blood is effused into the neighborhood of the corpus striatum, life is often maintained for a much longer period. I have notes of twelve cases in which death did not occur until between the second and the seventh days; and of six cases which terminated at the end of ten, twelve, thirteen, sixteen, nineteen, and again nineteen days, respectively. It is worthy of remark that two among these six cases were examples of what I believe to be a very rare occurrence, namely, the formation of a clot within the substance of the brain, which was large enough to destroy life, but which yet failed to reach either the lateral ventricle or the surface; and in a third case the seat of the hemorrhage was altogether exceptional, being the interior of one posterior lobe.

But, as I have already remarked, an apoplectic attack does not necessarily prove fatal. In many cases consciousness is regained. This always takes place more or less gradually. After a few hours, one is, perhaps, able to rouse the patient so that he will give his name, although, when left to himself, he still takes not the slightest notice of anything that goes on in the room. For several days he generally remains drowsy and apathetic, or his ideas may be confused and perplexed; or he may even be delirious, talking incoherently, and throwing himself out of bed. In such cases, however, the prognosis is not good, in spite of the fact that the coma has passed off. Nothnagel says it is exceptional for recovery to occur if insensibility persists for as long as forty-eight hours. In cases which terminate favorably, as the patient regains consciousness, the symptoms of local damage to the brain become manifest, of which the most important is hemiplegia, with or without aphasia.

Onset.—Hitherto, I have, as far as possible, avoided saying anything about the way in which an apoplectic attack begins. Etymologically, its commencement ought to be sudden; the patient falling down in an instant, as if struck upon the head. But we shall presently see that in the cases now under consideration such a seizure comparatively seldom occurs. The older writers laid great stress upon certain symptoms which they believed to be frequent precursors of apoplexy, and which they therefore designated "warnings" or "molimina." But under these names they included a great variety of complaints, without any definite limits as to time: an epistaxis, for instance, happening several months before the cerebral attack; or an ecchymosis of the conjunctivæ, perhaps the result of a violent effort in coughing or sneezing; or even the frequent recurrence of giddiness or headache in a person advanced in years. These last symptoms, indeed, are not unlikely to be due to a morbid condition of the blood vessels of the brain, which may presently lead to their rupture; and the local patches of softening, which we have seen to be more direct effects of such vascular changes, afford a ready explanation of other nervous disorders—such as thickness of speech, diplopia, partial ptosis, sensations of numbness and formication in the hand or in the fingers, partial loss of power in the arm, or dragging of the foot—any one of which, if occurring in an old man or woman, is pretty sure to be taken as threatening the supervention of a seizure, even though it may after a few weeks disappear, leaving the patient apparently as well as ever. One cannot altogether reject this view of the matter; but it is to be remembered that each of the symptoms in question may arise from other causes, and that even if one could be sure that they were due to a diseased state of the

cerebral arteries, it would not necessarily follow that an apoplectic attack was about to occur.

On the other hand, the very same symptoms may be actually results of cerebral hemorrhage, a vessel in the brain having already given way; and, unless the extravasation remains small in amount, the patient is very likely to become comatose a few hours later. In such cases it is obviously incorrect to speak of "warnings." Abercrombie, many years ago, pointed out that cerebral hemorrhage comparatively seldom leads to sudden loss of sense and motion, or (in other words) to the classical form of apoplectic seizure. A very precise clinical history of the cases in which extravasation of blood is most apt to be found is given in his celebrated work. The first symptom, he says, is a pain in the head, which may be so violent as to make the patient scream. The face at the same time becomes pale, the body cold, and the pulse very weak; there is sickness, or even vomiting; and he may fall to the ground faint and exhausted. Often a slight convulsion occurs. After a little while he may be able to walk home; he is quite sensible, but oppressed; he remains cold and feeble, with cadaverous pallor of the countenance. By degrees he recovers his warmth, his face regains its natural appearance, and his pulse improves in strength. Then he becomes flushed, he answers questions slowly and heavily, and gradually he sinks into coma, from which he very seldom recovers. All subsequent writers have recognized the truth of this picture. It has been shown that after fracture of the skull, when the middle meningeal artery is torn through, a very similar series of events take place; and the name of "ingravescent apoplexy" has been given to cases which run such a course. But, until quite recently, most observers have regarded this as only one form of the disease, and have supposed that cerebral hemorrhage very commonly begins with sudden coma. It is, indeed, the fact that insensibility is often present when the patient first comes under observation. Perhaps he is picked up in the street, or found lying on the floor, or in a water closet; or the seizure may have come on during sleep, so as to give no opportunity of tracing its earlier symptoms. But Trousseau says that, although his attention was for fifteen years directed to the question, he did not in that time meet with a single instance in which, when an attack of cerebral hemorrhage occurred in the presence of witnesses, it did not begin more or less gradually. An exception ought, perhaps, to be made for those cases which destroy life in a few minutes. Another exception is made by Trousseau himself, for certain cases which begin with an epileptiform seizure, and this is indorsed by Dr. Hughlings Jackson, who has laid special stress upon the frequency of this mode of commencement. Still, the conception of "ingravescent apoplexy" as being a particular modification of the disease has to be given up. Moreover, it has to be admitted that the symptoms before coma sets in are far more variable than would appear from Abercrombie's description. Pain in the head is often absent. Not unfrequently the only thing which is noticed by the patient is a numb feeling, or a sense of weight in one of the limbs. Thus Trousseau relates the case of a woman who while returning from market noticed that she dragged her right leg, and that her right arm felt heavy, so that she changed into her left hand a folded newspaper which she was carrying, lest it should fall into the mud. She walked up stairs into her room, took off her clothes and got into bed; after which she became hemiplegic and comatose, and remained in a state of stupor for three days. Another patient, who came under the care of the same physician, had noticed while at dinner that one of his hands felt heavy; he was not giddy, but faltered a little in his speech. He tried to rise from his chair, but one of his legs being paralyzed he fell down. His children lifted him up, and with their

assistance he walked into the next room, and sat down. Trousseau arrived in three-quarters of an hour, and found him perfectly conscious; but his left arm and leg were almost powerless. Profound coma set in a few hours afterward, and he died the following morning. Another modification of the symptoms produced by cerebral hemorrhage at its commencement is that in which it is attended with a transitory loss of consciousness, from which the patient quickly recovers, and which is separated by an interval from the final coma. To this Trousseau gives the significant name of "cerebral surprise." A satisfactory explanation of it is not easy. One can, indeed, imagine that the injury to the brain tissue which occurs at the moment when the vessel gives way, causes a kind of shock that is diffused over the nervous centres. Trousseau cites, in illustration, the experiment of trephining the skull of a dog or rabbit and introducing a leaden ball into its interior; symptoms of stupor, he says, are immediately manifested, which quickly pass off, leaving a degree of hemiplegia proportionate to the compression. But Nothnagel says that in hundreds of observations upon animals, in which he injected chromic acid into the brain, or produced artificial extravasation of blood, he never saw a similar effect produced. This writer cites, as an analogous phenomenon, the effect of suddenly cutting through the spinal cord, in instantaneously suspending for a time the reflex excitability of the centres below the line of section.

Locality.—Some writers suppose that the symptoms of an apoplectic attack commonly present modifications which enable the exact seat of the lesion to be determined, if it is other than the usual one, close to one of the corpora striata. But I do not think that this can often be done, at least while the coma lasts, nor in cases that prove directly fatal. I have already, at p. 519, pointed out that some forms of cerebral hemorrhage are more likely than others to destroy life rapidly; this, however, goes but a very little way toward a differential diagnosis. In apoplexy of the pons the pupils are often contracted to pins' points, and the respirations are exceedingly infrequent —perhaps not more than six or four in the minute. But I have notes of one case in which dilatation of the pupils was present, and similar instances are mentioned by Nothnagel; the breathing, too, is sometimes hurried. Nothnagel seems to think that convulsions are more apt to occur than when blood is effused into other parts of the brain, but of this I do not find any evidence. Other symptoms enumerated by him are rigidity of the neck and tonic contractions of the limbs generally. He says that paralysis of the limbs on both sides is observed only in cases which quickly end in death; and I have no doubt that he is right. It can seldom happen that an "alternate hemiplegia" is made out satisfactorily while the patient is insensible. I have already mentioned that this form of apoplexy sometimes leaves behind it a permanent difficulty of articulation.

The fact that lesions affecting the cerebral convolutions so often give rise to convulsive seizures has led some observers to suppose that such symptoms are especially apt to occur in cases of meningeal hemorrhage. But so far as I can judge from the cases which I have collected they are not really more frequent than when blood is effused into the interior of one of the hemispheres. Nor is rigidity of the limbs noted as having been commonly present—a fact which Dr. Goodhart has already pointed out in the "*Guy's Hospital Reports*" for 1876. On the other hand, it would seem that a definite hemiplegia is comparatively seldom observed in this form of apoplexy; the blood makes its way too easily along the subarachnoid space at the base of the brain to compress one hemisphere more than the other. In some cases the coma is preceded by delirium.

Another question is whether it is possible to make out at the bedside

the existence of laceration into the lateral ventricle, when the original lesion is in one of the basal ganglia. The recurrence of coma, after recovery from a first seizure, has been attributed to this cause by Nothnagel and some other writers; but we have seen that it is a common feature in all cases of apoplexy. Others have supposed that the presence of ventricular effusion is indicated by paralysis of all four limbs succeeding to hemiplegia, or by early rigidity of the arm and leg on the side opposite to the lesion. The latter opinion was maintained by Durand-Fardel. But I am not aware that the exceptional cases in which profound coma and death are caused by a clot limited to the substance of one hemisphere have as yet been made the subject of a critical comparison with those in which there is extravasation into the ventricle. In 1874 I made an autopsy in the case of a lad, aged seventeen, who had died fifteen hours after having been found lying on the ground in a state of insensibility. All the cavities of the brain, including the third and fourth ventricles, were found full of clot, which was present in equal quantities on the two sides. I could discover no cause for the hemorrhage, all the central ganglia being quite healthy. There had been repeated epileptiform fits, but I could not learn that the symptoms had differed in any other respect from those of an ordinary apoplectic attack. In 1876 an old woman died in the clinical ward of Guy's Hospital, under my care, of a seizure which was particularly noticed to be unattended with definite paralysis of any of the limbs. The right lateral ventricle was full of clot, which had come from the superficial part of the caudate nucleus, the internal capsule being quite uninjured. Thus the absence of hemiplegia is not peculiar to cases of meningeal hemorrhage.

Diagnosis.—In the diagnosis of apoplexy one has to bear in mind many varied morbid conditions with which it might be confounded; indeed, I do not know of any other disease that can be compared with it in this respect, nor in the importance, as well as in the difficulty, of forming a right judgment. The liability to error arises in two different ways, which it is well to keep distinct; sometimes from the absence of any history as to the origin of the patient's attack, or from the history being vague or untrustworthy; sometimes from the doubtful nature of the symptoms.

Thus a man may be discovered comatose and stertorous in bed or upon the pavement of the street, and there may be no one to say what has happened to him; or it may be impossible to depend upon the statements of those in whose company he is found, or there may be suspicion of foul play. In all such cases three alternatives must be taken into careful consideration before deciding that the case is one of apoplexy; these are (1), that his brain may have been injured by external violence, as by a blow or a fall, with or without fracture of the skull; (2) that he may have taken poison; (3) that the patient may be intoxicated.

1. As regards *injury to the brain*, difficulties arise in several ways. There may be no bruise upon the face or head, and no displacement of any part of the calvaria, and at the post-mortem examination we may for the first time discover that the skull is fractured, or that the prominent parts of the brain on one side are bruised in such a way as to show that external violence had been the cause of death. Even then, however, one is by no means justified in concluding that the case was originally one of accident or of injury inflicted by others. The man may have had a fit, or he may have been drunk, and in falling he may have fractured his skull. Thus, in 1859, a man was admitted into hospital who was driving in a cart, when he fell and was picked up insensible. He died after four days, and the surface of the brain was found to be extensively bruised, but as he was known to be subject to epilepsy, it appeared clear that one of these attacks had been the cause of his falling. Again, we must bear in mind that

meningeal hemorrhage often occurs as the result of disease, it is only when the brain substance itself is ecchymosed that we are justified in attributing to external violence cases in which blood is found effused on the surface of the brain. On the other hand, we have had several instances in which a severe injury of the skull, with laceration of the cerebral convolutions, has been associated with hemorrhage into the central parts, exactly like that which occurs in apoplexy. Some of these have undoubtedly been examples of spontaneous effusion of blood into the brain, the superficial injuries having been caused by the patient's falling upon a curbstone or a hard floor, or the like, but it is certain that a blow or fall upon the head may, in rare cases, cause rupture of an artery in the interior of the organ. Thus, in 1866, a man was admitted into a surgical ward an hour before his death, who had been steering a barge on the Surrey Canal, when a steamer came too close and struck the rudder; the tiller knocked him over, and he was picked up insensible. There was no injury to the cranium, nor to the surface of the brain. The right lateral ventricle was found full of effused blood, the septum was broken down, and there was a moulded clot in the fourth ventricle. In 1868, a patient in the ward for ophthalmic cases, being unable to see, missed his footing and fell, striking his left temple. A large quantity of blood was effused upon the cerebral convolutions, but the right corpus striatum was also extensively ecchymosed, and had in its interior a mass of blood of the size of a hazel nut. In 1870 a man came to the hospital with a cut on his forehead, saying he had slipped upon a flight of stone steps at the Victoria Theatre. He ultimately became hemiplegic and comatose, and died, and the right lateral ventricle was found to contain a large clot, the blood having oozed from a rent in the thalamus. In 1855 a patient was admitted who had been found lying by the side of a crane, the handle of which seemed to have struck him on the head, there being a wound of the scalp. At the autopsy, the left corpus striatum and thalamus were discovered to have been destroyed by a large mass of blood lying between them. Sometimes it is impossible to decide whether a case is one of injury or disease. In 1862 a man was admitted who had fallen several feet, upon his head, from a platform upon which he had been working. His skull was fractured, but it was found that the lateral ventricles were full of blood, and that the right corpus striatum was broken up by a large clot, which projected through an opening in its surface. The opinion of his fellow-workmen was that he had overbalanced himself, as he had shown no sign of having a fit, but Dr. Wilks, who made the autopsy, left the question an open one, in his report.

It will be noticed how closely the appearances found in these five cases resembled those of spontaneous cerebral hemorrhage. Now, in the reports of three of them, it is distinctly stated that the kidneys were granular, or wasted, or cystic; in the other two the state of these organs was not noticed. In other words, although the rupture of the artery was the direct result of a blow or fall in each instance, yet it seems that they may fairly be regarded as examples of ordinary apoplexy, the main cause having probably been indirect, namely, the existence of chronic disease in the coats of the vessel. Most likely, if no accident had happened, these patients would have died of spontaneous hemorrhage into the brain a few weeks or months later. One cannot be surprised that it should be impossible to diagnose such cases in the absence of a history during life, when after an autopsy their right interpretation is so difficult.

2. Narcotic *poisoning* may be mistaken for apoplexy, or *vice versâ*. We have seen that in some instances in which blood is effused into the pons Varolii, the pupils are contracted and the respiration is regular and very infrequent, just as in persons who have taken large doses of opium. I

have no doubt that I once made an error of diagnosis in a case of this kind. I was summoned, one night, to see a lady some six or seven miles away from London, and I found her comatose, with the symptoms I have just mentioned. There was a suspicion that she had poisoned herself, for the day was the anniversary of the death of a son of whom she had been very fond, and a few hours before her illness had commenced she had said to a sister, who had come to visit her from a distance, "Well, have you come to see me die?" or words to that effect. But I was told that she was sitting up in bed talking to this sister, and apparently as well as usual, when she *suddenly* fell back comatose. This fact seemed to me to exclude the possibility of her having taken opium; and I, and her medical attendant also, came to the conclusion that there was hemorrhage into the pons. I had to sleep in the village, the last train having left for London, and in the morning I went to call at the lady's house, expecting to hear that she was dead. To my surprise I found her quite well again. She never confessed that she had any knowledge as to the cause of her illness. The case is interesting, as showing that a patient deeply comatose from opium *may* recover without being walked about, or swallowing strong coffee or stimulants. It so happens that I have, at different times, been called to two persons, both possessed of some medical knowledge, who had taken fatal doses of prussic acid, but who were alive when I saw them. In one case, which has been recorded by Dr. Stevenson ("*Guy's Hosp. Rep.*," 1869), death did not occur for more than an hour and a quarter after the discovery of the fact that the poison had been swallowed. I do not think that the symptoms could have been distinguished from those of apoplexy; but all difficulty was removed by the fact that there was on the table a half-empty, although corked, bottle containing some of the poison. A marked odor of prussic acid was diffused through the room, but the patient's breath seemed not to smell of it.

3. It is often difficult, and even impossible, to distinguish apoplexy from *alcoholic intoxication.* A man who really is dead drunk may be supposed to have cerebral disease, but I think it may be said that the only result that is likely to follow from this mistake is that a patient who had been expected to die, or at least to become hemiplegic, should, in the course of a few hours, completely recover. A man may, indeed, kill himself by drinking an immense quantity of spirits. In 1868 a boy, aged fourteen, was admitted into hospital, under my care, who, with another boy, had stolen two bottles of brandy, and who (according to his companion's statement) had drunk a reputed quart (twenty-six and two-thirds fluidounces) without any admixture with water. This occurred at about 2.15 P. M.; by 4 o'clock he was insensible, and he was at once taken to the hospital. He was comatose, but without stertor; his pupils were at first of natural size, but they afterward became contracted. An œsophageal tube was passed, and the contents of the stomach were pumped out, with a most powerful odor of brandy. He was then put to bed, and an enema of coffee was administered. He remained unconscious for twelve hours, at the end of which time he asked for a glass of water. Dr. Stevenson, commenting upon this case in the "*Guy's Hosp. Reports*" for 1869, says that no doubt the boy owed his recovery to the prompt use of the stomach pump, and in all probability he is right. But it very rarely happens that any one swallows a fatal dose of alcohol, except for a wager or out of bravado, when the medical man who is called to the case is sure to be told of what has occurred, and it appears to me that to mistake intoxication for apoplexy is to commit an error which is scarcely ever likely to be injurious to the patient, even though one should omit to use the stomach pump. On the other hand, to suppose that a man who has apoplexy is merely intoxicated, is a most serious matter. This is the mistake which is

so apt to be made by the police, and into which medical men themselves have too often fallen, from failing to appreciate the real difficulties in the diagnosis between the two conditions. Perhaps the patient is found in a public house, or is known to have been drinking heavily, or smells strongly of spirits. The only safe course for us is to ignore these facts altogether. The odor of brandy, indeed, does not even prove that any stimulants had been taken before the attack commenced; it may have been administered afterward. I would lay it down as a rule, which should never be departed from, that a man who is insensible, and who cannot be roused, is never to be sent away to the police station, nor be left by himself to sleep off the supposed drunkenness. He should always be put to bed and be carefully watched. The presence of convulsions, hemiplegia, or inequality of the pupils may make one certain that the case is due to something beyond alcoholic intoxication, and the same conclusion may be drawn, although with less confidence, from the occurrence of rigidity of the limbs, or of stertor, but the *absence* of all these symptoms is no reason for supposing that there is no cerebral disease. It would, indeed, seem that by testing the urine one can obtain positive evidence that a poisonous dose of alcohol has been swallowed. According to Anstie, this inference may be drawn if one drop of his chromic acid solution (made by dissolving one part of bichromate of potass in 300 parts by weight of strong sulphuric acid) when added to fifteen minims of the urine turns it immediately of a bright, emerald green color. I may note that in the case of the boy who was under my care in 1868, it took two drops of the liquid to produce this effect, one drop giving only a greenish-yellow tint. Of course, this test cannot enable one to decide whether a man may safely be left in the hands of the police, or be sent away from a hospital, since it shows when the reaction is obtained that a large quantity of alcohol, which may be dangerous, has been taken. After all, however, the most important cases are not those in which coma, or even partial unconsciousness, is present, but rather those in which the patient is noisy and excited, throwing his limbs about restlessly in all directions; one is, perhaps, sure that such a man is drunk, but it may be impossible to say whether or not he also has hemorrhage going on within his skull. I have already remarked that the giving way of a cerebral artery seems often to be the direct result of indulgence in drink, and to detect its early symptoms must obviously be exceedingly difficult under such circumstances. Medical men who have had the largest experience are those who most freely acknowledge the impossibility of speaking confidently about cases of this kind, particularly when the patient is advanced in years, or is likely to have chronic disease of his heart, or of his kidneys and cerebral arteries.

4. Even when we have decided that a patient who is comatose is not suffering from an injury to the head by external violence, nor from narcotic poisoning, nor from the effects of alcohol, there are still some other questions to be considered before we can safely conclude that he has apoplexy, as I have defined at p. 516. One possibility is that the case may be one of *pyæmia*. Strange as it may appear, I have seen two cases in each of which a profound and rapidly fatal coma was proved by the autopsy to be due to this cause. One was that of a man, aged twenty-six, who was brought to the hospital insensible, and died in a quarter of an hour. Very little could be learned about him, but it was stated that he had very recently arrived on board ship in the port of London, and that he had been giving evidence in a court of law on the day of his death. The brain appeared healthy, but there was suppurative inflammation of the peritoneal cavity, and also of one knee joint. The other case occurred in a girl eleven years old. She was admitted into the hospital, one morning, at half-past six, and immediately after being put to bed, she gave one gasp and died. Her mother

said that she had been quite well until the previous day, when she complained of pain in the left side on returning home from school in the afternoon. She went to bed and fell asleep, but at 4 A. M. (two hours and a half before her death), she was found to be unable to speak, and to be rolling over and throwing her arms and legs about. When brought to the hospital she was unconscious, and was breathing heavily, with froth upon her lips; the right pupil was dilated, the left contracted. The temperature was 101·2°, the pulse 116, the respirations 32 in the minute. All that I could discover at the post-mortem examination was that the surface of the right lung was ecchymosed, and that there was a partial hepatization of the lower lobe of the left lung, with much lymph effused on the pulmonary pleura over a circular area of the diameter of a Tangerine orange. The interpretation which I should now be disposed to put upon these cases is that there probably was some early change in the brain or in its membranes, of so intense a kind as to kill before it had advanced sufficiently to be recognizable by the naked eye. I shall have to speak of tubercular meningitis as proving fatal at a stage when the microscope is required to demonstrate its presence.

5. Another possibility is that the coma may be due to *uræmia*. The older pathologists were familiar with cases in which, finding no blood effused upon the brain, they were driven to suppose that the cerebral symptoms which had proved fatal were due to the presence of fluid in the ventricles and beneath the arachnoid; and they described the affection under the name of "serous apoplexy." At the present day, however, this name, and the idea on which it was based, have alike fallen into oblivion. Several years ago Wilks suggested that the majority of the cases in question were examples of Bright's disease; and his opinion has been endorsed by all later writers. But I must confess that in reading the detailed clinical reports given by Abercrombie, I fail to see that the cause of death in his cases was uræmia, rather than some one of the less obvious cerebral lesions, which at that time would necessarily have escaped recognition. And, so far as I can make out, there have within the last few years been very few cases at Guy's Hospital in which the symptoms were like those of an apoplectic attack, and in which the only disease that could be discovered at the autopsy was in the kidneys. On the other hand, it has very often happened that albumen has been detected in the urine of a patient who had been brought into the ward in a state of insensibility, and that the seizure has consequently been attributed to uræmia, until a post-mortem examination showed that it was due to cerebral hemorrhage. The common occurrence of albuminuria in cases of this kind might naturally be ascribed to the frequency with which the kindeys are found to be granular. But that particular renal affection comparatively seldom leads to the presence of albumen in the urine in any considerable quantity. Moreover, this symptom is often met with in apoplectic patients whose kidneys are afterward proved to be perfectly healthy. Thus, it is probably to be regarded as a result of congestion of the venous system in general. And certainly no importance should be attached to it, whether in diagnosis or in prognosis. Among the few instances in which an apoplectiform seizure has been found in the post-mortem room at Guy's to have been caused by kidney disease, no cerebral lesion being discoverable, I may cite the following:—

In 1867, a man, aged forty-eight, was brought to the hospital, conscious, but unable to speak; his friends said that he had often suffered from headaches; the day before his admission he complained that his mind was wrong; his manner was strange, but he answered when spoken to. Afterward he became insensible, passing his excretions under him. At one time

he regained his senses sufficiently to say "yes" and "no;" and he could swallow, but he soon relapsed into coma, and died on the following day. His pupils were rather contracted, and his breathing was laborious. The brain was found pale and anæmic; the ventricles were nearly empty. The heart was hypertrophied; the kidneys were extremely wasted, as a result of dilatation of the calyces secondary to stricture of the urethra. In 1868, a man, aged forty-four, who had been intemperate and gouty, and who had been suffering from a severe headache for a fortnight, was brought to the hospital in a drowsy state, from which he could just be roused to speak. He had had a fit, and had bitten his tongue. His body and limbs were in a state of constant jactitation. His pupils were rather contracted. His urine was retained, but his fæces were passed into the bed. Before his death he had two more fits. The only changes found in the brain were that it was tough and wasted, with large ventricles, and with an excess of fluid in them. But the kidneys weighed only two ounces and a half, and were exceedingly granular. At the bedside it would probably have been impossible to say, in reference to each of these cases, that an artery had not given way within the brain. But I find only one or two similar instances in our records. It has sometimes happened that patients already under treatment in the hospital for Bright's disease have died quickly with cerebral symptoms; but under such circumstances hemorrhage is comparatively infrequent. And in the immense majority of cases, the stupor caused by uræmia alternates with convulsions, and passes off again and again without leaving hemiplegia, in such a way as to show that no considerable damage has been done to the actual structure of the brain. I quite admit that when a patient, of whom one knows nothing, is brought to a hospital insensible, uræmia is a possible cause of the coma. But if one should be told that he has not previously had any alarming cerebral symptoms, and if he should steadily get worse and die, I believe that one may almost dismiss from one's mind the idea that nothing but disease of the kidneys will be discoverable at the autopsy.

6. Of cerebral diseases the one which is most likely to be mistaken for apoplexy is a form of *epilepsy* which was described by Andral and other French writers as a separate malady, under the ponderous name of "apoplectiform cerebral congestion," until Trousseau pointed out its real nature and distinguishing characters. In all probability, Abercrombie would have included it under what he termed "simple apoplexy," in which after death no morbid appearance could be discovered in the brain. Indeed, I remember that Sir William Gull in his lectures used to teach that "simple apoplexy" was nothing but epilepsy; but a careful perusal of Abercrombie's cases leaves one doubtful whether all of them can be fairly interpreted in this way, and whether, if precisely similar ones were to occur now, the more accurate pathological methods which we possess would not enable us to place them in different categories. The question, however, is not of any practical importance.

Among the cases related by Trousseau are the following: In 1845, a gentleman, aged forty-two, was found in his bed insensible; his face was turgid and livid, there was stertor, and all power of motion and sensation was lost. How long he had been in this condition his wife could not tell—she had been awakened by a strange, snoring noise. Trousseau had the patient placed in a half-sitting posture, threw cold water in his face, and applied ligatures round the upper part of the thighs to retain the blood in the legs. Scarcely one hour elapsed before he regained his senses and the use of his limbs, and on the following day great lassitude was the only remaining symptom. Some time afterward the same physician was fetched in great haste to a neighbor, aged seventy, who was said to have been

attacked with apoplexy on the Boulevards. He had been unconscious for a quarter of an hour, but was recovering his senses when Trousseau arrived. He did not at first recognize him, and looked vacantly around, throwing his arms and legs about without knowing what he was doing. By degrees, but within a few hours, he recovered entirely, although no active treatment was adopted. In both instances the real nature of the disease was afterward established by the recurrence of the seizures at more or less frequent intervals, attended with all the symptoms of epilepsy. Indeed, it must be observed that in each case the attack began when no skilled observer was present, so that there is no proof that spasmodic movements did not occur. And Trousseau himself goes on to say that in almost every instance of the same kind in which he was consulted, and in which the commencement of the seizures had been seen, "nervous twitches" or convulsions had been present. Thus, after all, the identification of "apoplectiform cerebral congestion" with epilepsy involves little more than the recognition of two facts, that the spasms which usher in an epileptic seizure may be but slightly marked, so as to escape the notice of a non-professional observer, and that when one is called to a patient who is comatose one must make sure that the disease is *not* epilepsy before committing one's self to a diagnosis of apoplexy. And in determining this latter point the first thing to be ascertained is whether the patient has ever before suffered from any seizures which could be regarded as of an epileptic nature. I was one evening called out in great haste to see an old man living about a mile from my house. I found him lying on the sofa in his sitting room, comatose and stertorous, with puffing cheeks and a purple countenance. His pupils were dilated, but I should certainly have thought that he would probably die in a few hours had not his housekeeper, who had found him insensible, told me that he had once or twice before had epileptic attacks. Before long he completely recovered. One of Trousseau's cases is that of a solicitor, aged thirty-five, who was sent to him from the country, with the history that in the course of the previous six months he had had three apoplectic fits. They had lasted, however, an hour at the most, and they had left no paralysis behind them. Trousseau accordingly declared the disease to be epilepsy, and his diagnosis was before long found to be correct. But, of course, there must in every instance be a first attack, and then the criterion fails, and if this seizure should prove fatal, the real nature of the disease must remain a matter of inference. In 1865, a man, aged sixty-seven, a tanner in Bermondsey, was brought into the hospital comatose and died in a few hours. He had followed his occupation until the day of his death, when he was found insensible, having fallen to the ground. He had convulsive movements and appeared to be paralyzed on the right side. It could not be ascertained that he had ever before had a similar attack, but he was said to have suffered much with his head. No recent morbid change could be found in the brain, but there was chronic wasting, the convolutions being shrunken, with much fluid in the sulci, and the ventricles being enlarged and their surface granular; the skull also was dense and had no diploë, and the membranes were thickened and opaque.

A very similar case occurred in 1876. A woman, aged sixty-two, was brought into the clinical ward, having fallen down in the street in a fit. The right pupil was larger than the left. There was right hemiplegia, with complete anæsthesia in the right arm and in the right side of the face. After a time she partially regained her consciousness, but had several convulsive attacks. The coma then again became deeper, and she died at the end of five days. No morbid change could be found in the brain, except wasting, with dilatation of the ventricles; and there was only slight wasting of the kidneys.

There appears to have been no marked engorgement of the cerebral vessels in these instances; but from the way in which death is brought about it is evident that in similar cases such a condition is very likely to be found. And for my own part I should be inclined to regard as examples of epilepsy the cases which some writers (for example, Hammond) describe as instances of fatal congestion of the brain.

There is a further criterion by which we may often distinguish an attack of epilepsy from one of apoplexy, and on which Trousseau has laid special stress. I allude to the mode of onset of the coma. It has already been pointed out that a patient laboring under cerebral hemorrhage rarely falls down suddenly, deprived of sense and motion, and remains persistently comatose. There are, indeed, cases in which apoplexy begins with a fit which is perfectly epileptiform in character; but such cases are exceptional. The practical rule, therefore, is that if we are informed that the commencement of a seizure was gradual, we may safely conclude that the case is not one of epilepsy; but, if it began suddenly, we may be unable to say what is its nature. In such a case our prognosis must be most cautiously guarded; a few hours later the patient may be apparently as well as ever; or he may be dying, or already dead.

7. Several of the *organic diseases* to which the brain is liable may occasionally give rise to a rapidly fatal attack, resembling an apoplectic seizure in its symptoms. This is the case, for instance, with cerebral abscesses and tumors, and even with tubercular meningitis in certain instances. Sometimes, indeed, cerebral hemorrhage does really occur as a complication of the softer forms of tumor, the blood vessels within the growth giving way and pouring a large quantity of blood into its interior. It appears to me not improbable that in many of the cases in which hemorrhage has been found in the cerebellum, or in some other unusual seat—the patient being perhaps young and the arteries healthy—the original disease has been a soft, new growth of small size, which has been overlooked at the autopsy, having been torn up by the extravasated blood.

Diagnosis Between the Anatomical Causes of Apoplexy.—We now come to the differential diagnosis of the several lesions which are each capable of causing apoplexy or hemiplegia, namely, hemorrhage, embolism, thrombosis, and a syphilitic affection of the arteries. I have first to rectify what may well have seemed a strange omission, by giving an account of the clinical history of the last three of these diseases. It has been remarked at p. 491 that under these heads are included a large proportion of those cases which were formerly grouped together under the name of "cerebral softening." And since the older pathologists taught that the principal indication of that supposed morbid state was the occurrence of paralysis without loss of consciousness, it may appear that the diagnosis from cerebral hemorrhage ought to be simple enough. But a more extended experience has shown that the mere arrest of the circulation through one of the large cerebral arteries often causes coma as profound as that which is produced by extravasation of blood. I am, therefore, compelled to break up my description of the symptoms of embolism and the allied lesions into two parts, according as the patient becomes unconscious (*apoplexy*) or retains his senses (*hemiplegia*).

1. For example, among the cases of *cerebral embolism* that have occurred within the last few years at Guy's Hospital, I find the following clinical histories: In 1868, a man, aged forty, was brought into the clinical ward under my care, with paralysis of the left side. He had suddenly fallen down insensible while wheeling a barrow. He presently regained his senses and conversed about his symptoms; and then he again became comatose, and remained so until he died, a few hours afterward. In

1869 a man was taken in for paralysis and partial anæsthesia of the left side; he was reported to have suffered from continuous headache for two or three weeks; a few days before his admission he had suddenly become giddy and lost the use of his left arm and leg; afterward he was unconscious for two or three hours. In the same year a woman, who was in the hospital suffering from dropsy, had a fit which was followed by aphasia and right hemiplegia, and by her death within two days. In 1874 a woman, who was in the ward for uterine cases, under Dr. Braxton Hicks, became comatose with left hemiplegia, and remained insensible until she died, on the following day. In none of these instances was there any hemorrhage into the brain, nor any change beyond softening of the part which should have received its blood supply through the obstructed vessel. Yet, if no autopsies had been made, these very cases might well have been quoted as illustrating the chief varieties of the mode of onset of an apoplectic seizure due to the rupture of a cerebral artery. Nothnagel, indeed, has already stated, in the most explicit terms, that there is no absolute difference between the symptoms of embolism and those of hemorrhage. And there can be no question as to the inadequacy and the trivial character of certain points of distinction which he mentions as having been suggested by earlier writers; namely, that in the former affection the face should be pale rather than red; and that the latter should be accompanied by excessive pulsation of the carotids, stertorous breathing and inequality of the pupils, which symptoms they erroneously supposed to be wanting, unless an artery had given way.

Again, Nothnagel quotes Dr. Eliza Walker as having shown in her inaugural dissertation at Zürich, in 1872, that embolism is very often ushered in by an epileptiform attack; this was the case in twenty-four out of ninety-seven cases which were collected by her. In one point, indeed, the German writer seems to me to be in error; namely, when he says that the affection in question can never have any prodromata. This statement is contradicted by one of the cases already cited, in which the attack was preceded by headache for two or three weeks.

2. Among the cases that have been regarded as examples of spontaneous *thrombosis of a cerebral artery* at Guy's Hospital there has been the following: A man, aged forty-eight, was attacked with pain in the head a month before his death; he fell back insensible, with right hemiplegia; afterward he could walk, but he ultimately again became semi-conscious, and rambling in his talk. Another man, aged thirty-five, was attacked with unconsciousness and paralysis of the left side six days before his death; he then had convulsive fits, in one of which he died. Nothnagel says that it is altogether impossible to distinguish this affection from hemorrhage.

3. As regards the *syphilitic lesion*, the cases related by Heubner in his monograph suffice to show how closely its symptoms may resemble those of rupture of an artery in the brain. In eight cases out of twenty-two collected by him, the symptoms were ushered in directly by an attack of apoplexy. In some rapidly fatal cases the insensibility lasted until death. In others there was a transitory loss of consciousness, accompanied by hemiplegia, and followed, after an interval, by the supervention of coma from which the patient never again awoke; in other words, the clinical features of an "ingravescent" seizure were closely imitated. Case 33, for instance, is that of a man who had suffered from headache and sleeplessness, and who one day, after having appeared to be as well as usual in the morning, was found later on to be comatose and paralyzed on the right side; he could at first be roused a little, so as to make an attempt to open his eyes; but afterward the insensibility became more profound, the face flushed, the pupils immovable, and he died the next evening. Case 47 is that of a journeyman furrier, admitted into the Leipzig Hospital in a state of uncon-

sciousness. He had been attacked on the previous day with paralysis of the arm; this came on gradually, so that he watched its progress upward from the thumb to the shoulder, being then in perfect possession of his senses, and being able to point out with his left hand how high the numbness extended at any particular moment. There are, however, comparatively few syphilitic cases in which the symptoms and course resemble those of cerebral hemorrhage.

Thus—summing up what has been stated in the last four paragraphs—we find that an attack of apoplexy is by no means necessarily due to the extravasation of blood in the brain. Omitting spontaneous thrombosis, on account of its rarity, we have embolism and syphilis as possible causes of affections of the cerebral arteries which may sometimes give rise to precisely similar symptoms. The diagnosis is based entirely upon collateral circumstances. Age is an element of the highest importance. In persons more than fifty years old, one is seldom wrong in attributing an apoplectic seizure to hemorrhage; and the younger the patient the greater the probability that one of the other two causes is in operation. The whole of the body must then be searched carefully; on the one hand, for eruptions on the skin or fauces, enlargement of the testicles or lymphatic glands, and nodes; on the other hand, for indications of cardiac disease, alterations in the sounds of the heart, swelling of the liver and spleen, infarctions in the spleen and kidneys, etc. It is well to remember that cerebral hemorrhage may itself be an indirect result of embolism, in consequence of the formation of an aneurism in the obstructed artery. In most cases of this kind the primary disease is an ulcerative endocarditis, and the emboli possess septic characters.

The diagnosis between the several causes of *hemiplegia* in a patient who is conscious at the time when he comes under our observation must now be considered. The first question that one asks in such a case is whether coma was present when the attack of paralysis occurred. If so, the considerations advanced in the last paragraph are applicable. In general, it may be said that in proportion to the duration and the severity of apoplectic symptoms, is the probability greater that the lesion was the rupture of an artery. On the other hand, if the seizure was unattended with any, even transient, loss of intelligence, the presumption is strongly in favor of its being the result of a mere arrest of circulation in some part of the motor tract. This, however, may itself be dependent either upon embolism of a cerebral artery, or upon syphilitic disease, or upon common chronic arteritis or atheroma. As I have already pointed out, atheroma very often leads to the formation of patches of white softening in the substance of the brain; and that morbid change, if it affects the motor region, is exceedingly likely to cause paralysis. Now, on *a priori* grounds, one would have expected that a point of considerable diagnostic importance would have been the insidious and gradual commencement of a hemiplegia due to such a cause. But experience shows that this is not the case; the attack is generally sudden. It is probable that the nervous elements continue to perform their functions up to a certain point, notwithstanding impairment of their structure; and that several of them at length suddenly and simultaneously give way.

Hence, the chief way of distinguishing from one another the various lesions of the brain that may give rise to hemiplegia lies in a careful study of the other nervous symptoms that may happen to be present. I have already pointed out at p. 512, the diagnostic value of the association of aphasia with the paralysis, as showing that the Sylvian artery is obstructed, whether by embolism, thrombosis or syphilis. Again, it is probable that persistent hemianæsthesia, when associated with hemiplegia, points to hemorrhage, or at least to atheromatous change affecting a number of the

small cerebral arteries, rather than to the other morbid conditions just referred to. For this combination of symptoms shows that the posterior third of the internal capsule is involved, and this lies toward the periphery of the area of distribution of the vessel in question, and therefore might probably be fed from another source, if arrest of the blood supply were the cause of the hemiplegia. At any rate, I find that most of the cases hitherto recorded, in which loss of sensation has been permanent, have occurred in old people, so that rupture of an artery was probably the cause of the symptoms. Among fifteen fatal cases collected by Veyssière in 1874, there is only one in which the patient was young, that of a girl, aged twenty-two years; in several instances, indeed, the lesion is described as having been a patch of *ramollissement*, but no details are given which would have thrown light upon the possible causes of such a morbid change.

On the other hand, I have already pointed out that syphilis is apt to affect several of the great cerebral arteries, simultaneously or in succession. It is, therefore, not surprising that hemiplegia dependent upon this cause is sometimes accompanied by a series of incongruous symptoms, which cannot be referred to a lesion limited to any one spot in the brain. Thus there is sometimes a complete paralysis of one or more of the cranial nerves, or the patient may regain the use of the arm and leg which were first affected; and subsequently he may be attacked with loss of power on the opposite side. This, indeed, is not in itself characteristic of the syphilitic affection; for old people with atheromatous vessels are very liable to have patches of softening develop themselves in the two hemispheres in succession, or to suffer from the effects of small hemorrhages into both corpora striata in turn. But I think that symptoms due to syphilis are more apt to be irregular in their course than those which depend upon other causes; the patient, perhaps, becomes able to stand, or even to walk, and then after a few days relapses; and such changes may occur again and again. Heubner lays great stress on a peculiar somnolent condition, as indicating the presence of syphilis; the patient, he says, is half awake, half asleep, half dreaming. He, perhaps, lies with his eyes shut, taking no notice of anything, and refusing to answer questions; but when one tries to examine him, he resists and turns round in his bed; or he may submit quietly to this, and yet forcibly oppose the removal of the bed clothes. That such symptoms are comparatively seldom seen in cases of embolism or of cerebral hemorrhage is possible, although I rather doubt it. But I certainly think that they are common enough when the disease is a tumor or some other local lesion. And one must always remember that these affections may also give rise to hemiplegia, if they are so seated as to involve the motor tract. There is, in fact, no one of all the morbid changes to which the brain is liable that may not sometimes induce loss of power in the arm and leg on one side. And we shall hereafter see that other possible causes of this symptom are epilepsy, hysteria, chorea, and some other neuroses. Conversely, it occasionally happens that affections of the cerebral arteries may run their course to a fatal issue, without hemiplegia developing itself. Such cases may be attended with headache, loss of memory, drowsiness, delirium, vomiting, thickness of speech, difficulty of swallowing, involuntary evacuations, and a variety of other symptoms which it would be useless for me to attempt to enumerate. A diagnosis is generally impossible.

Treatment.—That the treatment of apoplexy is unsatisfactory is universally admitted. There is no disease in which it is more difficult to tell whether the chance of recovery is affected by therapeutical measures, of whatever kind. Rest is always essential; the patient should, if possible, be left in the room in which the seizure occurred; a small bed may be made up for him, but a mattress placed on the floor does perfectly well for a time. His head and

shoulders must be raised, and he should be turned over on to his side, so as to prevent the tongue from falling backward against the pharynx. In cases in which it is clear that death is actually impending, one ought to abstain from all active treatment. I fail to see what good can possibly be done by the application of a blister to the neck, or of mustard plasters to the calves. A patient whose coma is so deep as to threaten his life cannot be roused by such means; and if he is capable of feeling, the irritation must surely be injurious rather than beneficial. When the case appears not to be altogether hopeless, a few grains of calomel or two drops of croton oil should be placed on the tongue, particularly if there is reason to believe that there is an accumulation of fæces in the bowels. Or an enema of turpentine or castor oil may be given. One must be on the watch for the necessity of passing the catheter if the insensibility should last more than a few hours. Whenever there is serious difficulty of swallowing, the administration of food or drink by the mouth should be altogether abstained from, on account of the danger of its running into the air passages and setting up pneumonia. No harm results from keeping an apoplectic patient for a day or two without any nourishment; but if it is thought desirable, injections of beef tea or milk may be employed. The lips and mouth may always be kept moist with a feather.

Bloodletting, whether by venesection, or cupping or leeches, is now seldom practiced. Sir Thomas Watson, indeed, speaks of patients so insensible as not to feel the puncture made by the lancet, who have yet emerged from their coma while the blood was flowing. But it may be fairly doubted whether in such cases the disease was not rather that "simple apoplexy" which is now known to be really a form of epilepsy, and in which a spontaneous recovery of consciousness often occurs with unexpected rapidity. If the pulse be large and laboring, the face flushed, the carotid arteries full and throbbing, it may be thought right to bleed. But the majority of patients suffering from cerebral hemorrhage are advanced in years and impaired in health, so that one hesitates to employ a practice which can be of service only by its general effect in lowering the pressure within the vessels of the brain. And in a younger person the coma may be due to embolism or to syphilitic arterial disease, in which conditions the maintenance of a vigorous cerebral circulation is essential to recovery.

There is, however, one particular set of cases in which it seems probable that judicious management may sometimes prevent a fatal issue. I refer to those instances in which the symptoms of cerebral hemorrhage are slowly "ingravescent." When one is called to such a patient in the early stage (that of collapse), one should keep him absolutely recumbent, with head and shoulders raised, and he should neither be allowed to speak nor to move. The limbs may be warmed by friction with hot flannels, but the administration of brandy, and even of ammonia, must be rigidly abstained from. As reaction comes on, the question of bloodletting must be most seriously weighed. It is true that a rapid death almost always occurs, and that the autopsy generally confirms the opinion that the case was hopeless from the first. But I think it is possible that a free venesection, just at the time when the vigor of the circulation is being re-established, may now and then, by lowering the pressure in the cerebral vessels, prevent the further effusion of blood. Hæmostatics, such as digitalis, acetate of lead, or gallic acid, are not often prescribed; but Dr. Bristowe assents to their employment, and one is certainly encouraged to try them by the success which often seems to follow their administration in cases of hæmoptysis. Cooling lotions, or a bag of ice, should be applied to the head.

When coma is due to embolism, the only treatment is to keep the patient in a state of the most perfect quietude, so as to reduce to a minimum the func-

tional activity of the nervous centres, deprived of their blood supply. But in cases of syphilitic arterial disease it would appear that the inunction of mercury should be commenced with the first cerebral symptoms.

A person attacked with hemiplegia without unconsciousness should for some days be kept in bed, and as free from all cares as possible. If advanced in years, or suffering under heart disease, he should be supplied with soup, beef tea, milk, and perhaps even a little wine. But the syphilitic patient should at first have rather a scanty diet while the proper treatment is being carried out: say a twelfth of a grain of bichloride of mercury, with ten or twenty grains of iodide of potassium. Heubner relates some remarkable examples of recovery of power in the paralyzed limbs, under the method of inunction. A striking case is that of a student, aged twenty-six, who, after having suffered from headache and giddiness for some weeks, was attacked one night with left hemiplegia during sleep. There were cicatrices on the penis, indurated glands in the neck and at each elbow, and a radiating scar on the velum palati. At the end of a fortnight he was no better; he was feverish, very prostrate, sleepless and delirious; bed sores were forming; and spasmodic movements of the right arm were noticed. Mercurial ointment was therefore rubbed in for seven days, and then iodide of potassium was given, in large doses. Six weeks after the commencement of this treatment he was able to leave his bed, and a fortnight later he could raise his arm to form a right angle with the body. Little by little he regained power in the affected limbs, and several years afterward Heubner found him perfectly well, except that he used a stick in walking.

But in too many cases of hemiplegia—whatever its cause may have been—the limbs remain more or less completely paralyzed, in spite of the most judicious treatment. Recourse is then had to electricity, which undoubtedly is sometimes beneficial. The continuous application of a weak galvanic current to the head, as suggested by Remak, is said by Nothnagel to be occasionally followed by a decided increase of power and diminution of rigidity in the affected muscles. Not more than from four to twelve cells should be brought into operation; the poles should be gradually applied and withdrawn, and should never be suddenly reversed; they should be kept in position for not longer than three minutes at a sitting; whether they are both placed behind the ears, or one upon one mastoid process and the other upon the forehead, seems to be a matter of indifference. Other methods are to galvanize or to faradize the muscles. Dr. Reynolds lays down the rule that little or no good can be thus effected if the muscles contract to the normal extent. But if the contractility is diminished, electricity will often, in the course of a few weeks, restore it; at the same time the muscles will become less wasted, and the previously cold and blue limb will regain its normal temperature. By specially faradizing the extensor muscles of the fingers, one may be able to prevent or diminish contraction of the fingers into the palm. In general, it may be stated that the application of electricity should not be commenced before the lapse of five or six months from the hemiplegic seizure; that it should be at once left off if it causes headache, giddiness, faintness, sickness, or any unpleasant feeling at the epigastrium; and that the current, whether galvanic or faradic, should never be so strong as to cause pain, nor so long continued as to cause fatigue.

The internal administration of strychnia is, I believe, useless in hemiplegia due to the lesions described in the present chapter. Nor can one reasonably expect any benefit to result from the baths of Gastein or Pfeffers.

LOCAL ORGANIC AFFECTIONS OF THE BRAIN.

TUBERCULAR GROWTH — SYPHILITIC GROWTH—OTHER TUMORS—SYMPTOMS: HEADACHE—VOMITING—OPTIC NEURITIS—LOCALIZING SYMPTOMS—FOR THE BASE—THE CEREBELLUM—THE MOTOR TRACT—DIAGNOSIS FROM HYSTERIA, MENINGITIS, ETC.—DIAGNOSIS BETWEEN THE SEVERAL KINDS OF TUMOR—PROGNOSIS—TREATMENT.

RED SOFTENING—CEREBRAL ABSCESS.

In discussing in the last chapter the characters which enable us very often to determine with great accuracy in what part of the brain are seated lesions causing hemiplegia and aphasia, I entered upon one of the most interesting parts of the study of cerebral diseases; but I was not able to complete it, because the affections that depend upon lesions of the arteries scarcely occur, except in certain definite regions of the encephalon. There is, however, another important group of local organic affections which are under no such anatomical limitations as regards their position, and I think it will be in every way convenient that I should take them next; my doing so will save me from having hereafter to retrace my steps over any part of the ground which I have already traversed, and it will place the reader in possession of all the points that must guide him in what is termed the *localization* of diseases of the brain. The group in question would not, indeed, be recognized as such by the science of pathology. That science would rather place as far apart as possible affections so varied as syphilitic gummata, tubercles, tumors, and inflammatory changes leading to no softening or abscess. But here, as in many other parts of my work, I am compelled, by the necessities of practical medicine, to ignore all strict pathological classifications. At the bedside one can often distinguish these several affections only very imperfectly, or only upon indirect or collateral evidence. To describe them separately would be to repeat the same facts over and over again, and to suggest false views with regard to their clinical significance. I shall, therefore, begin with a brief account of such of the lesions as may be included among new growths, taking first *Tubercle*, secondly *Syphilitic Gumma*, and thirdly *Tumor*. Afterward I shall discuss their symptoms in common, the differential diagnosis between them, and their treatment. Lastly, I shall describe *Red Softening* and *Abscess*, by themselves.

1. *Tubercle of the Brain.*—The tubercular process may affect the brain in two different ways. Sometimes a large number of minute tubercles grow into it from the pia mater, or are scattered through its substance along the vessels. This condition is always associated with meningitis, and will be described when I am speaking of that disease. In other cases there is a single caseating mass, or a very limited number of such masses, which may reach a great size. By way of distinction, some writers call these "solitary tubercles." Obermeier has recently proposed to name them "tuberculous tumors," but neither term appears to me a very good one. In size they generally vary from that of a pea to that of a walnut; the biggest I know of is a specimen in the museum of Guy's Hospital, which was received from

Dr. Hughlings Jackson by Dr. Moxon. It is described as being like a large potato. Sometimes they have a flattened base toward the pia mater, but more commonly they are more or less globular in form, and touch the surface only at one spot, or are completely surrounded by brain substance. I do not know that they ever adhere to the dura mater lining the skull, but sometimes they become agglutinated to the tentorium when they occupy the cerebellum. Their substance is of a bright yellow color, firm, or even hard, and generally quite homogeneous, except that at the centre it may be softening down into a yellowish liquid, or may, on the other hand, be partially calcified. But they also have, in most cases, a narrow, pinkish-gray, soft-growing edge, which separates the yellow cheesy material from the brain tissue around. This edge may obviously consist of an aggregation of miliary tubercles, but more often it has no such appearance, and I think that it sometimes consists of elements which are larger than the lymphoid cells that characterize tubercles in other parts. Rindfleisch further insists on the fibrous texture of the marginal zone of the cheesy mass, and declares that many so-called cerebral tubercles are really non-tuberculous, deserving rather to be called fibroid tumors. But a similar transformation of the cellular elements of tubercles is well known to occur even in the lungs, and, whatever its microscopical characters, I do not think there can be a doubt that tubercle of the brain is distinctly a scrofulous affection. Among thirty-two cases that have occurred at Guy's Hospital, I find only two in which it is stated that no similar lesion could be discovered in any other part of the body. It is true that tubercular meningitis (which was found in several instances) would, by some pathologists, be regarded rather as an accidental result of infection from the caseous mass than as of the same nature with it, but in a very large number of instances there was chronic phthisis, or disease of the mesenteric or mediastinal glands. In no less than eighteen of the thirty-two cases the cerebellum was the sole seat of the affection; three times it was the pons Varolii, once the medulla oblongata, six times one of the hemispheres; in the other four cases there were several tubercles in different parts of the encephalon. Twenty-one of the patients were males, the remainder females, a proportion which accords exactly with that given by some previous writers. In three instances the age was under five years, in six between six and ten years, in twelve between eleven and twenty years, in seven between twenty-one and thirty, in three between thirty-one and forty, and in one older still, namely, forty-two.

In one case, that of a child aged four and a half, there is said to have been a fall upon the back of the head five or six weeks before the occurrence of a fit, which was the earliest sign of cerebral mischief; the seat of the tubercle was the pons. Dr. Crichton Browne mentions ("*West Riding Asylum Rep.*," ii) a similar instance; the same part of the head was struck; symptoms speedily showed themselves and proved fatal in two months, and a mass of tubercles of the size of a walnut was found in the cerebellum. I should have thought, however, that in Dr. Browne's case the time was hardly long enough for the development of such a lesion, and a suggestion made by Rilliet and Barthez seems to me very reasonable, namely, that when an injury seems to have been the cause of a tubercle in the brain, it may perhaps, in reality, have merely set up acute changes in the tissue around a tubercle which was there before.

2. *Syphilitic Gumma of the Brain.*—This affection also is attended with the formation of a firm, dry, yellow, caseous material, which, indeed, is not always very easily to be distinguished from a tubercle. One point of difference is the fact, long ago pointed out by Wilks, that it is constantly seated at the surface of the organ, growing into the cerebral substance from the membranes. Heubner states that an analysis of forty-five cases,

recorded by different observers, yielded only three exceptions to this rule, and not one of these was conclusive. Again, when a gumma occupies the convexity of the brain, or the summit of one of the cerebral lobes, the corresponding part of the dura mater becomes thickened and converted into a tough yellow layer and adherent, so as to form with it one inseparable mass. Thus the shape of a gumma is far more irregular than that of a tubercle, which we have seen to be generally more or less globular. Lastly, the syphilitic affection commonly shows a much more considerable proportion of translucent undegenerated tissue; this appears as a moist, grayish-red or gray mass, which is sometimes as soft as jelly; it is especially apt to be abundant at the base, where it may involve several of the cranial nerves and fill up the sella turcica as well as the diamond-shaped space. Microscopically, it consists of granulation tissue, containing some spindle-shaped and stellate elements, and having (according to Heubner) a distinctly alveolar structure in many instances. It is very vascular, and sometimes presents many little extravasations of blood. On the other hand, if anti-syphilitic remedies should have been freely administered during life, the only morbid change found after death may be a patch of superficial softening of the cortex, beneath a local adhesion of the dura mater to the brain; this was the case, for example, in a patient of Dr. Dreschfeld's ("*Lancet*," 1877).

It is a curious circumstance, that of ten cases of gumma of the brain of which I have notes all but one were in persons between the ages of thirty-one and forty, the exception being a woman, aged twenty-six. In five of them there was a definite history of constitutional syphilis, or else there were in the liver nodules, of which the nature was indisputable. Only one of the patients had distinctly stated during life that he had had no venereal disease; in that case the liver contained gummata. As might be expected, males preponderated over females in the proportion of seven to three. In several instances the report of the autopsy is incomplete, an examination of the testes, in particular, having often been omitted. In one case the patient's illness had been attributed during life to a sunstroke. This was, no doubt, a mistake, but many writers think that a blow or fall upon the head may determine the formation of a gumma in the brain, and it has even been imagined that an inherited tendency to diseases of the nervous system may dispose syphilis to affect that organ rather than any other part of the body; or, again, that a mental shock or excessive study may act as an exciting cause of such a lesion.

3. *Tumor of the Brain.*—There is no region of the body in which so many different kinds of new growth are found as in the brain and in its membranes, and yet histological distinctions are nowhere else of as little practical importance, because the clinical history and symptoms of a case are comparatively seldom, and only to a small extent, affected by them. I shall therefore content myself with a brief description of the chief varieties.

The *membranes* may present fibrous and some other simple tumors, such as occur almost anywhere, and malignant tumors of all sorts, primary or secondary. A favorite seat for the malignant growths is between the dura mater and the bone; or, perhaps, it would be more correct to say that they begin with the osseous substance itself, and either push inward the dura mater or penetrate it, protruding upon its inner surface, or even invading the cerebral tissue. They generally are sarcomata. Two or three kinds of simple tumor are peculiar to the coverings of the brain. To one of them Virchow has given the name of psammoma, from its containing calcified particles, like those which constitute the brain sand of the pineal body and choroid plexuses. The lime salts are deposited in curious little globular structures, made up of elements arranged concentrically, so as to resemble

somewhat the well-known "birds'-nest cells." Robin accordingly described this affection as a form of epithelioma; but such a view of its nature is incorrect. Virchow says it generally appears as a semi-globular mass, of firm consistence, pale red or white, and medulla-like. One that I examined had a loose texture and a flocculent surface; it grew in such a position as to indent the brain above the left Sylvian fissure. According to Virchow such growths are less frequently seated in the tentorium or in the falx than in the dura mater lining the skull—particularly at the fore part of the base. Another special form of tumor peculiar to the membranes, is that which was formerly called a cholesteatoma, but which has of late been termed a pearl cancer. It constitutes a dry, hard, rounded mass, occupying the pia mater generally at the base of the brain. Its cut surface has a pearly lustre, and it consists of lobules made up entirely of horny epithelial cells, and supported by a stroma of dense connective tissue. Lastly, Virchow has described, under the name of melanoma, a third kind of meningeal new growth; in a case which came under his observation there were numerous black or brown nodules in the pia mater, the rest of the body being entirely free from them.

Again, of the tumors that have their seat *within the brain substance* there is one kind that is almost special to it, while the rest are like those which are found in other parts of the body. Among the latter sarcomata are the most numerous, especially those of the spindle-cell variety. A fibrous tumor is sometimes met with; and sometimes, though very rarely, a myxoma. Primary carcinoma of the brain is exceedingly infrequent. I find only one case in our records at Guy's Hospital which is said to have been of that nature. Indeed, it is not very common for the organ to be affected, even secondarily, by cancer; I have notes of only four instances of such an occurrence. In the immense majority of cases in which secondary nodules are developed within the cranium the growth is one of the more highly infective forms of *sarcoma*. A peculiarity of secondary tumors in the brain is that they are almost always multiple. On the other hand, it is a rule, to which there are scarcely any exceptions, that a primary new growth is solitary. Among forty-four cases that I have collected from our records, I find only two in which there was more than one tumor in the brain, but in which no obvious source of infection existed elsewhere; and after all, it is quite possible that in those cases there was disease of one of the bones, or of some other structure, which was overlooked. The centre of a cerebral tumor now and then softens down into a *cyst*, and this may become so large that the presence of any solid growth may not be obvious. We have at Guy's Hospital had five cases in which one lobe of the cerebellum or the middle of it has contained a large thin-walled cavity, filled with a fluid that was highly albuminous or deposited spontaneously a fibrinous coagulum. In three instances no adventitious tissue could be discovered, except the vascular membrane forming the cyst itself, but in each of the other two there was a small tumor situated on one side of the fibrous wall. It therefore seems probable that such cavities always arise out of new growths.

Hydatids are of very rare occurrence in the brain. In a girl, aged nine, Dr. Moxon found a large *echinococcus*, occupying the middle and posterior lobes of the right hemisphere; she had also a similar affection of the liver. I believe that no other instance has been met with at Guy's Hospital during the last few years. The *cysticercus cellulosæ* sometimes infects the pia mater or the ventricular space.

The one kind of tumor to which I have already alluded as almost* special to the brain is that which Virchow termed *glioma*, and which he

* A similar affection occurs in the retina during childhood, and, perhaps, also in the suprarenal capsule.

regarded as an overgrowth of the neuroglia, the substance which cements together and supports the proper nervous tissue elements. This substance has a somewhat indefinite structure, consisting of a granular or faintly fibrillated matrix, in which are imbedded small nuclei with ill-defined cell walls. Accordingly, a glioma consists of similar cells or nuclei, more or less thickly set in a similar matrix. Such a growth is, of course, not very unlike a sarcoma; and many pathologists describe it as a mere variety of that form of tumor. Indeed, Virchow himself admitted that it was often impossible to say of a particular specimen whether it should or should not be called a sarcoma, and he expressly recognized the occurrence of intermediate forms, which he termed glio-sarcomata. But although the distinction may not be of great practical importance, it appears to rest upon a sound theoretical basis, inasmuch as the neuroglia is a completely developed or adult structure, whereas the elements of a sarcoma are embryonic, corresponding with those of a connective tissue which is at an early stage of its growth. Prof. Klebs, of Prague, has recently maintained (*"Prag. Vierteljahrschrift,"* 1877) that gliomata also contain newly-formed nervous elements. Certain cells with many-branched processes, which are well known to occur in these tumors, are regarded by him as ganglionic cells; and he believes that they are sometimes developed out of the axis cylinders of nerve fibres. A glioma may be of a pinkish-red color, or it may look so exactly like the normal brain substance that a microscope is required to demonstrate its presence. Its substance is always continuous with that of the surrounding cerebral tissue; for there is never a capsule, as with some sarcomata. Indeed, it often assumes the form of the part in which it grows, so that one might imagine the corpus striatum or the thalamus, or some particular convolution, to have become swollen to three or four times its natural size. But in some cases caseation takes place extensively in these growths; and according to Klebs, they are also liable to sclerosis, exactly like that which occurs in the normal brain substance.

A soft glioma often contains a great number of thin-walled vessels, which may rupture and pour out blood into its tissue, tearing it up so that one can hardly discover any trace of the growth; as Virchow long ago pointed out, the disease may then be mistaken for a simple cerebral hemorrhage (*cf.* p. 529).

Perhaps the most puzzling cases of all are those in which repeated extravasations occur, and in which the coagula become converted into tough, opaque, caseating masses, of various colors.

Not only gliomata, but all the less circumscribed forms of cerebral tumor, are apt to set up in the adjacent brain tissue morbid changes that can only be regarded as inflammatory, and as due to *irritation* caused by their presence. Such an affection sometimes assumes the form of "red softening," of which I shall give an example further on. More often it is what Rokitansky first described as "yellow softening,"—a state in which the medullary substance has a faint yellow tinge and looks glistening, like blanc-mange, but nevertheless retains its form when sliced or cut, and is not, in fact, softer than the rest of the brain. This affection appears to be an œdema; the microscope throws no light upon its nature. Another common result of the presence of a new growth in the brain is the accumulation of an increased amount of fluid in the ventricles, which may become greatly enlarged. It might sometimes be attributed directly to compression of the veins of Galen or of the choroid veins; as when the tumor is seated in the cerebellum or in one of the posterior lobes of the cerebrum. But Virchow has pointed out that hydrocephalus often occurs in cases in which such an explanation is obviously inapplicable; and this statement is confirmed by several cases of which I find notes in our records.

Statistics of Cerebral Tumors.—If I may judge from the cases that have occurred at Guy's Hospital, there is a remarkable difference in the liability of different parts of the brain to be the seat of tumors at different ages. We have had twenty-two cases in which there was a primary new growth in one of the hemispheres, sixteen on the right side and six on the left. In only five of these was the patient less than thirty years old; the rest were pretty evenly distributed between the ages of thirty and sixty. On the other hand, out of fourteen cases in which a tumor was seated at the base of the brain ten occurred in persons under the age of thirty. The five instances of cysts in the cerebellum were all in patients between twenty-one and twenty-six years old. There were only two cases in which solid tumors were found in children under the age of ten; in each instance the seat of the affection was the cerebellum.

Tumor of the brain is more frequent in males than in females. I find that among forty-two cases which have occurred at Guy's Hospital, and in which the sex is recorded, the proportion is as 27 : 15. This corresponds very closely with the ratio of 10 : 6 given by Obermeier. But it is interesting that in the cases in which one of the hemispheres was the seat of the affection the preponderance of males is much higher, namely, as 16 : 6. For men are much more liable than women to blows and falls; and Virchow has expressed the opinion that new growths affecting the upper part of the brain are often caused by injuries to the head. He has further stated that tumors in this position are generally gliomata, whereas at the base sarcomata and carcinomata are more often found. In the three cases that have last occurred in my knowledge, the patient's illness was attributed during his lifetime to a severe fall or blow upon the head, received some time previously.

In proceeding to discuss the *symptoms* of these various affections, I have, in the first place, to remark that in making post-mortem examinations one occasionally finds a tubercle or a tumor in cases in which there had during life been no suspicion of the presence of any cerebral lesion. Thus we have in the museum of Guy's Hospital a specimen of a very hard growth, nearly as large as a pigeon's egg, attached by a pedicle to the ridge of the petrous bone; it was discovered accidentally in the body of an aged pauper woman who was said to have had no cerebral symptoms. Another preparation is one of a large carcinomatous mass in the cerebellum, taken by Dr. Moxon from a patient who died of cancer of the breast and of the liver. But it may fairly be said that, if she had lived a little longer, the tumor might have manifested signs of its presence; and the same remark applies to the very few instances in which one or more small tubercles have been unexpectedly found in the brain in persons who have fallen victims to phthisis or to some other scrofulous disease. All that is really proved by such cases is that lesions of various kinds may be latent during the early periods of their development. But of this fact there is abundant evidence of another kind. Thus when tubercular meningitis has been the cause of death, and when the patient had appeared to be perfectly well up to the time of the commencement of that disease, one not uncommonly finds one or more yellow tubercles in the brain substance which must obviously have been of older date. Again, it sometimes happens that a person dies of what appears to be an acute illness, and that the only lesion found at the autopsy is a tumor, with softening of the surrounding cerebral tissue. Some years ago, a man was admitted into hospital insensible, livid, and in a high state of fever, so that the diagnosis was typhus; he lived only three days from the time when he was first taken ill, but at the autopsy a small spindle-cell growth, of the size of a bean, was found projecting into the fourth ventricle from the

side of the iter. But, with the exception of one or two instances in which psammomata or other meningeal tumors have been quite unexpectedly discovered, I do not find in our records a single instance in which a tubercle or a tumor has been met with in the brain of a patient killed by accident, or who died from some indifferent disease, such as heart disease, or ileus.

The symptoms themselves may be divided into two classes. Some of them are common to the large majority of cases; others are comparatively seldom observed, their presence being dependent upon the seat of the lesion. It will be convenient that I should first describe the former kind; and I think that they may be arranged under three heads: (1) cerebral symptoms proper, such as headache, giddiness, epileptiform seizures, loss of memory, mania, stupor; (2) disturbances of distant parts, including vomiting, constipation, etc.; (3) certain changes in the optic disc, which are revealed by the ophthalmoscope.

1. *Headache* is very rarely absent, and is generally the earliest indication that anything is wrong with the patient. Its seat sometimes, but not always, answers to that of the lesion. The superficial nerves of the corresponding part of the scalp may be tender to pressure or percussion; or (as Romberg first noticed) the act of holding the breath or of coughing may increase the pain. The museum of Guy's Hospital contains a large tumor, three inches in diameter, which I found many years ago in the left hemisphere of a girl, a patient of Dr. Wilks, who had suffered severely from pain in the head, and who had declared that when she turned her head to one side she felt something move in its interior. Another preparation consists of a small growth from the dura mater, taken by Mr. Day, of St. Neots, from an old woman who died of bronchitis. She had often expressed a wish that her head should be opened, because for years she had experienced anomalous pains in it, and a sense of coldness in one spot, not larger than a shilling; this corresponded very nearly with the seat of the tumor that was found after her death. The pain caused by a new growth may, in fact, be of every degree of intensity, from a dull aching to the most unbearable agony, such as is said to render a patient delirious. It is sometimes constant, but it generally undergoes exacerbation from time to time. In some cases, indeed, it is altogether intermittent or paroxysmal, so that it may closely resemble an ordinary migraine. Thus Abercrombie relates the case of a boy, aged six, who began to suffer from fits of severe sick-headache, recurring at first about once a fortnight, and leaving him in good health in the intervals. After five or six months the attacks assumed a different and more persistent character; and two months later he died, when a tuberculous mass was found in the cerebellum. A similar instance has been recorded by Lebert. These cases seem to me to have an important bearing upon the general theory of the production of "cerebral symptoms" by local affections of the brain. I shall hereafter have to describe a group of functional disorders of the nervous centres, including migraine, vertigo, epilepsy, and some others—under the name of "nerve storms" or "explosive neuroses." And I shall endeavor to show that each of these is liable to be set up by a variety of exciting causes. Now, my hypothesis is that a tumor or a tubercle causes a transitory vertigo, or an epileptiform fit, or an attack of sick-headache in exactly the same way as any other disturbing agent. I conceive that the nerve storm so produced has precisely the same seat as when it is merely the result of over-fatigue, or irritation of the generative organs, or disorder of the stomach. And I think it is probable that frontal headache, even when unattended with the other characteristic symptoms of an attack of migraine, is yet very often of that nature. If this be granted, it ought to follow that pain in the forehead

should point less directly to the anterior part of the brain as the seat of a tumor than occipital pain to the cerebellum or posterior lobes. I do not know whether growths situated in any particular region are more apt than others to be accompanied by vertigo or by epileptiform convulsions, as distinguished from those seizures which are limited to the muscles of certain parts, and to which I shall presently specially refer. But Dr. Reynolds was led, by the examination of a large number of cases, to the conclusion that convulsions in general were less common when the disease affected the anterior lobes than when it occupied the posterior lobes or the cerebellum. Irritability of temper, depression of spirits, loss of memory, a general impairment of intelligence may be observed even when a tumor lies in the membranes at the base of the brain. Thus we have in Guy's museum a specimen of a cholesteatoma, of the size of a pigeon's egg, which was situated so as to compress the under surface of the pons and of the cerebellum, but which had also insinuated itself into the Sylvian fissure to some extent; the patient was at one time in the hospital with paralytic symptoms, but he became maniacal, had delusions, and was unmanageable, so that he was removed to Colney Hatch Asylum, where he died. On the other hand, the mental faculties are not infrequently retained nearly to the last, when one of the hemispheres is the seat of the disease. Friedreich and Obermeier speak of excessive sleepiness as a principal symptom in two cases, which severally came under their observation. The most usual termination of all the affections described in the present chapter is by stupor, which gradually passes into coma. Sometimes the patient lies for weeks without taking the slightest notice, passing all his evacuations under him, and showing no sign of intelligence, except that he slowly swallows the food that is placed into his mouth. Some years ago Sir William Gull had under his care a boy who manifested a remarkable retardation of intelligence. If a question was put to him he seemed not to heed it; but after many seconds, when the questioner had passed on to talk to some one else, he would deliver, word by word, a reply which showed that he perfectly understood what had been said. In other cases the mental condition undergoes changes which one feels disposed to refer to varying degrees of pressure by ventricular effusion. After lying in a stupor for two or three weeks, the patient may recover his senses, and remain conscious for several days, relapsing then into his former state.

2. *Vomiting* is a frequent symptom, and also constipation. As a rule, there is loss of flesh, and extreme emaciation sometimes occurs. But a boy, of fourteen, who was in Guy's Hospital in 1867, became remarkably fat during his illness, and remained so until he died; and two or three years previously, the body of a young woman in a similar condition was brought down into the post-mortem room. In each case there was a tumor at the base, growing upward into the third ventricle. The temperature of the body is often one or two degrees below normal; but before death fever may develop itself. Obermeier speaks of a rapidly advancing case in which every exacerbation of the headache was associated with a marked fall in the rate of the pulse. The mode of death is often by cessation of breathing, the heart continuing to beat for some little time; in one instance, of which I have notes, it went on for thirty-five minutes, while artificial respiration was vigorously kept up. In such cases it is wonderful how quietly life departs, without a gasp or a moan, or the movement of a limb. Some years ago I was called to see a female patient, whose symptoms pointed to the presence of a cerebral tumor, with Dr. Blades, of Kennington. On my arrival at the house, he came out of her room to speak to me, leaving her husband and two women with her. When we went to her bedside a few

minutes later we found her dead, except that a slight flickering of the pulse was still perceptible. Not the slightest change had been observed.

3. *Changes in the Optic Disc and Retina.*—In 1860 von Graefe communicated to the German "*Archiv für Ophthalmologie*" a short paper in which he pointed out that inflammation of the optic nerves within the eyes sometimes occurred as a complication of cerebral diseases. It was already known that blindness was a frequent effect of tumors of the brain, even when their seat was not such as to involve any of those parts which were supposed to be functionally related to the sense of vision, but the belief had been that paralysis of the nerves of sight was the cause of the amaurosis, and that if any anatomical change was to be found it would be a simple atrophy from disease. Thus, von Graefe confesses the surprise that he felt when he first discovered (about the year 1857) that the optic discs were swollen and reddened in a case of this kind. His observations were soon confirmed by others, and after a little while the remarkable fact was elicited that precisely similar appearances could often be detected when the patient was not conscious of any visual defect. Thus the ophthalmoscope has acquired a very great value in the diagnosis of diseases of the brain.

It was suggested by von Graefe himself in his earliest communication that there were two separate ways in which changes in the discs could arise, and that these were characterized by different appearances. On the one hand, an increase of intracranial pressure, acting upon the cancerous sinuses, might mechanically obstruct the return of blood through the retinal veins; on the other hand, an inflammatory process at the base of the brain might be propagated, as a "neuritis descendens" along the optic nerves to their terminations within the eyeball. Subsequent writers have to some extent modified the interpretation of these two conditions, and it has been shown that they very often coexist. But, with the distinguished exception of Dr. Hughlings Jackson, I believe that almost all competent observers continue to recognize the theoretical difference between them. The one is known to the Germans as the "Stauungs-papille;" following Dr. Allbutt, English writers term it the "Choked Disc," or (with less propriety) "Ischæmia of the Disc." The other is called "Optic Neuritis" or "Neuro-retinitis descendens." There is also a third change, "Atrophy of the Disc," which may either arise independently or be consecutive to one of the other two.

The Choked Disc.—I do not find any more precise description of this affection than that which von Graefe gave in his account of the first case that came under his observation. "The papilla," he says, "was greatly and irregularly swollen, rising steeply on one side and falling gradually on the opposite side to the level of the retina. . . . Instead of being transparent, its tissue looked gray and opaque, with an extremely deep reddish tint, and the adjacent part of the retina had the same appearance, so that the choroidal margin was completely hidden. The opacity was diffused, except that with the direct method of examination one could perceive a striated appearance following the course of the fibres of the optic nerve. The retinal veins were dilated and exceedingly tortuous, and obscured here and there by dipping into the opaque tissue; the arteries were comparatively small. The turbidity of the retina diminished gradually from the disc outward over a zone of rather more than 2‴ in breadth, so that it occupied an area 5‴ in diameter (including the disc itself)." In a second case "the disc was still more red, and there were ecchymoses in the adjacent part of the retina."

The prominence formed by the optic disc in a case of this kind appears to be easily recognized after death when the eye is removed and laid open; an admirable illustration of it is given by Dr. Allbutt. In one instance in

which an intercurrent attack of smallpox destroyed life at an early stage of the ocular affection, Cornil found that the microscopical appearances consisted in an infiltration of the connective tissue with serum, and in a few hemorrhages into the adjacent part of the retina ("*Arch. Gén.*," 1868, ii, p. 679). The absence of grave morbid changes is proved by a case of Mr. Lawson's, in which the presence of a hydatid cyst within the orbit caused an extreme state of "choked disc;" four days after puncture of the tumor the engorgement was found to have almost entirely disappeared. A drawing in "*Pagenstecher's Atlas*" (Pl. xxxi, fig. 7) shows the nerve fibres bulging outward so as to separate from the choroid the peripheral layers of the retina, throwing them forward and outward. In some more advanced cases of von Graefe's, examined by Schweigger and Virchow, the connective tissue elements, the vessels, and the nerve fibres were all found swollen, and the latter were beginning to degenerate, but in the outer coat of the vessels there was an overgrowth of nuclei.

The choked disc was by von Graefe attributed to compression of the cavernous sinus, aided by what he termed the constricting action of the sclerotic ring. He argued that if from any cause the flow of blood along the retinal veins was obstructed, the unyielding structure would give rise to a sort of *strangulation* of them at the point where they pass through it, and so he explained the fact that the congestion was limited to the intra-ocular termination of the optic nerve; but (as Dr. Hermann Schmidt has urged in vol. xv of the "*Arch. f. Ophth.*") the ophthalmic vein communicates so freely with the facial vein that it is difficult to see how pressure upon the cavernous sinus could appreciably interfere with the escape of blood from the eyeball, nor does it appear probable that the wall of the sinus would yield to any moderate force. These considerations are confirmed by a case under my observation, in which the sinus on one side was completely obstructed by softening thrombus; the corresponding optic disc had been noticed, during life, to be perfectly normal. In all probability I think Schmidt's explanation is the correct one. He finds experimentally that an injection of Prussian blue into the arachnoid cavity (subdural space of the brain) makes its way through the optic foramen into the space between the two sheaths of the optic nerve, and fills a fine network of lymphatic channels within the lamina cribrosa (formed by the sclerotic ring) between the very fibres of the nerve themselves. He, therefore, suggests that some of the fluid which is always present in small quantity in the arachnoid cavity is driven into the optic nerve in a similar manner whenever the intracranial pressure is from any cause increased. And as a matter of fact, I can from my own observations confirm the statement that a watery liquid is found distending the sheath and giving it a bulbous appearance, and that this liquid readily escapes as soon as a puncture is made. One difficulty is that, on Schmidt's theory, I do not quite see why cerebral hemorrhage should not cause choked discs. Is it because the great force suddenly exerted flattens the sheath of the nerve, and closes the channel through it? A notion of Benedikt's, that the changes in the fundus of the eye are in some way explained by referring them to sympathetic vaso-motor disturbance, seems at present too vague to be met by a serious argument.

Neuro-Retinitis.—The distinctive features of this affection, as compared with choking of the disc, are that the swelling and redness are less marked, but that it looks more opaque, and that the morbid process extends further into the retina itself, and (according to von Graefe) involves its middle and outer layers, as well as its inner layer. Dr. Allbutt endeavors to describe the appearance of the disc by saying that there is not "a circumscribed intense redness, or brownish-gray, but rather a wash of reddish lilac or a gray tint."

And he adds that one does not perceive "a multitude of minute branches and capillaries," such as give a "mossy" look to the choked disc, but that the vessels which become distended and tortuous are rather the main trunks. Moreover, these are often completely concealed in part of their course by the opaque tissue beneath which they dip.

One of the chief microscopical appearances seems to be that the optic nerve fibres within the disc are enlarged and beaded, presenting a series of fusiform swellings. The connective-tissue bundles are said by Virchow to present no excess of cells or nuclei, but the coats of the vessels are thickened. The trunk of the nerve in its whole length shows an accumulation of cells and nuclei within its sheath, and also between the fasciculi of which it is made up.

I have already remarked that von Graefe ascribed neuro-retinitis to the direct extension of inflammation downward; as, for instance, from a basal meningitis. We shall find that this explanation is not always applicable. The probability is that the affection is then secondary to a choked disc, being developed in exactly the same way as those chronic affections which are well known to occur in the lungs and liver and kidneys when there is obstructive heart disease. And it is not surprising that (as Mr. Hulke has remarked) mixed forms of ophthalmoscopic changes are more commonly seen than typical specimens of a "Stauungs-papille" or of a descending inflammation of the optic nerve.

Atrophy of the Disc.—Neither choking of the disc nor neuro-retinitis is a permanent affection. They are not even stationary, for each of them, if the patient lives long enough, must subside or quickly end in a destructive process. The latter is by far the most common termination. The disc becomes less and less swollen, and at length is quite flat, or even sinks below the level of the surrounding retina. Its red and gray tints grow into a dirty white color. The tortuous veins diminish in size; spots of hemorrhage fade and are absorbed. The outline of the disc remains for some time blurred and irregular, its margins are ragged, and streaks of exudation are to be seen in the course of the retinal vessels. I have taken this description of "consecutive atrophy" from Dr. Allbutt, but it accords with those given by all other writers. He goes on, however, to say that even these appearances are transitory. Little by little the disc clears up; its edge becomes sharply defined; its surface dead white and glistening. Its condition is then undistinguishable from one which has been preceded by no swelling or inflammation of the disc—the "simple white atrophy" of writers. Pagenstecher, indeed, states that in microscopical sections of the dead tissues the origin of the affection may be recognized by the permanent displacement outward of the two external layers of the retina from their normal commencement close to the edge of the choroidal ring. As to the histological changes in the disc itself, he shows that all the nervous elements may disappear, and be replaced by a coarse connective tissue.

It is surprising how little impairment of vision attends these changes in the optic discs in many cases. Pain and over-sensitiveness to light are constantly absent. If there is any affection of sight, it is that the patient either cannot see at all, or sees objects more or less indistinctly, as if through a mist. Dr. Allbutt speaks of several patients with choked discs who could read a badly printed news sheet with ease. One should, therefore, make it a rule to examine with the ophthalmoscope all cases in which symptoms of cerebral disorder are present; unless, indeed, an exception may be made for some of the simple uncomplicated neuroses. On the other hand, if a patient is found to present morbid appearances in the fundus of the eye, one should not be contented with his statement that his sight is perfect. The extent of the visual field in all directions should be accurately

explored; and perhaps it may turn out that although vision is good at the centre, it is yet very defective at the periphery or toward one side. When blindness sets in, it sometimes seems to come on almost suddenly, although the changes in the discs were no doubt developed very gradually. This occurred, for instance, in a patient under my care in 1878; about three weeks before her admission she went to sleep one afternoon, and on waking up, found that she was totally blind. If perception of light is completely lost, I believe that the irides are always widely dilated and motionless. But in those cases in which the impairment of sight is partial, the pupils often appear to be of normal size, and their movements are sometimes not even sluggish. The suggestion of M. Jaccoud, that the excitation of reflex contraction of the iris by light is a proof that blindness is due not to any affection of the optic nerve within the eye itself, but to a lesion situated above the corpora geniculata, seems to be altogether fallacious.

When a choked disc, or one affected with neuritis, passes into a state of atrophy, the patient's sight often becomes progressively worse; but sometimes, on the other hand, it shows a marked improvement. Thus a patient of Dr. Goodhart's, to whose case I shall presently refer, and who, at one time, could see absolutely nothing with her right eye, was ultimately able to read Snellen's 2½, though with difficulty, at a distance of about a foot. When the power of vision has been but slightly impaired during the early stage of the disease, it is sometimes perfectly regained; but even in such cases, if actual neuritis has been present, the disc seems invariably to become more or less atrophied; it never resumes its normal appearance.

Localization of Definite Cerebral Lesions.—Other symptoms not only indicate in general the *presence* of some local lesion—whether a tubercle, gumma, or tumor—but also point more or less definitely toward its exact *seat;* they constitute what Dr. Jackson terms "localizing symptoms." Now it is important that, as physiologists, we should be able to interpret any phenomena of perverted action of the nervous centres which our patients may present, and that, as physicians, we should pay attention to every feature of a case which may enable us to understand it better. But I do not see why we should impose upon ourselves a task which we are in no way called upon to perform, that of attempting to determine during life the position of all, or even most, of the tumors of the brain that come under our observation in clinical practice, still less why we should feel disappointment at the failure that seems to be the inevitable result. We might, indeed, have anticipated that the seat of an intracranial lesion would greatly affect the prognosis; but this is not the case in any marked degree. For all practical purposes, I believe it may be said that the study of localizing symptoms is at present really useful only in so far as they either enable one to clinch the diagnosis of the *existence* of a definite intracranial lesion, or throw some light upon its *nature.*

In the interpretation of some symptoms there is a preliminary difficulty, to which I must briefly allude. When at an autopsy one finds a tumor, or a tubercle, in some particular region of the brain, one cannot always tell whether the disturbances of function to which it gave rise were the results of *irritation* of the structures around it, or of *destruction* of those among which it grew. In the latter case the symptoms themselves would be those of the uncontrolled or unbalanced action of the corresponding parts on the opposite side of the brain, or of some other parts physiologically antagonistic to those containing the lesion. It is precisely this difficulty which makes it impossible to attach an exact meaning to inequality of the pupils; and several other instances will present themselves further on. Dr. Jackson, indeed, has endeavored to distinguish certain affections as "de-

stroying," while he speaks of others as "discharging" lesions. And it is remarkable how a great size may be reached by a mass in the brain, without its depriving the structures in which it is imbedded of their function ; while again a tumor sometimes compresses adjacent parts, so as to annihilate their activity. I will not attempt to lay down a positive rule, but I think it may be said that, in all the diseases now under consideration, the presumption is always in favor of irritation, rather than of destruction, when either explanation is admissible.

The Base.—Beginning at the base of the brain, and passing from before backward, one meets with the simplest of all localizing symptoms, namely, those which depend on interference with the various cranial nerves.

It is easy to see that complete *anosmia* points to the presence of disease near the ethmoid bone, or involving the inner and lower parts of the two anterior lobes; while loss of smell on one side may be due to a lesion implicating one olfactory bulb, or the root of the nerve extending outward to the Sylvian fissure (see p. 370).

A growth pressing upon the optic chiasma produces various effects, according to its exact situation. Thus there may be more or less complete loss of sight in one eye, or even total blindness; but as changes in the discs are almost certain to be present, it would be scarcely possible to draw any inference from this symptom. What is really significant is the limitation of a visual defect to half the field in each eye, or (as it is termed) *hemiopia*. Of this there are three different forms, but the interpretation of them is still somewhat doubtful, because opposite views are now held as regards the structure of the chiasma. On the theory of semi-decussation, which to me appears the more probable, they may be described as follows:—

(1) Compression of the centre of the chiasma, cutting off the decussating inner fibres of each optic nerve, will cause loss of vision over the temporal field in each eye (double temporal hemiopia); (2) compression of one optic tract will deprive the patient of the temporal field in the corresponding eye, and of the nasal field in the opposite eye (homologous lateral hemiopia); (3) loss of vision in the nasal fields of the two eyes (double nasal hemiopia) can only be due to a double lesion, symmetrically placed at the lateral angles of the chiasma, so as to interfere with the non-decussating outer fibres on each side. Examples of all these forms of hemiopia are known to occur. Homologous lateral hemiopia is very common in attacks of migraine; of double temporal hemiopia two cases have been recorded (one by Sämisch, the other by E. Müller), in each of which a large tumor lay in the middle line, involving the centre of the chiasma; of "double nasal hemiopia," one by Knapp, in which the pressure at the necessary spots was effected by dilated and atheromatous branches of the circle of Willis.*

Intracranial growths sometimes produce *deafness* when they occupy, or extend into, the space between the internal auditory meatus and the side of the pons. Dr. Hughlings Jackson has even met with one case in which there was loss of hearing from this cause without any facial paralysis. In another instance, a complete bilateral destruction of the auditory sense was imperfectly explained by the autopsy, for the only growth at the base was

* I may note that on the theory of complete decussation "double temporal hemiopia" would be caused only by pressure on the *front* of the chiasma, while pressure on the *back* of the chiasma by a single growth would account for "double nasal hemiopia." So far, that theory might hold, although both these kinds of hemiopia are very rare, but it admits of no explanation of the occurrence of the far more common "homologous lateral hemiopia," excepting the untenable one of the presence of a lesion placed *on one side* of the chiasma, in such a situation as to compress a part of the optic nerve, and also a part of the optic tract on that side.

one of the size of a hazel nut on the *left* side. It might be suggested that this symptom ought sometimes to be referable to an affection of the brain itself; but Dr. Jackson's experience appears to negative such an opinion.

With regard to affections of the other cranial nerves I have little to add to the remarks which I made when describing their symptoms (see pp. 357–371). Only I would observe that an anatomist can sometimes infer the seat of a growth with very great accuracy from the implication of some nerves while others escape. Thus I remember one instance in which the fact that all the muscles of the eyeball were paralyzed, while no part of the fifth nerve was interfered with except its ophthalmic branch, seemed to prove that the disease occupied the sphenoidal fissure. On the other hand, it is perhaps sometimes possible to draw the conclusion that no single lesion will explain every feature of the case, a conclusion which (as we shall presently see) may be of considerable diagnostic importance.

The Cerebellum.—The localization of a tumor or tubercle in the cerebellum is often attempted, and is not infrequently successful. But one must not forget that this part of the encephalon is a very common seat of such growths. I confess that I feel doubts as to whether the result of the diagnosis ought not sometimes to be attributed to good luck rather than to skill. Beside pain in the back of the head and neck, vomiting, convulsions, blindness, and vertigo—none of which symptoms are special to lesions of the cerebellum—there are some others which are believed to be more characteristic, and chief among these is a reeling, staggering gait, like that of a drunken person, but with a tendency to fall in some particular direction. It was long ago shown by the experiments of Flourens on pigeons that excision of the cerebellum rendered the bird unable to walk or to fly, or to perform regular determinate movements; and he suggested that the function of the organ was one of co-ordination. Subsequent investigations have proved that a large part of the process by which the actions of the individual muscles are harmonized and combined take place in the spinal cord. But this fact does not exclude the possibility that a higher co-ordination may be effected by the cerebellum. And a fair hypothesis has been suggested by Dr. Broadbent, namely, that what it does is to bring muscular action into relation with visual impressions, as when movements have to be guided by sight; whereas in the spinal cord they are arranged for tactile and other cutaneous impressions. However, a patient suffering from disease of the cerebellum may still stagger, although, in consequence of his optic discs having undergone atrophy, he can no longer see anything.

When the staggering is accompanied by a tendency to fall in one particular direction, we might fairly expect that this circumstance should not only indicate the presence of disease in one side of the cerebellum, but also show which side is affected. But tumors of the cerebellum often attain a very great size, and annihilate a large part of the organ. Thus it is peculiarly difficult to avoid the difficulty referred to above; the same affection may produce diametrically opposite effects, according as its action is that of a "destroying" or of a "discharging" lesion. In 1877 I made an autopsy in a case of Dr. Frederick Taylor's, in which a cheesy tubercle occupied the whole thickness of the cerebellum, rather to the right of its centre; and the right lateral lobe was universally pale and softened, and full of granule masses. The patient was a boy, five years old. It so happened that the exact seat of the mischief had been indicated several months before his death, by a bulging of the right side of the occipital bone, which was so much thinned at one spot, that it would yield on pressure and rebound like a piece of tin or cardboard. At the same time it was noticed that when he sat up in bed he had no balancing power; but

not that he rolled over on one side rather than on the other. The eyes, however, were affected with a peculiar form of nystagmus, which might, perhaps, have been turned to account as a localizing symptom. "It came on only when he lay on the left side; the eyes would then gradually fall over to the left, and be suddenly jerked back to the right; and these movements would be repeated rhythmically. When he sat up in bed there was a continuous rhythmical movement of the head from left to right." Afterward, six weeks before he died, "he lay on his back, with his head turned to the left; his eyes were then directed to the left side, and jerked from time to time upward." Now Hitzig, following Purkinjé, has shown ("*Reichert's Archiv*," 1871) that by passing a galvanic current through the head of a healthy person, from one mastoid process to the other, one can obtain oscillatory movements of the eyes exactly like those observed in Dr. Taylor's patient; and in a subsequent paper he accounted for those effects by referring them to disturbances of the cerebellum. Dr. Ferrier, too, found that galvanizing the exposed cerebellum of monkeys and other animals caused the eyes to deviate in different ways, according as the poles were applied to different regions of its surface. Indeed, his experiments seem to indicate a very simple law of localization, namely, that when any part of the cerebellum is the seat of irritation, the eyes become turned in the corresponding direction, whether to the right or to the left, upward or downward, as the case may be. In many cases analogous movements of the head accompanied those of the eyes. Thus I suppose that a tendency to fall backward, which has sometimes been noticed when there has been disease of the cerebellum, would point to the upper surface of the organ as its seat; for Dr. Ferrier found that galvanizing that part in monkeys led to upward movements of the eyes, and to throwing back of the head. This writer speaks of Hitzig's results, referred to above, as being essentially the same with those which he has himself obtained; but the correspondence does not seem to me to be so close.

Dr. Taylor's patient had also left hemiplegia, which was, no doubt, due to compression of the right side of the pons by the tumor. I believe that whenever this form of paralysis occurs in such a case, it is always produced in that way, and that it is invariably "crossed," affecting the limbs on the opposite side to the cerebellar lesion.

But the functional relations of each half of the cerebellum are, nevertheless, chiefly with the limbs of the same side of the body, through the connections of the fibres of the middle peduncle with nuclei in the opposite half of the mesencephalon. This is proved, not only by anatomical and physiological investigations, but also (most convincingly) by the pathological fact that when there is congenital unilateral atrophy, affecting one hemisphere of the brain, one crus cerebri, and one side of the deep part of the pons, the wasting is found in the other side of the cerebellum, as it is in the other side of the spinal cord.

The Motor Tract.—Passing now to the regional diagnosis of lesions involving the motor tract, I may begin by observing that the greater part of the description of hemiplegia given in a former chapter applies also to the same symptom when it is produced by a new growth. But whereas we have seen that certain parts of the brain substance are far more liable than others to suffer from the effects of embolism, or of rupture of a cerebral artery, the distribution of tumors is comparatively irregular and capricious. They may interfere with the fibres which pass down to the spinal cord at any point in their course, and may either destroy the whole or part of them. On the other hand, it is wonderful how large a tumor may be found in the very substance of the motor tract, without completely abolishing its functions. In 1869, a boy, aged four years and a half, died in

hospital, under my care, after an illness of fourteen months' duration. Three months before his death he could sit up in bed, although he rolled about and his balance was easily upset. A month later he "could move his legs a very little." Dr. Moxon found that the pons was occupied by an immense caseous tubercular mass, which consisted of two halves, fused together, and preserving almost exactly the normal shape of the part; only a thin shell of nervous matter remained. It seemed marvelous that life could have been maintained while the mass was growing to such a size. And a few years ago I made an autopsy in which a tumor (certainly a very minute one) was found in one of the corpora dentata of the bulb, there having been no symptoms of disease of that part.

Then, again, paralysis of the limbs on one side of the body, or even of all four limbs, may be due to a mass pressing on the motor tract from without; for instance, to a sarcoma connected with the base of the skull, and compressing the medulla oblongata or the pons; or to a glioma in the hemisphere above the lateral ventricle, pushing downward and flattening the basal ganglia. And, in other cases in which the growth itself lies altogether outside the region, lesions of which are ordinarily attended with hemiplegia, this symptom is caused by the yellow softening that so often develops itself secondarily.

But a growth situated in the superficial convolutions of one *hemisphere* may affect the movements of the opposite side of the body, without either disturbing the corpus striatum mechanically by pressure, or involving it in any morbid action. The symptoms so produced are among the most interesting that come under the observation of physicians, and within the last few years they have attracted much attention from physiologists. In the main, their character is generally convulsive; but the spasms are often followed by a transitory paralysis; and it is still a question whether a loss of power may not occur primarily and alone. Bright was the first to point out, in the earliest volume of the "*Guy's Hospital Reports*" (1836), that some cases in which fits were due to a local lesion presented the peculiarity that consciousness was not lost. But, so far as I know, this form of convulsions had never been systematically investigated, until Dr. Hughlings Jackson took up its study, in the hope that it would throw important light upon the nature and seat of epilepsy in general. He insisted on the fact that *hemispasm*—the "mobile counterpart" of hemiplegia—must indicate a condition of "instability" in the convolutions which discharge through the corpus striatum, and must therefore be a symptom of disease in their convolutions or (possibly) in the corpus striatum itself. And he set to work to determine, so far as pathological opportunities might offer themselves, the exact seat of the lesions found where this symptom had been present. Very little, however, had been done in this direction when, in 1870, Fritsch and Hitzig made known the fact, that instead of the surface of the brain being insensible to galvanic currents, there were in the dog certain parts of the convolutions which reacted to such currents in a very definite way, each setting in motion some particular limb, or even some special set of muscles. Thus they laid down the position of "cortical centres" for the movements of the neck, face, fore and hind leg respectively. Three years later Dr. Ferrier repeated these experiments with the faradic current, employing various animals, and at length monkeys, in whom the convolutions are comparable, one by one, with those of the brain of man. Afterward Hitzig himself operated on a monkey. Between the results of these two observers there are some discrepancies in details. Hitzig used a comparatively weak galvanic current; thus he obtained movements over a far more limited area; indeed, his main object was to discover what parts of the cortex would yield movements limited to isolated groups of muscles, on

the most feeble stimulation. He maintains that in the monkey the motor centres for all parts of the body are situated in the anterior central (ascending frontal) convolution. That for the leg is nearest the falx cerebri, at a distance of about three millimètres from it; that for the arm lies three millimètres further outward; that for the upper part of the face, supplying the muscles of the ear and eye, is situated rather more behind and externally; that for the lips, tongue and jaws is six millimètres inward from the fissure of Sylvius. Hitzig does not deny that irritation of adjacent convolutions by more powerful (especially by induced) currents give rise to movements in distant parts; but he has found that the movements so produced are of a more general character, and he attributed them to the action of the current upon parts beneath the actual surface of the brain.

Ferrier places his centres in the posterior central (ascending parietal) as well as in the anterior central convolution of the monkey; and also in the postero-parietal lobule, in the back part of the third frontal, in the angular, and in the superior temporo-sphenoidal convolutions. He agrees with Hitzig in placing the centre for the leg close to the median line, but he divides it into two; one in the postero-parietal lobule, for advance of the hind limb, as in walking; the other in the upper parts of the ascending parietal and ascending frontal convolutions, for movements bringing the hind foot up to the abdomen. He makes the centres for the arm and hand occupy the ascending parietal convolution nearly as far outward as the fissure of Sylvius, as well as the ascending frontal and the superior frontal convolutions outside and in front of one of the leg centres. The movements of the lips, tongue, mouth and larynx are located in a series of centres occupying the outer part of the ascending frontal, and in one in the supra-marginal convolution. Centres for the movements of the eyes are believed by Dr. Ferrier to occupy an extensive area in the two upper ("first" and "second") frontal convolutions. Stimulation of the angular gyrus or of the superior temporo-sphenoidal convolution, also causes the eyeballs to move, with dilatation of the pupils; but he supposes that these parts are centres for the senses of sight and hearing, and that the movements are reflex.

Such are the data, so far as we at present possess them, upon which we have to base the localization of lesions attended with spasmodic movements in particular limbs or in special parts of the face. Obviously they are incomplete, and in all probability they are not perfectly accurate. As to those points upon which Hitzig and Ferrier differ, other experimenters, as Munk and Goltz, have made themselves heard; but even if Hitzig should prove to be right in assigning very narrow limits to the area within which motor centres occupy the surface of the convolutions, Ferrier's statements would not necessarily lose their pathological value. For tumors and other lesions extending some distance into the cortex might still be found to produce effects like those of the more powerful currents which Ferrier employed. The only spot on the surface of the brain to which these two observers respectively give distinctly opposite functions, appears to be the middle of the ascending frontal convolution; where Hitzig places the centre for the muscles of the upper part of the face, Ferrier puts one for flexion and supination of the forearm.

In the first place, however, it is to be noted that the occurrence of convulsive attacks without unconsciousness beginning in, or limited to, some particular part of the body, is not of itself a proof that a recognizable local lesion exists in the brain, as Bright supposed. When I have to speak of epilepsy I shall cite several instances in which there was every reason to believe that attacks of the kind just referred to were such as are provisionally termed "functional." And in a case of Dr. Hughlings Jackson's, that

terminated fatally, the only morbid change discovered at the autopsy was atrophy of both hemispheres, although the fits which had occurred had begun in the right index finger and thumb, and had often been unattended with loss of consciousness ("*Med. Times and Gaz.*," 1872). Other symptoms of the presence of a new growth must therefore always be carefully looked for before one hazards a diagnosis.

Again, it is perhaps worthy of notice that, according to Dr. Jackson, the seat of localized convulsive seizures, or of general seizures beginning with local spasms, is, in the majority of cases, some part of the hand, and generally the thumb or the forefinger; that the next most frequent seat is the cheek or the tongue, and that it is much more rarely the great toe (the part of the foot most liable to be affected in this way). For one would be led by these facts to attach comparatively little value to the circumstance that a lesion should now and then be found in some particular convolution when the hand had been the starting point of the movements, unless cases in which they had begun in the face should constantly show morbid changes in a different part of the cortex. Whether the observations hitherto made are too numerous to be regarded as mere coincidences may perhaps be doubtful. So far as I am acquainted with them they are as follows: (1 and 2) Two cases of Dr. Jackson's (one recorded in the "*Med. Times and Gaz.*" for 1872; the other, so far as I know, unpublished, but cited by Dr. Ferrier) in each of which the spasms always began in one thumb, and a lesion was found in the hinder part of the opposite first frontal convolution. (3) A case in which I made an autopsy at Guy's Hospital, and in which there was a small glioma in exactly the same spot on the right side, there having been repeated fits without unconsciousness, starting mostly in the left foot, but occasionally in the left arm. (4) A case of Griesinger's, that of a man who was attacked with transient spasms in the right leg, afterward affecting the right arm, the face, and the tongue: a cysticercus, of about the diameter of one inch and a half, lay close to the left side of the falx cerebri in such a position that its anterior extremity coincided with a line drawn vertically upward from the ear. These cases correspond equally well with Hitzig's and with Dr. Ferrier's statements; the next one accords better with those of the English observer. It is (5) recorded by Dr. Dreschfeld, of Manchester ("*Lancet*," 1877). The convulsive movements began with a sudden clinching of the left fist, flexion of the wrist, and pronation of the forearm, while the left angle of the mouth was at the same time strongly drawn downward; a local syphilitic lesion—probably the residue of a gumma—was found in the adjacent parts of the ascending parietal and supra-marginal convolutions. (6) A case observed by Hitzig himself seems, on the whole, not to answer more closely to his than to Dr. Ferrier's experiments. The patient was a French soldier who was wounded on the right side of the head, near Orleans, on December 14th, 1870. On February 4th, 1871, he was attacked with clonic spasms affecting the left side of the mouth and nose, the eyelids, and afterward the fingers on that side. After death there was found to be a local necrosis of the parietal bone, and an abscess in the right ascending frontal convolution at the level of the sulcus between the second and third frontal convolutions. (7) Another instance, very similar, has been recorded by Wernher ("Virchow's Archiv," lvi). The patient, who had received a wound which penetrated the left temporal bone, was attacked with convulsions limited to certain muscles on the right side, especially those of the angle of the mouth, the ala nasi, the eyelids, and the tongue, but also spreading to the fingers and to the neck; a small part of the surface of the brain was found to have been crushed, including parts of the convolutions on each side of the Sylvian fissure in a line with the lower end of the fissure of Rolando. On the other

hand, there is a case (8) of Dr. Gowers ("*Brit. Med. Journ.*," 1874) in which spasms began in the left angle of the mouth, involving afterward the frontal muscle on each side, but in which the only local lesion appeared to be a clot of blood situated above the right lateral ventricle just inside the gyrus funicatus. This case is certainly far from corresponding accurately with the observations of Hitzig and Ferrier, but it may justly be said that no instance hitherto recorded stands so directly in antagonism with the inferences drawn from these observations, as might be the case if, for example, a small tumor were found in the extremity of one of the posterior lobes, when spasms limited to some one part of the body had been present during life.*

A further question, which is of the highest theoretical interest, is whether affections of particular convolutions are capable of causing limited paralyses instead of spasms. Dr. Hughlings Jackson long ago pointed out that unilateral convulsive seizures were often followed by a more or less complete hemiplegia; but he maintained that this form of paralysis was always transitory, passing off in a few days or weeks, and he thought that it was dependent, not on the organic disease, but on "overwork" of the nerve fibres passing to the muscles which were convulsed. And it is no doubt true that a persistent local paralysis is seldom or never caused by a cerebral tumor, or by any similar affection, unless it interferes directly with the corpus striatum, or with some part of the motor tract. But the very distinction on which Dr. Jackson has laid so much stress—between "discharging" and "destroying" lesions—would tend to show that, although such an effect may not be capable of being produced by a new growth situated in a convolution, it may yet result from softening of the very same part. When discussing the effects of arrest of blood supply to different regions of the brain (p. 504), I expressly reserved this question; and almost every instance of aphasia is a case in point. Thus the most careful scrutiny of the convolutions near the fissure of Rolando should in future be made whenever a patient in whom a local patch of softening is likely to occur has had unilateral ptosis, or paralysis confined to part of the face, or to a finger, or any part of the upper limb on one side. Dr. Ferrier describes the results of his experiments in this direction as most striking. In one monkey he destroyed by the cautery all the centres for the opposite arm and leg, except that for flexion of the forearm; the result was that the animal retained only this particular movement. In another monkey he cauterized the centre for the biceps muscle alone; the animal could move all other parts of its limbs, but its arm hung in a state of flaccid extension

* I shall, however, have to mention a case of Sir William Gull's, in which a large abscess in one posterior lobe was attended with attacks of spasm limited to one arm; and a few other cases have been recorded which would appear to be inconsistent with the experimental results given above, were it not that legitimate criticism seems to deprive them of definite significance. One of these was observed by Dr. Jackson. The foot had been the starting point of the spasms, and a tumor was found which involved the lower part of the ascending frontal convolution; but the brain also presented other lesions. Another is a case of Dr. Gowers', in which there was thrombosis of the superior longitudinal sinus and of some of its afferent veins, with hyperæmia of parts of the three frontal convolutions, situated further forward than any recognized "cortical motor centres;" but in that instance the fits were general, although they began with a slow movement of the hand to the head; and death occurred within two hours from the time of their commencement. Again, in a patient of Dr. Buzzard's, a girl of 18, a tumor of the size of a walnut was found in the white substance of the *left* hemisphere, extending as far as the gray matter of the gyrus fornicatus; the cause of death was phthisis. The symptom which had suggested the idea that a local lesion might be found, was that she had fits, beginning with an aura in the *left* wrist; but this is, of course, quite a different thing from the occurrence of spasm in the same part. Moreover, the application of a blister to the seat of the aura sufficed to transfer it to the opposite arm.

by its side. The only clinical observations on this head that I know of are two of Löffler's quoted by Hitzig. One is the case of a man whose two parietal bones were both fractured at the vertex by a gunshot wound, and who had paralysis of both legs. The other is that of a man who was wounded at the upper and anterior angle of the left parietal bone; his right leg instantly became paralyzed, and he fell to the ground. On the seventh day the loss of power extended to the right arm also, but this quickly got better, while in the leg the recovery was very slow. Theoretically, a paralysis caused by a cortical lesion ought sometimes to be characterized by its being limited to some special movements of the affected muscles. An instance of this is afforded by Hitzig's case of the French soldier referred to above. His spasms were followed by a partial paralysis of the lower part of the face on the left side, and it is expressly stated that he could voluntarily bring the muscles into action as well as on the opposite side, although that half of the face remained almost motionless, when it should have moved in common with the right side, for the purpose of expression. This is exactly the opposite of what occurs when disease of the corpus striatum causes paralysis of the face (see p. 500).

The Cerebrum Outside the Motor Tract.—We have still to consider what "localizing symptoms" may be present when a lesion is seated in the substance of one hemisphere, or in the superficial convolutions, beyond the region in which motor centres are placed by Hitzig, or by Ferrier. Now if the latter is right in thinking that he has discovered cortical centres for the special senses, unilateral blindness or deafness may, perhaps, hereafter serve to indicate the presence of disease in the angular gyrus, or in the superior sphenoidal convolution respectively; or, again, loss of smell and taste may be associated with an affection of the under surface of the temporo-sphenoidal lobe. But in many cases the only symptoms that could be called local seem to be lethargy, listlessness, an oddness of manner, taciturnity, and unwillingness to speak, or even loss of memory and impairment of intelligence—the very symptoms that we are accustomed to associate with the existence of diffused morbid changes affecting the whole of both hemispheres more or less uniformly.

Diagnosis.—The diagnosis of the affections described in this chapter involves two distinct questions. First, they have to be distinguished from other diseases of the nervous centres or of distant parts; secondly, they have to be differentiated from one another. The answer to the former question is often wonderfully positive and exact. To the young student—who, perhaps, knows the difficulty of distinguishing between tumors and other surgical affections of parts that can be seen and handled, or is accustomed to those symptoms of pathological classification in which new growths come at the end of long lists of more simple morbid changes—nothing in the art of medicine is more striking than the easy confidence with which the physician sometimes asserts the existence of such a lesion in the interior of the cranial cavity.

Cases do, indeed, occur which are only cleared up in the post-mortem room. A man of whom little is known, or who has hitherto displayed no very marked symptoms of brain mischief, may die in a succession of fits, or in coma of a few hours' duration, and it may be difficult to make out that the cause is not cerebral hemorrhage, or thrombosis of an artery, or uræmia. Or, if he lives for two or three weeks, the disease may appear to be meningitis.

But, as a rule, the illness caused by a tumor, or a tubercle, or a syphilitic gumma, in the brain begins gradually, and goes on for several months, or even for some years. In the cases that have occurred at Guy's Hospital

within the last few years the duration of well-marked symptoms has generally been from three to nine months, but one patient had had fits for four years. Dr. Hughlings Jackson has recorded the case of a woman who had optic neuritis and staggered in walking in 1865, and who did not die until the summer of 1872, when she had become insane; there was a growth springing from the "floor of the sphenoidal fossa." Now I believe it may be said that whenever cerebral symptoms of the kind described in the present chapter have lasted for more than five or six weeks, the only other organic affections of the brain by which they can be caused are abscess and certain diffused morbid changes.

With these two exceptions, the only cases in which it is really difficult to diagnose a local lesion in the brain, are those in which it is doubtful whether there is anything more than functional disturbance. In women the symptoms are sometimes so vague that for a long time they are mistaken for those of hysteria. And some years ago a painter died in Guy's Hospital, of what were believed to be the effects of lead poisoning (drowsiness and epileptiform fits), when a spindle-cell sarcoma of the size of a marble was found in the right hemisphere, with extensive softening around it. When a patient's sole complaint is of having suffered for some weeks, or a few months, from a continuous (or even a paroxysmal) headache, unlike any pain to which he had previously been liable, it is often impossible to be quite sure whether organic disease is present. In such cases, and in many others, the opthalmoscope is of the greatest service. The discovery of choked discs, or of optic neuritis, or of atrophy, goes far toward establishing the presence of an organic lesion. At one time, indeed, the significance of these appearances was undoubtedly rated too highly; and I am not at all sure that this is not still the case In the first place, it is to be observed that neuritis, or choking of the disc, in one eye only, is sometimes a sign that the cause lies in the orbit rather than within the skull. I have already referred to a case of Mr. Lawson's, brought before the Clinical Society in 1876, in which an hydatid cyst pressed upon the nerve behind the eye, and so caused great swelling of the disc on that side. But, in other instances, a uni-ocular neuritis is due to pressure upon a single optic nerve between the chiasma and the opening through which it escapes from the cranial cavity; a gumma at the base seemed to have acted in this way in a case recorded by Mr. Hulke in the "*Ophthalmic Hospital Reports.*" Indeed, Dr. Hughlings Jackson has met with two cases in which the ophthalmoscope revealed an affection of one eye, dependent upon the presence of a tumor in the opposite cerebral hemispheres. But it is a rule, to which there are very few exceptions, that both optic discs suffer whenever disease of the brain is the cause. It does not always happen that the changes advance with equal rapidity in the two eyes; according to Dr. Allbutt that disc which is on the same side as the cerebral lesion is sometimes first and chiefly affected.

In some rare instances ophthalmoscopic changes seem to precede all other symptoms. Thus Dr. Allbutt speaks of a patient under his care, who had amaurosis from atrophy for three years before any signs of cerebral tumor began to manifest themselves, although these afterward became well marked. But that writer himself frankly acknowledges that further experience has compelled him to modify the strong opinion which he at one time entertained, that the existence of disease of the brain might be almost certainly inferred from morbid appearances in the optic discs. Indeed, among a series of unselected cases, recorded by Mr. Hulke in the "*Ophthalmic Hospital Reports*," there are several in which optic neuritis, terminating in atrophy, seemed either to be a spontaneous affection, or to be attributable only to such vague causes as child bearing, lactation,

leucorrhœa, sexual excesses, or to an antecedent attack of diphtheria or rheumatic fever. And as far back as 1866, von Graefe spoke of it as being probably sometimes due to menstrual disorders.

But even when unmistakable indications of cerebral disturbance are present, it is a question how far one is justified in concluding from ophthalmoscopic appearances that there must be a definite organic lesion in the brain. A remark of Mr. Hulke's appears to me very striking, namely, that out of a considerable number of examples of optic neuritis, believed to be dependent upon intracranial affections, he had, in 1868, only three times been able to verify his diagnosis by an autopsy. Now, there would be nothing wonderful in this if pathologists were often finding the residues of long past diseases in the brain, or even if it were known that life could be prolonged indefinitely, when a local lesion was present. But the facts are exactly the reverse; except, indeed, that patches of old softening are met with, and the remains of hemorrhages, such as do not cause any changes in the discs. Nor do the other symptoms of the cases of which Mr. Hulke gives the details seem to me to support the view that they were dependent upon organic mischief in the brain. Three of them, all of which terminated in recovery, were thought to be probably examples of meningitis; but apart from the ophthalmoscopic appearances, I doubt whether this diagnosis could be justified. Another case would certainly have been regarded as one of ordinary epilepsy if the eyes had not been examined. Two patients, both advanced in years, had hemiplegia, and ultimately died of apoplexy; one, a man, aged thirty-eight, attributed his illness to watching a solar eclipse; another, a woman, aged twenty-four, ascribed hers to standing in the garden with her head uncovered.

Dr. Hughlings Jackson, indeed, thinks that a double optic neuritis is almost certain evidence of what he terms "coarse disease" within the cranium, but he admits that he has himself met with a few instances in which, on post-mortem examination, no such disease could be found. Of one case of this kind he has published full details. A woman, aged thirty-four, had for about a year been subject to attacks of severe headache accompanied with vomiting; for three months she had been blind. Her illness began by her being seized with vertigo and momentary unconsciousness, after which she had headache for four days. At another time the pain lasted for three weeks. She was admitted on December 19th, 1874. On January 6th, 1875, she had an attack of pain so intense as to make her toss her head from side to side, holding it in her hands, and crying, "Oh, my head; I don't know what I shall do." She retched and vomited frequently. Both optic discs were greatly swollen, and the veins in them were dilated and tortuous. After the 10th she sank gradually into what appeared to be natural sleep, which, however, passed into coma, and on the 12th she died by failure of the respiration. A tumor or some similar disease was confidently anticipated, but Dr. Sutton, who made the autopsy, found only certain microscopical changes in the substance of the cortex. Dr. Jackson speaks of this case as exceptional, but may one not fairly doubt whether it is peculiar in the absence of a visible cerebral lesion, so much as in its having proved fatal without the presence of such a lesion? Compare with it a case that was brought before the Clinical Society, in 1876, by Dr. Goodhart and Mr. Higgins. A girl, aged twenty-one, was attacked with intense headache and vomiting on December 21st, 1874, at the very time when Dr. Jackson's patient had just entered the London Hospital. Some months previously she had been stunned by a severe blow from a stone on her right temple. On the 24th, there was well-marked double optic neuritis, her pulse was irregular, and only 52 in the minute, and she had no fever. Afterward she had paralysis of each sixth nerve, suffocative attacks in which she could

hardly breathe, constipation, transient hemiplegia, delirium, and an affection of the speech, so that her mother could not understand her. But between the 6th and the 14th of January, 1875, all her symptoms began rapidly to subside, and before long the impaired state of her sight seemed to be the only thing that troubled her, except that she was unable to take solid food without vomiting. Dr. Goodhart discussed the diagnosis as between meningitis and the presence of a yellow tuberculous mass at the base of the brain; he does not seem to have thought that any other view of it could be taken.

In describing meningitis I shall have to mention cases in which symptoms very like those of that disease have been associated with double optic neuritis, but in which, although they terminated fatally, no lesion could be discovered, whether in the membranes or in the cerebral substance. Surely it is but common sense to suppose that, if one could see the state of the brain in cases which recover, it would be found equally free from obvious morbid changes. I am quite ready to assign to ophthalmoscopic appearances a value co-ordinate with that of other symptoms in the diagnosis between organic and functional diseases, but it seems to be thought that they stand on quite a different footing from the rest, and I cannot see that this claim has as yet been established.

Another question is whether any special influence can be drawn from the exact character of the ophthalmoscopic appearances in a case believed to be one of local organic disease of the brain. Meningitis often accompanies a gumma of the base; and a tumor in any part may set up extensive yellow softening. It might be thought that neuro-retinitis, as distinguished from choking of the disc, would indicate the presence of such secondary affections. But one of Mr. Hulke's cases seems to show that the former change may depend upon the presence of a sarcoma attached to the floor of the skull, without there being any evident inflammation of the membranes or of the brain itself. Nor do I know that simple atrophy of the disc, without antecedent exudation into its substance, could be taken as a proof of the existence of hydrocephalus, although Dr. Allbut suggests that distention of the third ventricle often causes such an atrophy, by stretching of the optic tracts and the chiasma.

The *differential diagnosis* between the several forms of local organic disease of the brain often rests on no higher basis that that of probability. In a child, there is a strong presumption in favor of tubercle. I may take the opportunity of remarking that, although Dr. Hughlings Jackson has recorded several instances of epileptiform convulsions or hemiplegic paralysis in children the subjects of inherited syphilis, there is as yet no pathological evidence that intracranial gummata are ever developed in such cases; so far as we know at present the association may have been a mere accident. The older the patient, the greater the chance that the lesion is some other form of tumor than tubercle; and above the age of forty the latter may be left out of consideration. To distinguish between these two affections, however, is of very little consequence; what is really important is that one should never overlook syphilis as a cause of cerebral mischief. It is particularly to be noted that in many instances in which gummata are developed in the brain, no nodes upon the bones can be discovered, nor any indications of past or present orchitis, iritis, or cutaneous eruptions. In only three or four of the ten fatal cases of this kind that I have recently met with was it ascertained during life that the patient had suffered from any venereal disease. There are, of course, persons—especially unmarried ladies—in whom it would be unpardonable to suspect the existence of a syphilitic taint. But, on the other hand, it may be of the highest importance that neither the high social standing of a

patient, nor even a general reputation for purity of life, should prevent our giving him the chance of being saved from what may be the remote effects of a long-forgotten transgression. Dr. Buzzard lays great stress on the presence of a muddy complexion, and other signs of a cachexia not traceable to any definite visceral disease, as pointing to syphilis. It is well known that in most cases the pains caused by this disease regularly occur, or at least undergo a marked aggravation in the evening. What seems to have more weight than anything else in suggesting syphilis as the cause of obscure nervous symptoms, is the impossibility of referring them to any single lesion. Dr. Jackson long ago insisted on the value of this principle; and Dr. Buzzard has illustrated it by cases in point. Thus two of his patients had paralysis of one arm and of both legs at the same time; the inference was that there was an affection of the spinal cord as well as of the brain. I must confess that it is not clear to me that one can ever positively assert that there must be more than a single lesion in the brain itself. Moreover, it would have to be remembered that secondary tumors are often multiple, and that the presence of a primary new growth elsewhere is very apt to be overlooked. Thus a patient coming to a physician for headache and paralysis may very likely never mention that there has for years been a tumor in the breast, or that one testicle was excised some months before. Again, the original seat of the malignant disease may be some internal organ, where such an affection is difficult of detection. Out of sixteen cases of secondary growths in the brain there were no less than six in which the starting point of the mischief was a sarcoma surrounding the root of one lung, and in five of them this was not discovered during life.

Prognosis.—All that I know of the progress of cases of cerebral tumor or tubercle would tend to show that these diseases are inevitably fatal. The nearest approach to a recovery that I remember to have heard of occurred in a boy, aged fourteen, who was under my care in Guy's Hospital in 1867. About two years previously he had been taken ill with "pain in the back of the head, loss of sight, and fits in which he used to clinch his hands." On May 1st, when he had been in my ward for three weeks, it is noted that "he lies apparently unconscious of everything, and cannot be roused. His head is constantly thrown backward; and when he is touched there is a sort of opisthotonos. He is completely amaurotic; his pupils are equal and slightly dilated." On May 4th the report is, "He is slightly more conscious, he recognizes his mother, and will raise his hand into the air when told to do so. His evacuations are passed involuntarily." During the next three months the chief change was that he slowly wasted away, until he was reduced to a mere skeleton; the only sign of intelligence that he ever manifested consisted in lifting his hand, as already described. But one day, in going round the ward, I spoke to him; and to the astonishment of every one he slowly articulated a few words in reply. From that moment he began to improve. He took food well, regained flesh, talked more and more every day, got up, walked about the ward, and at last was discharged perfectly well, except that he was blind. Afterward he attended among my out-patients. I have no notes of his symptoms during this period, but I remember that he complained of paroxysmal headache, and of epileptiform fits. He was readmitted, and died. The notes of the autopsy have unfortunately been mislaid. But I recollect enough to be able to state that there was an irregular calcareous mass, of about the size of a marble, imbedded in the floor of the ventricle, with some mucoid fluid round it.

Even when the local lesion in the brain is a syphilitic gumma, the prognosis is not very favorable, so far as concerns the patient's ultimate restoration to health. For a time, active treatment is generally followed by very striking results; consciousness is regained, paralysis is recovered

from, headache subsides, epileptiform convulsions cease to recur. But after an interval the symptoms return; perhaps the opposite limbs are now paralyzed, or the affection may assume a paraplegic instead of a hemiplegic form. A second course of medicine may again be successful, but at last our efforts are baffled, and the patient succumbs. Such cases are often prolonged over a period of several years. On the other hand, it sometimes happens that the cure is permanent; and more often, that each return is milder than the last, until the disease gradually wears itself out.

Treatment.—In the treatment of this affection one should not trust wholly to iodide of potassium, even in large doses. A course of mercury should always be prescribed. The bichloride may be given internally, or recourse may be had to calomel-vapor baths or to inunctions with blue ointment.

Such a line of practice often proves brilliantly successful where there was no proof that the symptoms were due to a syphilitic lesion. The question then arises whether the proof is supplied by the result of the treatment. Upon this point I would not speak dogmatically, but it seems to me not improbable that absorbent remedies may, in some cases, possess the power of arresting the growth of sarcomatous tumors. Wunderlich believed that small doses of arsenic, continued for a long period of time, were sometimes useful.

The palliation of symptoms is always possible. If there be epileptiform convulsions, full doses of bromide of potassium should be given. The same remedy will often relieve headache or giddiness. Or it may be necessary to administer opium or morphia by the mouth, or to inject the alkaloid subcutaneously. Dr. Reynolds speaks highly of Indian hemp, as sometimes altogether removing pain in the head. He also recommends the local application of ice.

Red Softening.—I have still to describe those organic diseases of the brain which consist in local, circumscribed inflammation. These are commonly said to be two in number—red softening and abscess.

The former, *ramoulissement rouge*, is among the most curious morbid changes to which the cerebral tissue is liable. It is attended with great swelling; convolutions so affected are far broader, and the sulci between them much deeper than natural; while a corpus striatum or a thalamus is rounded, prominent, and generally increased in size. As is implied by the name, there is a marked diminution of the natural, firm consistence; and the color is altered, gray matter assuming a deep purple tint, while white matter becomes pink or red, with numerous minute ecchymoses. Under the microscope the capillary and other vessels are found dilated and engorged; blood corpuscles are seen extravasated among the nervous tissues; if the affection has been of sufficient duration, they are fused together, having undergone conversion into shapeless masses. According to Rindfleisch, pus cells are collected about the smaller blood vessels, so as to form sheaths for them. Dr. Moxon, however, says that he has been surprised to find how slight were the histological changes in some cases of this kind. The elements of the tissue were softened and granular, but they still retained their form; and no characteristic inflammatory products could be recognized.

Red softening may arise from a variety of causes. One of them is injury. Some years ago a woman died in Guy's Hospital, who had fallen three or four months previously, and struck her head against the wall. Three weeks before her death she had a fit, which was followed by a partial left hemiplegia, affecting the side of the face and tongue, and accompanied with ptosis. On post-mortem examination all the parts at the base, between the optic commissure and the cerebellum, were found by Dr. Wilks to be in

a state of softening, "partly red and partly white;" one-third of the thickness of the pons was so affected, and nearly the whole of the crus cerebri. Again, red softening may accompany an inflammation of the membranes, as tubercular meningitis. I have already spoken of it as sometimes arising in the neighborhood of an apoplectic clot, round a tumor, or in a part of the brain deprived of its blood supply by thrombosis or embolism.

It is a question still undecided whether red softening of the brain ever occurs as a primary and independent morbid process. In the records of post-mortem examinations at Guy's Hospital I find very few cases that could be so interpreted. There are two instances, in both of which more or less of the posterior lobe of the left hemisphere of the brain, and a considerable part of the cerebellum on the same side, presented the change in question; but in each of them the cardiac valves showed recent vegetations, so that it seems probable that the affection was dependent upon embolism of some of the smaller arteries. In 1876 I made an autopsy in which parts of the right superior and middle frontal convolutions were swollen, soft, and of a gray-pinkish color. There was no caseation and no definite edge such as would have suggested the presence of any new growth; I should have regarded the affection as an acute cerebritis had not the microscope revealed the presence of a large number of oval and round cells infiltrated between the nerve fibres. I should therefore be skeptical about any case of supposed primary red softening or local cerebritis, unless I had made a careful examination of every part of the affected structures. Some years ago a woman, aged twenty-six, died in the hospital under one of my colleagues, after an illness of three months' duration. The falx cerebri was found adherent to the anterior parts of both hemispheres by granulation tissue. On section the frontal lobes appeared of a brick-red color; their cineritious substance was swollen, and the boundary line between it and the white matter was ill defined. On the left side this change extended down to the lateral ventricle. The affected parts were rather harder than natural; but in all other respects the disease corresponded perfectly with the descriptions which writers have given of a local cerebritis; and, unless there was a new growth which was overlooked, I think it must have been of that nature. The microscope only showed a corpuscular infiltration, with granular matter and compound granule masses; but I am not sure whether attention was directed to the possible presence of a small new growth in the midst of the inflammatory mischief. There was, of course, nothing in the symptoms to indicate the character of the morbid process.

Huguenin expresses doubts as to the existence of a "spontaneous encephalitis," but he, nevertheless, describes various retrograde changes as consecutive to red softening in such a way as to imply that it may be a substantive disease. He says that the patches may either subside entirely, or pass into a condition of yellow softening with cavities full of serum, or undergo cicatrization, or become converted into tough, dirty-white, indurated masses. I have never seen such appearances in any case in which there was reason to suppose that a condition of red softening had existed, rather than an effusion of blood or an infarctus; and I do not understand on what evidence Huguenin's statements are based.

Abscess of the Brain.—This, again, is an affection which is decidedly of rare occurrence, both absolutely and in comparison with many other cerebral diseases.

Origin.—Most pathologists speak of suppuration in the substance of the brain as a further stage of red softening. It is true that in cases of pyæmia one sometimes finds reddened patches apparently antecedent to the forma-

tion of the pus; indeed, a similar condition is also now and then seen in the neighborhood of an actual abscess. In the latter case, however, it might fairly be regarded as the result of a distinct morbid process affecting the brain tissue round the wall of the abscess, just as it may affect that which surrounds a tumor or an apoplectic clot. And there are not wanting facts which seem to show that abscesses generally arise independently: one is that when several suppurating cavities are found in the same hemisphere—however small and recent they may be—the intervening cerebral substance is commonly pale and of firm consistence; the other is that the causes of red softening are different from those of abscess. We have seen that the former affection is attendant upon some other morbid change in the same part of the brain; but this is very rarely the case with the latter.

Cerebral abscess, indeed, is itself seldom or never a primary affection, except when it is caused by injury; but the diseases to which it is secondary belong not to the brain itself, but to more or less distant parts. Chief among them are *diseases of the ear*, especially suppurative inflammation of the tympanum. Otorrhœa, if prolonged, always involves the risk of extension of mischief through the bone to the internal surface of the skull, so that a person affected with it should never be taken for life assurance at ordinary rates. In the "*Medical Times and Gazette*" for 1863 a case is recorded of a patient who for several years had a discharge from the ear, but who lived to the age of sixty-six, and then died of a cerebral abscess. And it often happens that an aural affection which had been present from early childhood kills a grown-up man or woman. In many instances the several stages of the morbid process are plainly traceable after death. The tympanic cavity is found to be bare and carious, or even necrotic; the dura mater over its roof is raised from the bone by pus, or it is softened and sloughing; the arachnoid and pia mater adhere firmly to it, and at this very spot the middle lobe of the cerebrum contains an abscess, which comes close to its under surface. Or, on the other hand, the caries may pass from the mastoid cells or from the petrous bone to the posterior fossa of the basis cranii, and then the abscess occupies the corresponding half of the cerebellum. I find that among twelve successive cases at Guy's Hospital, there have been seven in which the middle lobe of the cerebrum was the seat of the inflammatory process, five in which it lay in the cerebellum. According to Huguenin and Meyer, the right side of the encephalon is much more apt than the left to be affected with abscess from disease of the ear. Indeed, among twenty-four cases of this kind collected by Gull and Sutton, that side was affected in no less than eighteen, but it is curious that out of eleven of the cases that have occurred at Guy's, the abscess was on the left side in six, on the right side in only five. Aural surgeons formerly laid great stress on the diagnosis of caries of the tympanum, as indicating the danger of extension to the brain. They did not hesitate to pass a probe into its interior in search for rough and denuded bone, but apart from the risk which such a procedure carries with it, of breaking through the wall, and so of setting up the very mischief which is dreaded, experience has shown that the detection of caries is less important than was supposed. In many instances of cerebral abscess set up by inflammation of the middle ear, the bone is found after death to be healthy. There is more than one route by which the morbid process may reach the interior of the skull in such cases. It may pass along the bony canals which transmit the superficial petrosal and other veins, or through the spaces in the diploë which convey vessels from the tympanum to the dura mater, or it may first extend to the labyrinth, and then follow the course of the seventh pair of nerves. A curious circumstance is that in many instances there is a tract of apparently healthy cerebral substance between the wall of the abscess and the

petrous bone. It is especially when the inflammation of the ear assumes a putrid character that one should be on the lookout for the speedy supervention of disease within the cranium. Von Tröltsch mentions, as a sign of danger, the circumstance that lead lotions are blackened by the pus; other indications are the suppression of the discharge from the external meatus, and a sudden increase of pain in the ear, which may amount to the most intolerable agony. But in some cases there is no pain at all: there may even be no otorrhœa, for the secretion may be retained behind a perfect membrana tympani; and thus, unless one tests very carefully the patient's auditory powers, we may easily overlook the fact that he has anything the matter with his ear.

Another, but a far less frequent cause of abscess of the brain, is chronic *disease of the nose*. Two such instances were recorded by Sir William Gull in the "*Guy's Hospital Reports*" for 1857. Each patient had had mucus discharge from the nostrils; in one case the abscess was in the middle lobe, in the other in the anterior lobe. Other writers have given cases in which a nasal polypus was the starting point of the mischief. I once made an autopsy in which an ulcerating epithelioma of the lip and cheek extended to the base of the skull along the third division of the fifth nerve, and set up an abscess in the middle lobe of the brain. Necrosis or caries of the calvaria from any cause, if attended with sloughing of the dura mater, may have a like effect.

General *pyæmia* sometimes leads to the formation of one or more abscesses in the brain, as in other parts of the body. In the post-mortem records of Guy's Hospital I find six instances of this during the period within which the twelve cases occurred that I have mentioned of cerebral abscess arising from disease of the ear. Usually the blood disease was directly fatal, but in one remarkable case a man, aged thirty-one, after perineal section for stricture got a number of abscesses about his body, which disappeared without being opened; he left the hospital and worked in the fields for more than a year, and was then attacked with brain symptoms, which killed him about eighteen months after the operation.

This case is of special interest because it forms to some extent a connecting link with a very curious group of cases of cerebral abscess, which are distinctly secondary to pre-existent suppurative inflammation in a remote part of the body, but in which there are at no period any symptoms of blood poisoning, and in which no pyæmic infarcts or abscesses occur anywhere but in the brain. I believe that Sir William Gull was the first to point out the ætiology of such cases, although a similar instance had before been recorded by Abercrombie. The most remarkable point about them is the fact that the lung is generally, if not always, the seat of the primary lesion, upon which the abscess in the brain is dependent. At Guy's Hospital I find that within the last few years we have had six cases of this kind; six others are given by Gull and Sutton in the second volume of Reynolds' "System of Medicine," and some have been recorded in Germany by Biermer, Huguenin, and Meyer. The supposition has generally been that the cerebral inflammation is set up by a portion of thrombus washed out of a pulmonary vein, and carried to the brain in the blood; and Böttcher is said to have found in the floor of an abscess of the brain, which was secondary to a pulmonary abscess, some pigment which he was able to identify as having come from the lung. The nature of the thoracic disease has varied in different instances. Adding the cases which have recently occurred at Guy's Hospital to those related by Gull and Sutton, we obtain a series of twelve examples of this form of abscess of the brain. Among them there are three in which the primary affection was a pleurisy, two of tubercular phthisis, two of some form of acute pneumonia, one of

simple bronchitis; in each of the remaining four it seems to have been a cirrhosis (chronic pneumonia), with dilatation of the bronchial tubes or sloughing cavity in the indurated tissue. Huguenin places bronchiectasis with stagnant, putrid secretion, at the head of the list of pulmonary diseases which have given rise to cerebral abscess. It is important to note that the mischief in the lung may be altogether latent. In one of the above cases Dr. Moxon says that he almost despaired of finding a primary lesion, until at last he discovered that the mucous membrane of the right bronchus was extensively ulcerated, with its cartilages exposed and necrosed; and in the case which I have spoken of as having been one of simple bronchitis, the only sign of any pulmonary affection was the presence of some viscid mucus in the tubes. It is, of course, open to question whether this had anything to do with the cerebral abscesses that were the cause of the patient's death.

Another cause of suppuration within the substance of the brain is direct *injury to the head*, as from a fall or blow. Generally the skull is fractured, and serious symptoms are present from the time of the accident until death releases the patient from his sufferings. Such cases come under the care of the surgeon, and do not require mention from me; but in very exceptional instances the fact that the head has been injured may be overlooked, or the accident may have occurred some time previously, so that its connection with the existing illness is overlooked; and thus the physician may find himself in attendance.

Sir William Gull records the case of a boy, aged sixteen, who was, in 1844, taken into Guy's Hospital for what appeared to be slight fever. After he had become convalescent, he was attacked with cerebral symptoms, and ultimately fell into a comatose state. His friends then for the first time mentioned that three weeks before his admission he had been stunned for a minute or two by falling backward from a cart, so as to strike his head upon the ground. There was neither wound nor bruise, but it seems that he complained of nearly constant pain in the head up to the time of his coming into the hospital. He died about two weeks after the accident: a large abscess was present in the left hemisphere. There was also an abscess in the situation of the sphenoidal sinuses beneath the carious olivary process of the sphenoid bone. Could this have been caused by the fall? and, if so, was it not the cause of the cerebral abscess?

Another case occurred to Dr. Tuke ("*Med. Times and Gaz.*," 1861, i, p. 196). It is that of a man, aged forty-seven, who died after a week's illness with cerebral symptoms. A few months previously he had fallen from a ladder upon his head, without having any severe symptoms immediately afterward. Dr. Tuke believed that the accident was the cause of the cerebral symptoms, but it should be noted that, although the patient had suffered from "chest symptoms" a year previously, there is no mention of the state of the lungs at the autopsy.

One would be the more ready to attribute a cerebral abscess, for which we could find no other cause, to any injury of the skull that might have occurred within a year or two previously, because it appears that there is scarcely a case to be met with in which one is absolutely driven to admit that the abscess is primary and spontaneous. Among seventy-six cases collected by Gull and Sutton for the "System of Medicine," there are, indeed, a few in which no cause was found; but the only one of which it can be said with certainty that the autopsy was complete is the last of the series, that of a man who died in Guy's Hospital in 1863, and whose body was examined by Dr. Wilks. He had been employed at a music hall, and some years before he had had his chest crushed in an accident, but he was not known to have injured his skull. However, he had led an irregular life, and it is not unlikely that he had at some time or other fallen

or struck his head. I believe that there has not since been a single instance in which an abscess of the brain has been found in the post-mortem room of the hospital, and in which the demonstrator of pathology has been unable to trace it to one or other of the causes which I have enumerated.

Varieties.—Cerebral abscess, consecutive to disease of the ear, is about equally frequent in males and in females; of the cases secondary to chronic pulmonary affections the larger proportion seem to occur in the male sex. The great majority of cases of both forms are met with in persons between fifteen and thirty years old.

Abscesses of the brain arising from different causes are to some extent different. Those which result from injury are almost always solitary, and so are those which are secondary to affections of the ear or nose. Out of twenty-seven cases due to aural disease collected by Gull and Sutton, there are only two in which more than one abscess was present; in one the cerebellum contained three abscesses, in the other there was a collection of pus in the cerebellum as well as one in the cerebrum. And in all the twelve cases of the same kind that have recently occurred at Guy's Hospital the abscess was single, with the exception of a case in which there was a second smaller abscess by the side of the principal one. On the other hand, I find the fourteen cases of pyæmia, in which the brain was the seat of suppuration, yield eight in which the abscesses were multiple, and in most of them there were four, five, or more of them in different parts of the cerebral substance. So, again, with those cases which are consecutive to affections of the lungs, in seven out of eleven the abscess was multiple. The presence of numerous centres of suppuration might thus go far toward determining the real nature of an abscess of doubtful origin.

The cerebral abscess caused by disease of the ear or nose is generally ill defined, its wall shreddy and surrounded by softened, cerebral substance; but sometimes it is inclosed in a thick capsule. In pyæmia the abscesses seem to be very rarely circumscribed; but in Dr. Moxon's case, which lasted eighteen months, the limiting membrane was so firm that it could be lifted out of the brain tissue in which it lay. When the affection is secondary to lung disease there are almost always well-marked capsules. In the "*Guy's Hospital Reports*" for 1857, Sir William Gull pointed out that the cyst wall is made up to a great extent of spindle cells. Rindfleisch has since shown that they constitute its middle layer, there being outside them a plane of fibrous tissue, while the cavity is immediately lined by embryonic tissue, the superficial cells of which are opaque and yellow from fatty degeneration. Even in abscesses of recent formation the pus is commonly greenish and viscid, although it has an acid reaction; but in those which have been of long standing it is often quite mucoid, of a bright green color, and alkaline. It may be odorless, or have a nauseous, sickly smell, or (when arising by extension from bone disease with necrosis) be horribly fetid and sloughy. Mixed with the leucocytes there is always a great deal of granular matter and fat, probably derived from the brain tissue, the place of which is occupied by the abscess; but in very old cases one can hardly recognize any pus cells at all; they have undergone complete degeneration, and nothing is left of them but *débris*.

It is only in cases arising from injury that the commencement of the cerebritis can be fixed with sufficient accuracy to enable an opinion to be formed as to the length of time required for the production of a capsule. So far as I can interpret the evidence which has been collected by Lebert and Meyer with regard to this point, it goes to show that by the end of the third week the abscess cavity may be found circumscribed, but that a definite

membranous cyst wall is not found before the sixth or seventh week, and sometimes not until a much longer period has elapsed. Sir William Gull has pointed out that this might become a medico-legal question in some cases of a man dying of cerebral abscess after receiving a blow from another person, if an interval had elapsed so that no direct clinical connection could be traced between the supposed cause and the effect. He cites one instance in which the absence of a limiting membrane was taken as proof that the disease could not have been the result of a severe fall eighteen months previously; and another, in which its presence showed that the suppuration in the brain had not been due to a pyæmia, which might have been set up by an attack of smallpox within the last three or four weeks before death.

An abscess of the brain, when solitary, generally reaches a considerable size before it destroys the patient. It is frequently as large as a hen's egg, and sometimes much larger still. It often reaches close to the surface of the hemisphere, and is said sometimes to break through beneath the pia mater, and set up a rapidly fatal meningitis; of this, however, I have met with no certain example. When it is secondary to disease of the ear with extensive destruction of the bone, and when local adhesions of the membranes have been found, it sometimes happens that pus from an abscess in the brain is discharged externally through the auditory meatus; such cases have received the name of "cerebral otorrhœa." So, again, an abscess set up by mischief in the nose may break through the ethmoid bone. Some writers suppose that it is possible for an abscess of the brain to cause adhesions of the membranes over some part of its surface, and then to eat its way through the skull, and burrow beneath the temporal muscle, or under the aponeurosis of the scalp; but I am very skeptical about such an occurrence; except, indeed, in cases of severe injury to the head, in which the bone is destroyed by an independent inflammatory process. Far more frequently, a collection of pus within the hemisphere penetrates inward toward the lateral ventricle. At Guy's Hospital this has been found in four or five out of the last seven cases in which such an affection has been set up by mischief in the ear; and it is by no means infrequent in cases which are secondary to some pulmonary disease. Sometimes there is merely an extension of the process along the brain substance which intervenes between the abscess and the ventricle; sometimes the one ruptures into the other. In the latter case the pus may be found collected in one of the cornua, into which it had fallen by gravitation, or the whole of both lateral ventricles may be full of pus, their ependyma intensely inflamed, thickened, gray, and velvety; and the morbid action may even spread through the third and fourth ventricles to the subarachnoid spaces of the brain and spinal cord.

Symptoms.—The symptoms of abscess of the brain vary widely in different cases. It has been said that there may be absolutely none, and the affection be accidentally found after the death of the patient from some other cause. Of this, however, I have met with no example, either in books or in our records of post-mortem examinations. But in the large majority of cases, symptoms are present during a period much shorter than that through which the mischief in the brain must have been going on; in other words, the disease is commonly altogether latent during a certain part of its course. And in cases of general pyæmia, the occurrence of suppuration in the brain is not infrequently masked under the delirium and stupor which may accompany any form of severe blood poisoning.

The earliest symptom of cerebral abscess is, as a rule, *pain.* This varies greatly in severity; sometimes it is of the most agonizing character. Gull and Sutton speak of one patient as continuously holding his head

with both his hands, and of another as walking about with his hands pressed against one side of his head, and calling out, "Oh, my head! Oh, my head!" Another could not help screaming, and, although perfectly sensible, would tear and bite anybody or anything near him, at the same time expressing contrition for what he was doing. The pain is generally continuous; but it is sometimes intermittent, especially at first. Anstie mentions the case of a boy who for three months complained of no symptom whatever, except of a pain which came on in attacks very closely resembling those of a migraine, not oftener than once in ten days or a fortnight, and lasting for some hours at a time, nearly always ending in vomiting, and disappearing after sleep. In some instances the seat of the pain corresponds closely with that of the abscess; Gull and Sutton mention the case of a boy who had almost constantly a burning pain over the front and right side of the head, and in whom the disease was in the anterior lobe of the right hemisphere. But they go on to speak of a patient who had an abscess in the cerebellum with pain in the forehead, and of another who complained of the left side of his head, but had an abscess on the right middle lobe.

Much less frequently, an *epileptiform seizure* forms the starting point of the symptoms; and such seizures may be repeated at intervals for a considerable period, before any further sign of illness manifests itself. Or *rigors* may occur, sometimes with such regularity that the case might be mistaken for one of ague. Or there may be *vomiting* from time to time, without any apparent cause. Or the first thing noticed may be a dullness of expression, a change of disposition from cheerful to morose or melancholy, a disinclination to speak, an apparent loss of memory, or an inability to sleep. The pupils are sometimes sluggish, and they may be unequal in size; the optic discs have been found congested and œdematous. Gull lays stress on rapidly increasing emaciation as having been a principal symptom in some cases. The pulse is sometimes slow; Huguenin relates a case in which it fell to fifteen and even to ten in the minute.

Locality.—In the symptoms hitherto mentioned there has been little or nothing to indicate that one part of the encephalon rather than another is the seat of disease. But in exceptional cases "localizing" symptoms are not wanting. Aphasia may be present, which, of course, shows that the abscess is in the back part of the left frontal lobe. Much more frequent is hemiplegia, partial or complete. This indicates that the part affected is near the fissure of Rolando; or that the inflammatory process extends inward, so as to involve the outer edge of the corpus striatum. A paralysis of the seventh or of the ninth nerve, the arm and leg being free, is said by Huguenin to show that the mischief is seated in the anterior lobe. I have already cited, at p. 552, a case of Hitzig's in which spasms confined to certain muscles were set up by a small abscess limited to a small part of the cortex. But, on the other hand, Sir William Gull has related in detail the case of a gentleman who on many occasions had a sudden convulsive affection of his right arm—so violent that he had to support himself by holding on to the table with his other hand, and yet so devoid of pain that he was amused by it—but in whom the abscess—a large one—was in the occipital lobe.

It seems to be very doubtful whether there are any "localizing" symptoms peculiar to abscess of the cerebellum. Huguenin says that the pain is not only referred to the occiput, but often extends down the back of the neck. He also states that the pupils of both eyes are commonly dilated, that vomiting is peculiarly severe, and that the gait is sometimes unsteady, like that of a person affected with locomotor ataxy. In one case which occurred at Guy's Hospital I find it noted that the patient was so feeble as

to be hardly able to sit up in bed; but in that instance (as in many other cases of cerebellar abscess) the lateral ventricles of the cerebrum contained a large excess of fluid, a result, in all probability, of pressure upon the veins of Galen.

Mode of Death.—None of the symptoms which have been described are such as would suggest to an unskilled observer that the patient's life is in any imminent danger. But after they have been present for some days or even for several weeks—or, sometimes, without any one of them having existed to indicate that the brain is diseased—he falls, often suddenly, into a state which is obviously most alarming. Violent delirium may set in, followed by stupor and coma; or a severe epileptiform fit may occur, after which he may never regain his consciousness. In 1876, a girl, aged eighteen, was admitted into the clinical ward one afternoon at five o'clock. She had for six days been suffering from sickness and diarrhœa, with severe headache, so that she was said by the medical man who attended her to have typhoid fever. She then spoke rationally and answered the questions that were put to her, but seemed rather strange in her manner. At eight o'clock the same evening she suddenly made a great noise; the house physician was hastily summoned, and found her partially insensible, but capable of being roused so far as to say that she was going to die. She seemed to have loss of power in her left limbs. An hour later, she all at once ceased to breathe. Artificial respiration was kept up, and the heart continued for some little time to beat rapidly, but it soon slackened, and in ten minutes she was dead. I found four or five abscesses in the posterior and middle lobes of the right hemisphere of the brain.

In the case just referred to—and in several other cases in which the fatal termination was equally sudden—there was no extension of the inflammatory process toward the lateral ventricle. I am inclined to be skeptical as to the possibility of diagnosing that occurrence. Huguenin thinks that the rupture of an abscess into the ventricle may sometimes be recognized by the supervention of spasms in both sides of the face, or in both legs, without loss of consciousness; and he gives cases in which these symptoms were present. The point, however, is one of no practical importance.

The terminal stage of a cerebral abscess is seldom of long duration. But it may last for a week, and, in exceptional cases, longer still; during which time the patient is alternately delirious and in a state of stupor, and commonly passes all his evacuations under him in bed. Sometimes there is a transient recovery from such symptoms, only to be followed by their return, and by a fatal termination of the case.

Diagnosis.—That the diagnosis of abscess of the brain is often beset with difficulties must be sufficiently apparent from what has been said of its symptoms and of the conditions under which it occurs. More than once—when speaking of ague, of enteric fever, and of neuralgia—I have had to point out how necessary it is to bear in mind cerebral abscess as a possible explanation of cases that appear doubtful. As between abscess and other organic affections of the encephalon, the only positive criterion is the presence of shivering; and that symptom is often absent.

Thus, in practice, we are able to detect the disease in question only when some one of its recognized causes is known to be in operation. And since it seldom, if ever, arises spontaneously, this would generally give us a good opportunity of diagnosis, but for the fact that both the patient and his friends too often ignore, or even explicitly deny, the existence of these several causes, such as otorrhœa, deafness, and bronchial irritation. Moreover, each of the affections that may set up suppuration in the brain is liable to give rise to other forms of cerebral disease. Thus injury is not infre-

quently followed by meningeal apoplexy, and by certain diffused inflammatory processes, which will be described in the next chapter. Again, we can seldom speak with certainty of cerebral abscess as being consecutive to any pulmonary affection, unless we are in a position to exclude the possibility of its being phthisis; for that disease is sometimes accompanied by solitary tubercle of the brain, and very often sets up tubercular meningitis, which is itself a very insidious malady, and attended with the most varied symptoms. Lastly, disease of the ear may either be the starting point of a general meningitis, or cause thrombosis of the lateral sinus, which for some unexplained reason is now and then accompanied by well-marked signs of cerebral disturbance. To this affection I shall again refer when I am speaking of inflammation of the membranes. And I will leave until then the little that can be said with regard to the *treatment* of abscess of the brain.

CHRONIC AFFECTIONS OF THE BRAIN DUE TO DIFFUSED ORGANIC LESIONS.

Chronic Diffused Inflammation; ITS GREAT RARITY—HYPERTROPHY OF THE BRAIN.
Hydrocephalus: IN CHILDREN, AND IN ADULTS.
General Paralysis of the Insane—OTHER FORMS OF CEREBRAL ATROPHY.

In 1860, Griesinger, in a paper in the "*Archiv der Heilkunde*," drew attention to the importance of distinguishing, among organic affections of the brain, those which are limited to some particular region (*herdartig*) from those which are diffused. Of the diseases which he placed in the latter class, some, belonging to the membranes rather than to the cerebral substance, have already been described. But there remain several others, all of which are not indeed very closely related to one another, but which yet fall somewhat naturally into a single group; and these I propose to discuss in succession in the present chapter. As might be expected, they are mostly characterized by an absence of those symptoms which, at p. 546, have been spoken of as "localizing." Indeed, Griesinger briefly, but with tolerable accuracy, sums up their effects as consisting of "giddiness, diffused headache, delirium, and mental failure—from slight dullness of intelligence up to imbecility—drowsiness and stupor, tremors and involuntary quiverings of the muscles, vomiting, and, lastly, the more positive indications of augmented pressure within the cranial cavity, slowing of the pulse and of the respiration."

In the diagnosis, two points have to be borne in mind. (1) That local affections may produce like symptoms, if they happen to take up a large space, and especially if they are seated in a part of the brain which is not yet known to possess a special function; (2) that they are very apt to be confounded with certain neuroses, and particularly with the various forms of insanity in which no organic change is present.

One peculiarity of the chronic diffused affections of the brain is that many of them occur at particular periods of life with especial frequency, or that their symptoms vary to a greater or less extent with the age of the patient. And, as the order in which they are to be taken is otherwise unimportant, I will, for the convenience of clinical comparison, arrange them in an ascending series, beginning with those which are seen in children, and leaving to the last those which are most common in middle-aged and in old persons. We shall then have—

1. Chronic diffused cerebritis.
2. Hypertrophy of the brain.
3. Hydrocephalus of early childhood.
4. Hydrocephalus of adult life.
5. Atrophy of the brain, including—
   - *α*. General paralysis of the insane, or dementia paralytica.
   - *β*. Atrophy from old age, alcohol, lead, or perhaps from other causes.

1. CHRONIC DIFFUSED CEREBRITIS.—If we consider how important a

part in the pathology of the spinal cord is played by diffused chronic inflammatory changes, we shall surely find it an extraordinary circumstance that similar lesions are hardly known to occur in the brain; certainly there is no form of disease which comes under that description, and which is met with commonly and indifferently among persons of both sexes and at various ages. I have, however, made autopsies in two cases in which such a lesion was the cause of death.

One of them occurred in the practice of Dr. Wilks, who has already recorded it in the "*Guy's Hospital Reports*" (Series iii, vol. xxii, p. 22). A girl, aged fourteen, was admitted, in 1876, for a general failure of mind or body, which had been coming on for about a year. The earliest symptom was said to have been an attack of weakness and loss of sight, which came on, one day, while she was out on an errand. Soon afterward she had two epileptiform seizures, which began with a scream. She generally lay quiet, with a vacant stare, making no complaints, but smiling when spoken to. She could evidently see, and the optic discs were normal, but the pupils were dilated. If asked what her name was, or any other simple question, she answered sensibly, but remarkably slowly. When food was put into her mouth she would cease to chew without swallowing it, so that it lay there until removed by the nurse. She often vomited. She was unable to stand, and had very little power over her legs; even her arms were moved but slowly and feebly. However, a month before her death she tried to strangle herself, and was found with a towel tied round her neck. Her skin appeared to be sensitive. Toward the last she lay with her eyes open, looking forward, but making no attempt to speak beyond a slight motion of the lips. Her temperature ranged from a little below to a little above normal. Before death her extremities became remarkably livid; on the feet large bullæ made their appearance, which looked as though gangrene were beginning.

At the autopsy a deep purple discoloration was seen extending to above the ankles, the cuticle was detached from the rete mucosum, and the deeper tissues were all infiltrated with blood. The calvaria was thick and heavy and very asymmetrical. The membranes were a little opaque. There was an excess of fluid at the base. But over the convolutions the pia mater was everywhere abnormally adherent, so that in attempting to strip it off one peeled away a thin, granular-looking layer of the cortex, leaving a roughened surface. The brain was small, weighing only thirty-four ounces. Its cortex was not obviously wasted. Its substance generally was tough. The boundary line between the gray and the white matter was everywhere well defined. The ventricles were very considerably dilated, but their ependyma was not granular, except in the fourth ventricle.

The other case, one of much less clinical interest, occurred in 1878, in a child eighteen months old, a patient of Dr. Pavy's. Its illness was said to have begun with a fit at the age of four months, and from that time it had been subject, especially at night, to seizures, in which it would fight for its breath and appear to be choking, but would afterward lie insensible for three days at a time, taking very little food, and twitching at the corners of the mouth. Fifteen days before its death it had a fit more severe than any previous one. Eleven days later it was again attacked, and on the following day it was brought into the hospital. It then lay comatose on its left side, with the corners of the mouth drawn down and constantly twitching; when it was moved over, its whole body would become rigid, and the twitching at its mouth was more marked. It appeared to have lost sensation in the left side. The temperature, at first 102.4°, rose to 103.8° before death, and the pulse became scarcely perceptible.

At the autopsy I found the child to be wasted, with a pigeon-shaped

chest and rickety enlargement of the ends of the ribs and of other bones. The brain was pale on the surface. Its membranes looked healthy; they were not at all thickened or opaque, but very thin and delicate, as is usual at an early age. On attempting to strip the pia mater from the convolutions, however, it was at once found to be abnormally adherent, so that a thin, uniform, superficial layer of the cortex peeled off with it, leaving a rough, granular surface behind. The substance of the hemispheres also was indurated, cutting firmly, and giving sections with sharp, resistant edges, but the most striking appearance of all was a discoloration of the white matter of the hemispheres, which had a yellowish tint. This was more obvious on the right side than on the left, and in the anterior and lateral parts of the hemispheres as compared with the posterior parts. It was also more marked in the prolongations of the white matter into the bases of the convolutions than toward the centre of the brain, and in some of the convolutions the limit between the white and the gray substances was ill defined. The gray matter itself looked natural. The ventricles were not dilated. The basal ganglia, the pons, the bulb, and the cerebellum were all of the natural soft consistence.

Portions of the affected tissues from each of these cases were examined, after the most approved methods, by Dr. Savage, of Bethlem Hospital, and also by Dr. Frederick Taylor, but no decided histological changes could be detected in them, except possibly a slight excess in the cellular elements of the interstitial neuroglia.

2. Hypertrophy of the Brain.—It has long been known that in certain cases in which the head appears unduly large, so that the presence of hydrocephalus is suspected, a solid brain really fills the whole interior of the skull. Instances of this kind were recorded in the early part of the present century by several French observers, among whom were Laennec and Andral, and many other writers have since alluded to the affection, which has generally received the name of hypertrophy of the brain; but its nature and its relations to other diseases still remain among the most obscure questions in pathology. Virchow several years ago suggested that it consisted in an overgrowth of the neuroglia rather than of the nervous elements of the hemispheres. Sir William Jenner, not long afterward, maintained that it was due to an "albuminoid infiltration" of the tissues, like that which he believed to cause enlargement of the liver and spleen in rickets; he regarded it, in fact, as one of the minor effects of that diathetic disorder. But it appears to be certain that the subjects of cerebral hypertrophy are not always rachitic. On the strength of a case in which Magnan is said to have minutely examined the structure of a brain so affected and to have found it perfectly normal, D'Espine and Picot, with other writers, continue to describe the disease as a true overgrowth of all the various elements of the cerebral substance. This conclusion, however, I am unable to accept, not only because of its inherent improbability, but still more on account of there being other lesions of the brain (*e.g.* "diffused chronic cerebritis" and "yellow softening") which are easily recognized by the naked eye, but in which the microscope equally fails to reveal marked histological changes. Indeed, it is worthy of notice that in each of the two cases of which Andral has given full descriptions, the gray substance of the convolutions is said to have been undistinguishable from the white by its color, while the white substance itself resembled white of egg hardened by boiling, and in some parts was even of the consistence of cartilage. How can such a condition be distinguished from sclerosis? It seems to me that no reliance can be placed on any cases of supposed hypertrophy of the brain which are recorded at a time when the knowledge of the pathology

of the organ was so meagre in comparison even with that which we now possess.

I have therefore been very glad to avail myself of the kindness of Dr. Fletcher Beach, of the Darenth Asylum for Idiots and Imbecile Children, who has given me some notes of two among six cases of this affection that have come under his notice. He has found that the white matter showed "a uniform granular appearance under the microscope, with nerve cells scattered sparsely throughout. There were a number of leucocytes present. . . The increase in size was evidently due to the large amount of granular matter."

In each instance the size of the head was a conspicuous feature during life. In one, a boy, aged sixteen, it measured twenty inches in circumference, in the other, a boy, aged ten, the measurement was twenty-two inches. Its form was, in each instance, square, not, as in hydrocephalus, globular. Other points of distinction from that affection on which Dr. Beach lays stress are that the enlargement is more marked just above the superciliary ridges than at the temples, and that the fontanelle is often depressed instead of being full and elastic. This last character seems hardly to consist with the accounts which have been given of the dura mater bulging as soon as the calvaria was opened, and of flattening of the convolutions. Indeed, although the brain in the older of Dr. Beach's patients weighed no less than sixty-two ounces, he speaks of the quantity of subarachnoid fluid over the convex surface being increased, and of the ventricles being rather dilated.

None of Dr. Beach's patients presented any signs of rickets, but, as he himself remarks, it is possible that they may have been rachitic at an earlier period, since they were, with one exception, above the age of ten years when they came under his observation, and since they were in fairly good bodily health. It appears to be still doubtful whether or not the present category will hereafter be found to include all cases of rickety enlargement of the head without hydrocephalus. Another affection, the relations of which to hypertrophy of the brain have still to be determined, is one which is liable to be mistaken for tubercular meningitis (*vide infra*, p. 607).

Clinically, it would seem that hypertrophy of the brain is characterized by a more or less marked deficiency of intelligence, drowsiness, headache, and a liability to epileptiform convulsions; the gait is sometimes slow and tottering, the weight of the head may cause it to hang forward, or may even from time to time throw the patient down upon his face when he is walking. Under favorable circumstances life may be prolonged for years. The disease ends either by some intercurrent pulmonary affection, or by gradual exhaustion, or by the supervention of acute symptoms and coma which, in one of Dr. Beach's cases, were due to acute suppurative meningitis.

With regard to treatment nothing is as yet known.

3. Hydrocephalus of Early Childhood.—Among the diseases to which infants and young children are liable there is one which consists in a distention of the cerebral ventricles with fluid. This is generally called "chronic hydrocephalus," or "water on the brain." The epithet "chronic," although until lately needed to distinguish the affection in question from that which was known as "acute hydrocephalus," is unnecessary now that the latter is always spoken of by its proper name of tubercular meningitis.

Hydrocephalus is not infrequently *congenital*. At birth, the fœtal head may be so big as to prevent its passage through the maternal pelvis until it has burst or has been perforated by the instruments of the accoucheur, with a necessarily fatal result. Or, the enlargement being less considerable,

expulsion may at length take place, and the child may live for a shorter or a longer time. Or it may be noticed that the head of the newly-born infant is softer and more pulpy than natural, there being no obvious increase of size until a few weeks have passed. Or, lastly, no one may observe any thing is amiss with the child until it is some months old. Among twenty-six cases collected by Dr. Dickinson ("*Lancet*," ii, 1870) there were four in which the disease was said to have been present from birth, sixteen in which it was discovered within the first six months, six in which it was not noticed until between the sixth and the twenty-sixth month. One must bear in mind that these figures express, not the relative frequency of the congenital and acquired forms of the disease (for the list probably includes no infants born dead, or dying within the first week or two), but the extent to which a congenital origin is traceable among cases of hydrocephalus brought under medical care later on in childhood. It is evidently impossible to determine in how many of them the hydrocephalus really began *in utero;* but in all probability the brain was healthy at the time of birth, in the large majority. We shall afterward see that the affection sometimes arises at a more advanced period of life than in any of Dr. Dickinson's cases.

Thus it is clear that great practical inconvenience must result from the attempt to describe separately the congenital and the acquired forms of hydrocephalus, as is done by Huguenin in Ziemssen's "Handbuch," nor does there appear to be any difference in their pathology such as would warrant one's drawing a sharp division line between them. There is, indeed, one supposed cause of "water on the brain" which can give rise to the disease only in the fœtus, and during early infancy, namely, a deficiency of resistance on the part of the parietes of the head, allowing the normal ventricular fluid to accumulate in excessive quantity, and to distend both the ventricles themselves and the parts outside them. Thus, Huguenin says, it is very common for rachitic children, if they are attacked with whooping-cough and bronchitis, to get a form of hydrocephalus, attended with considerable enlargement of the head, and with widening of the intervals between the bones, but in which there is no flattening of any of the cerebral structures, nor any morbid change in the brain except dilatation of the cervical ventricles. But I should doubt whether Dr. Dickinson and other observers, who have insisted on the great frequency with which water on the brain occurs in those who are the subjects of rickets, would be prepared to admit the validity of these distinctions. The occasional association of hydrocephalus with congenital syphilis is supposed by Dr. Dickinson to depend on a defective growth of the cranial bones, resulting from the constitutional malady.

What little beside is known as to the ætiology of hydrocephalus tends, for the most part, to support the view that it is of an *inflammatory* origin. Even when it arises in the fœtus some writers have attributed it to a blow or fall affecting the abdomen of the mother. Gölis, however, adduced some facts to show that drunkenness on the part of either of the parents may cause it. A few striking instances of its occurrence in several children of the same father and mother have been recorded. Frank saw in one family six, in another seven cases. Gölis reported the case of a woman who aborted six times in succession with dead hydrocephalic fœtuses at the sixth month, and bore three living children, two of whom died of the disease when eighteen months and three years old respectively.

When it begins during extra-uterine life, hydrocephalus appears sometimes to be directly traceable to an injury which has set up inflammatory changes in the brain. West records the case of a little girl who, some months before her head began to enlarge, had fallen out of the arms of her

nurse, and had subsequently been convulsed and comatose for some hours. In that instance the only structure that is said to have been thickened was the ependyma. But in many cases the membranes at the base of the brain are found opaque and adherent. There has been much difference of opinion as to whether the disease often arises out of an acute meningitis. We shall see that in the epidemic form of that affection such a termination is not infrequent (cf. p. 615); but I do not think that this is the case so far as the simple and the tuberculous forms are concerned. Huguenin, however, speaks most positively of having seen hydrocephalus begin with acute symptoms exactly like those of infantile lepto-meningitis, but which, instead of terminating fatally, have subsided and passed into those of a chronic cerebral affection, attended with great enlargement of the head. He says that in such instances he has found the pia mater at the base of the brain thickened and opaque, and the choroid plexuses also showing traces of a former inflammatory change. But it is worthy of notice that in a case of this kind related in full detail by Rilliet and Barthez, in which the early active symptoms lasted six days, it is expressly stated that the ventricular fluid contained only a trace of albumen, that the ependyma was normal in appearance, and that there were no adhesions of the pia mater at the base. On the other hand, it must not be forgotten that such adhesions, when they are present, may have been the result of a *chronic* meningitis.

It has been thought by Huguenin and others that in many cases of hydrocephalus the physical and chemical properties of the fluid enable one easily to determine whether the morbid process was of a passive or of an inflammatory origin. The normal cerebro-spinal fluid is well known to have a very low specific gravity and to contain scarcely a trace of albumen. Now, in some cases of hydrocephalus the fluid has possessed similar properties; and according to C. Schmidt, the proportion in it of potass salts to soda salts, and that of phosphates to chlorides, have differed altogether from those which are met with in ordinary serum, and even in fluid derived from the membranes on the surface of the brain, a fact which is cited by Vogel as proving that the hydrocephalic fluid is a specific secretion of the choroid plexuses. But there are other cases in which the fluid has been of higher specific gravity, and has contained from 3 to 11.5 parts of albumen in 1000. Huguenin takes this as proof that there has been an inflammatory change in the plexuses or in the ependyma; in his opinion such a conclusion is justified by the presence of albumen in any proportion above 2.5 parts in 1000. He, of course, recognizes the fact that inflammation is very likely to come on secondarily in cases in which at first there was a mere accumulation of the normal ventricular fluid; something of this kind is seen when the operation of tapping is several times repeated, the quantity of albumen in the fluid being greater on each successive occasion. But what he does not seem to be prepared to admit is that in cases which are from the first of an inflammatory origin, the fluid may be almost free from albumen. I have notes of no less than four instances of this kind which I have observed in the post-mortem room. The patients were all adults, and there was no reason to believe that they had had hydrocephalus for more than a few months. Their brain symptoms, at any rate, were of quite recent development, and in one instance were distinctly traceable to an injury.

There is, indeed, a view which would account on mechanical principles for the presence of a normal cerebro-spinal fluid even when inflammation had been present; but I doubt whether it is tenable. Several pathologists have shown that there is sometimes closure by adhesions, either of the foramen* at the lower angle of the fourth ventricle, or of the aqueduct of

* See Cruveilhier, tom. iii, p. 385, and Hilton, on "Rest and Pain," 3d ed., p. 23, and figs. 1, 2, 8, and 9.

Sylvius. And Hilton propounded the doctrine that the obliteration of these channels is often the cause of hydrocephalus, by preventing the outflow of the secretion of the choroid plexuses into the subarachnoid space of the cord, which ought normally to occur whenever an increase in the physiological activity of the brain leads to an augmentation of its blood supply. But it is difficult to see why the fluid should continue to be poured out under the increased pressure which must necessarily result, and which certainly is present in most cases of hydrocephalus. And as the adhesions themselves are the results of a more or less widely diffused meningitis, such as is often attended with an inflammatory change in the ependyma, it would seem more reasonable to regard this as the cause of the effusion than to adopt Hilton's theory. It is well known that in one and the same case the various serous membranes may pour out fluids of very different specific gravity; and I think it is not improbable that the lining of the ventricles of the brain may continue to secrete a fluid containing scarcely any albumen, even when it is affected with a low degree of inflammation. But the question requires further study, and can only be settled by careful observations as to the state of the openings into the fourth ventricle in a series of cases in which the physical and chemical properties of the fluid are also accurately determined.

The quantity of fluid in cases of hydrocephalus of long standing is sometimes very great; six, eight, twenty, even twenty-seven pints have been measured after death in different cases. The hemispheres are then transformed into a thin shell, which, perhaps, is not more than a line or two in thickness, so that it is difficult to understand how they could have retained any of their functions. The distinction between the white and the gray matter is lost; and the sulci, if visible at all, appear only as shallow grooves beneath the pia mater. The nerve cells are said to be more or less completely atrophied and destroyed; but accurate histological details seem to be still a desideratum. The corpora striata and the thalami are flattened and very broad, and so are the crura, the optic tracts, the pons, and all the structures at the base. The weight of the brain is in most cases much reduced; but Huguenin says that it has sometimes been found normal or even excessive. Some of the more delicate structures, such as the septum lucidum and the soft commissure, are defective or absent, probably in consequence of the stretching to which they have been subjected. The foramen of Monro, and the aqueduct between the third and fourth ventricles are generally widely dilated, the latter being sometimes large enough to receive a pencil. The ependyma is generally thick, tougher than natural, and of an opaque white or gray color. It often contains a large number of amyloid bodies. Its free surface is covered with granulations, or with a number of translucent beads which give it an appearance that has been aptly compared by Dr. Moxon with the leaf of an ice plant.

Hitherto I have assumed that the fluid is contained within the ventricles. Most pathologists, however, describe under the name of *Hydrocephalus externus* a separate variety of the disease, in which the seat of the effusion is said to be the subdural or arachnoid space. Now, there is no doubt that this may be the case where there is an extreme degree of malformation of the brain, as in some microcephalic or anencephalous fœtuses, but I am much disposed to agree with Dr. Wilks, who has always expressed an utter skepticism as to the occurrence of external hydrocephalus, such as is supposed to be clinically undistinguishable from the ordinary form. The classical example which is quoted by Huguenin and other writers, is that of the young man, James Cardinal, recorded by Bright. The total quantity of fluid in this case was seven or eight pints, and all of it, with the exception of one pint, lay beneath the dura mater at the time of the

autopsy. There was, however, a hole in the corpus callosum, and Bright himself supposed that the ventricles were the original seat of the effusion, although he seems to have imagined that the fluid increased in quantity after it had escaped from them, so as gradually to bring the hemispheres down to the base of the skull, but Dr. Wilks has suggested that the rupture through the corpus callosum did not take place until just before death, and though this way of interpreting the facts is not free from difficulties, I am, on the whole, inclined to adopt it. It is at any rate evident that the case cannot be fairly cited as an instance of a special form of hydrocephalus.

I must not omit to mention that a congenital hydrocephalus is often associated with malformations of other parts as well as of the brain itself. I have two or three times seen it in combination with spina bifida, and in one case the central canal of the cord was also greatly dilated. Again, as I have already remarked, a certain degree of dilatation of the lateral ventricles is commonly found in cases of cerebral tumor. This is seldom a matter of clinical interest, but sometimes when the cranium becomes greatly enlarged the disease has been supposed to be an ordinary hydrocephalus, until at the autopsy the presence of a new growth has been brought to light. A case of this kind is recorded by Huguenin; the patient was a girl, aged six, whose head measured twenty-three inches in circumference. A point of interest is that during the operation of paracentesis an attempt was made to measure the pressure exerted by the fluid, which was found to equal that of a column of mercury an inch and a half high. The fluid contained 3.5 parts of albumen in 1000, so that it could not be supposed to be a mere accumulation of the normal secretion, the result of pressure upon the veins or upon the subarachnoid openings.

The general result of this discussion as to the pathology of hydrocephalus is to lead to the conclusion that the congenital and acquired forms of the disease which are seen in early childhood are alike dependent, in most cases, upon a chronic change, which is probably always of an inflammatory nature, in the ependyma and choroid plexuses. There can be no doubt that a deficiency of resistance on the part of the parietes of the head is often an auxiliary cause, but it does not appear that any sharp line of demarcation can be drawn between cases arising in this way and those which are due to the ependymal affection. Now, as we shall hereafter find, older children and adults are liable to a disease which from a pathological point of view is almost precisely similar, and which, therefore, might seem to have a claim to be described in common with that which occurs at an early age. Clinically, however, this is altogether impracticable. In its symptoms and in its course the hydrocephalus which occurs in those whose cranial bones are firmly united together is entirely distinct from that which is observed in infants and young children; the other diseases with which they may be confounded, and from which they have respectively to be diagnosed, are different, and there are other points of difference in regard to treatment.

Symptoms.—In fact, in the majority of cases enlargement of the head is the first, as well as the principal, symptom of the hydrocephalus of early childhood. Among forty-five cases collected by West there were twelve in which the frequent repetition of fits first drew attention to the existence of cerebral mischief, four in which it began with some other indication of brain disturbance, and six in which it arose out of an acute attack; in the remaining twenty-three cases no definite cerebral symptom preceded the discovery that the head was increasing in size, or that the fontanelles and sutures were unduly wide, although it must be added that the child had often been noticed for some little time to be failing in strength and losing flesh, or to be otherwise out of health.

The effect of the disease upon the cranial bones is to separate them more and more widely, except at the base. Trousseau aptly compares the change in their relations to one another with the falling back of the petals of an opening flower. The frontal bone rises vertically above the eyebrows, or even overhangs them; the temporal and the parietal bones arch outward, so as to hide the ears when the scalp is viewed from above; the occipital bone extends backward almost horizontally. Thus the head may acquire an enormous circumference. Dr. Dickinson had under his care a child, nine months old, in whom it measured thirty-one inches, and instances in which it ultimately reached forty and even fifty-two inches are cited by Trousseau. A strange contrast is afforded by the shape of the face, which appears unnaturally small, with angular features and a sharp chin. The base of the skull, too, is generally narrow, with shallow fossæ, but the presence of the fluid within the anterior lobes of the brain affects the orbital plates, so that they become convex downward instead of concave, and thus arises a very important character of the disease in an abnormal position of each eyeball; a large part of the iris, and even of the pupil, is hidden by the lower lid, while the sclerotic above the cornea is exposed to view.

The whole of the vertex of the head forms an open area, which may be said to correspond with the natural fontanelles, expanded so as to meet together between the parietal bones with a breadth of some inches, and dividing the two halves of the parietal bone by a cleft that reaches nearly to the root of the nose. There is, however, always a work of ossification going on which tends to cover the brain in, provided that the process of expansion is not the more active of the two. In examining the crania of hydrocephalic children, whose death has occurred while the disease was in progress, I have found that the original outlines of the bones were still plainly visible, but that they were surrounded by broad zones of new osseous material, marked by radiating lines which showed the direction of their growth. In some cases the closure of the head is greatly advanced by the formation of a large number of *ossa triquetra*. The date at which it is completed varies widely in different individuals. In Dr. Bright's patient, Cardinal, the anterior fontanelle is said to have been finally ossified at about the twenty-seventh year. Sometimes irregularities seem to occur in the union of the several bones. At least this has been assumed to be the cause of a want of symmetry in the cranium which has sometimes been noticed. As a rule, the shape of a hydrocephalic skull approaches that of a sphere. It often, however, appears rather quadrilateral in consequence of the very marked character of the frontal, the parietal, and the occipital prominences. The bones themselves are generally very thin and have no diploë; they may even be transparent. But where life had been prolonged past middle age they have sometimes been found greatly thickened; the museum of Guy's Hospital contains a specimen of extensive ossification of the dura mater, which was taken from a case of this kind.

The separation of the bones of the head by an accumulation of fluid within the cranial cavity is not altogether confined to very young children. There is a classical case, recorded by Matthew Baillie, of a boy, aged seven, whose skull had appeared to be firmly united, but in whom at the time of death there was an interval of three-quarters of an inch at the sagittal, and one of half an inch at the coronal suture. Dr. Dickinson says that the same thing has been known to occur in adults. I believe, however, that even in children it is exceedingly infrequent where the bones have once become interlocked.

In well-marked cases of hydrocephalus, it is easy to transmit a wave of fluctuation from one hand to the other across the distended scalp. The skin and the subcutaneous tissues are exceedingly thin, and the ramifica-

tions of large veins are often plainly visible through the scanty hair. The head may appear obviously translucent when a candle is held on the opposite side of it. In Cardinal's case this is said to have been observed when the sun was shining behind him, and up to the time when he was fourteen years old.

Some German observers state they have elicited a *bruit de pôt fêlé* on percussing over a hydrocephalic head, especially when the mouth was opened; it is supposed to have been due to resonance of the air in the vault of the pharynx. Auscultation has also been brought to bear upon the diagnosis of the disease. It is well known that on listening over the fontanelle of a healthy child one hears a systolic murmur; this has been supposed to arise in the veins that open into the longitudinal sinus, as a consequence of their being compressed at the moment when the cerebral arteries become distended with blood. Now, Rilliet and Barthez maintained that in hydrocephalus this murmur is not to be discovered; but more recent observers have shown that it may often be plainly heard, although they admit that it is absent in acute meningitis or where effusion is going on very rapidly. The contradictory statements which have been made with regard to it appear to be in part due to the fact that even under normal conditions it is only audible within certain limits of age (between the eighteenth week and the fourth year).

One consequence of hydrocephalus is a difficulty in keeping the head supported. An infant may be unable to raise it from the pillow, or, if placed in a sitting posture, may let it roll backward and forward, as if it had no power in its cervical muscles. An older child perhaps sits with the head resting on the table, or walks with it carried between his hands, "just as a milkmaid steadies her pail." This, however, is simply a mechanical effect of the weight of the fluid. The extent to which the vital functions of the brain are interfered with varies widely in different cases. In those rare instances in which a skull already fully developed expands under the influence of the disease (as in Baillie's patient, already referred to), headache, stupor, and paralysis of all the limbs may be present for several months before any enlargement is noticed. Such cases, in fact, are to be regarded as transitional between the hydrocephalus of early childhood and that of adult life. But in young children, in whom the cranial cavity readily yields, it is often surprising how few symptoms of cerebral disturbance can be made out, even when the quantity of fluid is already large. Headache is often present; even very young children often show that they are in pain, by restlessness and a sad, whining cry. Vomiting is of frequent occurrence, and, like the headache, it is apt to be excited by movements of the head, especially when the child assumes the erect posture.

The sight sometimes remains good throughout the whole course of the disease. But in many instances there is blindness almost from the commencement. This, however, is due to changes in the optic discs, which are commonly white and atrophied. It is probable that such a condition sometimes occurs primarily as the result of the pressure of the ventricular effusion upon the optic tracts; but in many instances it is secondary to "choking" or to neuritis. Huguenin examined three infants with congenital hydrocephalus between the twentieth and the thirty-fifth day after birth, and in each case found the discs reddened and swollen. The hearing is very seldom impaired; as Dr. Dickinson remarks, a young child who is perfectly blind may at once recognize its mother by her voice. The smell is said to be sometimes altogether absent, and the olfactory lobes atrophied. I suppose that in such cases there would generally be other malformations of the nervous centres as well. The taste seems often to be perverted; the appetite, at any rate, is voracious and indiscriminate. Impairment of the

muscular power of the limbs may be present in all degrees from a slight failure of co-ordination in standing or walking, up to a total paralysis. There is sometimes a more or less complete hemiplegia or paraplegia, but such symptoms probably depend in most cases upon some additional local lesion of the brain or cord. Spasmodic affections of various kinds occur; nystagmus especially, but also partial clonic or tonic spasms of the limbs, and even general epileptiform convulsions, the latter being very apt to be brought on by any accidental circumstance which shakes the head or gives it a mechanical shock. The legs and arms are often stunted in their growth, and their muscles very small. Huguenin mentions that in one case in which the lower limbs were for a time contracted, but afterward became relaxed, the muscles were found to have lost their contractility to faradic currents; this, of course, indicated that there was some secondary or concomitant affection of the gray matter of the cord.

In some cases of congenital hydrocephalus there is no advance of intelligence from the time of birth onward; they generally terminate fatally at an early age. In other instances the brain develops to a greater or less extent, but very slowly, taking, perhaps, ten times as long as under normal circumstances. During childhood such patients would generally be classed as imbeciles. Cardinal, however, went to school at the age of six, and he soon learned to read well and to write a little; but he was obliged to give up the latter accomplishment, as stooping caused a pain in the head. When he was twenty-nine years old his mental faculties are said to have been "very fair. His memory was tolerable, but it did not retain dates and periods of time; and it was stated of him that he had never been known to dream. There was something childish and irritable in his manner, and he was easily provoked. He was stated not to have sexual desire, but he was fond of society and affectionate to his mother. His voice was not manly, but feeble and somewhat hoarse." He died about three months later, having become exceedingly feeble, with a protracted cold, febrile symptoms, diarrhœa, and loss of appetite. Perhaps, if the lungs had been examined, it would have been found that phthisis was the cause of death. In the Fulbourn Asylum, Dr. Bacon some years ago showed me a woman, aged fifty-three, whose head measured twenty-seven inches in circumference, and who could sing and talk well, and had a tolerably good memory and intelligence. I do not know at what age the disease arose. In some rare cases, in which hydrocephalus thus becomes arrested, the intellectual faculties are said to reach a normal or even an exceptionally high standard. The name of the author of "*Vanity Fair*" has often been cited as affording an illustrious example of this. But it is doubtful whether the difficulty of distinguishing a rachitic enlargement of the head from "water on the brain" has been sufficiently taken into account in regard to such cases.

For, strange as it must appear, it is a fact that errors have not infrequently been committed, even by skilled observers, as to the presence of fluid within the cranial cavity; and not only has hypertrophy of the brain been mistaken for it, but it has sometimes turned out that the disease has been nothing more than rachitis. However, one can for the future avoid this particular blunder by measuring the circumference of the skull and comparing it with what it should be, according to the age of the child, in a table of standard measurements, such as one which is given by Huguenin. And that writer says that even where the yielding of the softened bones leads to an accumulation of ventricular fluid in a rachitic infant, the ophthalmoscope enables the true character of the affection to be easily recognized, since the optic discs retain their normal appearance. But, for my own part, I doubt very much whether these statements are applicable to the cases in

which there is really most danger of diagnosing hydrocephalus wrongly. These are probably examples of the affection which has been described as hypertrophy of the brain (p. 571), in which the skull is really much enlarged. I suppose that it was a case of this kind which many years ago occurred to me at the Evelina Hospital, but of which I have unfortunately preserved no notes; during the child's life the presence of hydrocephalus was never doubted by me, nor by any one else who saw it, although the necessity of caution in the diagnosis of that disease had already before that time been impressed upon my mind; but when the skull was opened it was found to contain nothing but a large, solid brain.

Course.—I have already incidentally remarked that the course and termination of hydrocephalus vary widely in different cases. Sometimes the disease undergoes a rapid advance, and destroys life in a few months by coma or by means of a succession of epileptiform seizures. Very often the child dies through some intercurrent malady, such as measles or whooping-cough; or, if it is rachitic, it is cut off by laryngismus stridulus or by some other complication. In certain very rare cases the fluid makes its way through the parietes of the head, and may even be discharged externally. Rokitansky once saw it poured out through the open sutures and diffused beneath the tissues outside the cranium. Several writers have recorded instances in which it has escaped through the nose or even through the upper eyelid; and some of them have ended in the recovery of the patient. When the discharge takes place into the nasal fossæ, it is supposed that the ethmoid bone must have been loosened from its attachments by the pressure to which it is subjected.

Prognosis.—If the quantity of fluid is not large, and if the tendency for it to go on accumulating has never been very active, it often happens that the morbid action undergoes arrest after a time, and that as the child grows older the increased size of the head ceases to be conspicuous. Some years ago, in examining the body of a man, aged forty-three, who had been killed by an accident, I found well-marked hydrocephalus, which I supposed to have been a residue of an attack of the disease in childhood; I could get no information as to the man's mental capacity or attainments. Persons have been known to live under such circumstances to advanced age.

It is, however, believed that cases of this kind have a marked tendency to relapse, and that excitement or overwork is very apt to light up active morbid changes afresh. And some writers have supposed that there is a special risk of their occurrence at the period when the ossification of the skull is finally completed.

Treatment.—The treatment of hydrocephalus by medicines very rarely leads to satisfactory results. As might be expected, the drugs which are chiefly prescribed are those which increase the secretions of the kidneys or of the bowels, and those which are believed to possess the property of accelerating the absorption of the fluid products of inflammation. Small quantities of gray powder or of calomel, pills containing mercury, squill, and digitalis in doses adapted to the age of the patient, the liquor hydrargyri perchloridi, and the various preparations of iodine are those which are most frequently prescribed. I cannot say that I have ever seen any good effect from the use of such remedies.

Indeed, the very frequent presence of rickets in hydrocephalic children would rather suggest the administration of cod-liver oil and of the preparations of iron; with which, however, one may combine digitalis, or the acetate of potass, or the perchloride of mercury, if it should be deemed advisable. But the mode of treatment which has the largest amount of testimony in its favor consists in the application of pressure to the head. At one time it was usual to cover the whole of the scalp with

strips of adhesive plaster. But of late it has been found sufficient to surround the head with a fillet of elastic webbing, two or three inches wide, and of which the length must be carefully adjusted so as not to cause redness, or to impress the pattern of the material upon the skin. Care must also be taken to shift it from time to time, so that it may not irritate the frontal eminences. The necessity for such precautions is shown by the fact that sloughing of the integuments, leading to the death of the patient, has been known to occur when they have been neglected. Dr. Dickinson speaks in very high terms of this procedure, and says that it may be expected to succeed in arresting the disease in the majority of cases, provided that the child is young and that the enlargement of the head is of recent development. One instance which he relates in detail, is that of a boy, aged thirteen months, whose skull had been growing out of proportion to the rest of the body for nine months, and measured twenty-two inches round. The eyes were depressed. There were occasional convulsive attacks. An elastic bandage was put on, and it was worn for three years, being renewed as often as was necessary. Diuretics and cod-liver oil were given during the same period. Within four months the circumference of the head became reduced to 20⅝ inches. At the end of the treatment the bones were fully ossified; and although the head, which was then completely covered with hair, measured 21½ or 21¾ inches, its disproportionate size was much less conspicuous. The position of the eyes was natural. There had been no convulsions for eighteen months. The child, at this time more than four years old, was sensible, and could make use of simple words for talking; but he appeared to be somewhat deficient in memory.

Paracentesis of the head with a fine trocar, at the outer angle of the great fontanelle, has been recommended by some physicians, and I have seen it performed on several occasions. Perhaps the best thing that can be said in its favor is still the fact that in a case recorded many years ago by Mr. Greatwood, a hydrocephalic child, having accidentally fallen upon a nail, which penetrated its skull, recovered after three pints of fluid had slowly escaped through the wound. Scarcely less successful results have been reported as having followed the intentional carrying out of a similar process; but the cases in question seem not to have been able to withstand criticism. Only a small quantity of fluid (not more than two or three ounces) should be withdrawn at a time, on account of the danger of setting up convulsions. If the result appears to be good, one may have recourse to the same procedure again and again, a bandage being applied in the intervals. But such repeated operations are very likely to be followed by an acute meningitis, or to set up suppuration within the cavity of the ventricles. And I am inclined to think that this mode of treatment should not be adopted, unless it be in the hope of warding off death for the time, when it is threatened by epileptiform seizures or by coma. The injection of iodine into the interior of the brain has occasionally been practiced, and has not been followed by the serious results that might have been anticipated; but there is no ground for supposing that it is likely to do any good.

4. Hydrocephalus of Adult Life. Chronic Meningo-Ependymitis. —Many writers mention the fact that adult patients are sometimes affected with a chronic disease of the brain, in which the most conspicuous anatomical change is the distention of the ventricles with a more or less considerable quantity of fluid. Dean Swift is said to have died of such a complaint, after an illness of three years' duration, in 1745; but one would hardly like to speak very confidently of the accuracy of an observation made so long ago, when scarcely anything was known of pathology, and when no

account could have been taken of the possible relations between the effusion and a primary atrophy of the nervous tissues, or a cerebral tumor, or even a chronic affection of the kidneys. Sir Thomas Watson, however, speaks of a young and distinguished lawyer, who, after one or two sudden attacks of unconsciousness, became dull, stupid and insensible, and at length died; the only alteration that could be detected in the brain was the presence of a large quantity of serous fluid in the ventricles.

I have already remarked that pathologically there is a close correspondence between cases of this kind and those of hydrocephalus occurring in childhood. Similar changes are found in the ependyma of the ventricles; it is tough and thick, and may be granular and feel rough to the touch, or may have an areolated appearance (like that of the capsule of the liver in some cases of perihepatitis). The floor of the third ventricle often forms a protrusion like a bladder of fluid. I once saw the distended descending cornu of the left lateral ventricle projecting in a similar manner when the middle lobe had been removed. The membranes at the base, too, are found greatly thickened and opaque, and matted together; even more so, I think, in the affection of adults than in that of children. In the case just referred to, the velum interpositum could not be dissected off from the corpora quadrigemina, nor could the pineal body be isolated. There is, of course, a great difference in the quantity of the fluid, which seems not to have exceeded fourteen ounces in any of the cases observed at Guy's Hospital within the last few years. Whenever its characters have been recorded, it has been clear, and has contained only a very small quantity of albumen. The cranial bones are generally thin; there is a deficiency of the diploë; the interior of the skull is marked by sharp ridges and projecting points, with sulci between them which are obviously attributable to the outward pressure of the brain. There is a very marked flattening of the cerebral hemispheres.

Symptoms.—Clinically, however, the relation between the hydrocephalus of adult life and that of childhood is one of contrast rather than of resemblance. The enlargement of the head which renders the diagnosis of the disease so easy is wanting; and in the symptoms which are present, there is scarcely anything to distinguish it from other chronic affections of the brain. The only way in which I can attempt to give any account of them is by briefly abstracting the notes of some of the more striking among fifteen cases of which I have notes.

1. A man, aged twenty-three, a patient of Dr. Wilks, said that he had been well until a year before, when he noticed a numbness in his feet and legs, which gradually extended up to his face. He had kept his bed for three months. A fortnight back he once found himself unable to micturate, so that a catheter had to be used. Within the last week he had had two fits, in which he was insensible. On admission there was a slight convergent squint; the pupils were dilated; the optic discs were ill defined and red, with some plugging of the veins and retinal hemorrhages. He had numbness of the feet and legs, and of the face about the mouth. He did not chew his food properly; portions of it would fall out of his mouth while he was eating. His mind seemed not to be clear; and his statements about his case varied from day to day. He passed his urine into the bed. After a few days he died suddenly.

2. A man, aged thirty-two, under the care of Dr. Pavy, had been obliged to give up work eighteen months before his admission into the hospital, on account of a pain in the head which prevented his sleeping. At that time he was laid up a week or ten days. Nine months ago he lost his speech, became hemiplegic on the right side, and was insensible for three weeks. From that time his memory was imperfect. Eight days before his

death he again became affected with partial hemiplegia and inability to speak. He passed his urine and fæces under him. During the last few hours of his life he had a series of fits, which began in the left side of the face, and in which the eyes were turned to the right.

3. A man, aged fifty-seven, a cooper, was taken in on account of a partial right hemiplegia, which he said had come on suddenly four days previously, while he was in the act of striking a piece of iron; he managed, however, to get home afterward, the distance being half a mile. He did not seem very ill on admission, and looked a strong, healthy man; but he soon afterward died rather suddenly, having been ill only nine days. It must be mentioned that in addition to an immense distention of the ventricles, with marked roughening of the ependyma, there was in this case softening of the superficial parts of the corpora striata, and here granule masses were discovered with the microscope.

4. A man, aged fifty-five, was admitted for chronic dilatation of the stomach, in order that the treatment with the stomach-pump might be carried out. But he became light-headed, and two days later he was convulsed and fell into a semi-comatose condition, in which he lay groaning and muttering until at the end of a few hours he died. When his cerebral symptoms set in, it was found that he had complained of severe pains in the head three months previously.

5. A boy, aged fifteen, who had two sisters in an asylum, was taken in on account of very obscure symptoms, which at first seemed to point to some chronic disease of the peritoneum. He was anæmic; he vomited occasionally; there was slight fever; some of the superficial glands were swollen; he complained of pains in the head and in the back of the neck. After a time his mind began to wander; he lay on his side, with his legs drawn up; when questions were put to him, he answered slowly and unwillingly; his eyes were half closed; his pupils were dilated; the temperature was now below normal, being sometimes not above 97°. He died very gradually.

6. A man, aged twenty-two, was admitted under the care of Dr. Habershon, in 1871, for severe cerebral symptoms, which were supposed to be the result of an accident. He had once fallen from a scaffold, striking the left side of his head; he was insensible for a fortnight, and had bleeding from the mouth, the nose, and the left ear. At the end of three months he resumed his work, but it was noticed that he was strange in manner. For a time he was free from headache. Seven months before his death, however, he was attacked with violent pain in the head and with shivering. He gradually became unable to stand, and passed his urine and fæces under him. While in the hospital, he lay all day in a drowsy condition. He would answer questions, but soon began to ramble in his talk. He was occasionally sick. His pupils were dilated. His head was drawn back. Two months before his death he had a fit; a month later he had another fit. After this he lay perfectly still, saying nothing, and eating no solid food; toward the last he became extremely emaciated. I made the post-mortem examination, and found well-marked indications of chronic meningitis, and a greatly dilated state of the ventricles. The anterior and middle lobes of the brain were also adherent to the dura mater at the base, especially on the left side. There was a little ochery-yellow discoloration, extending into the brain substance, and no doubt the result of effusion of blood at the time of the injury. The foramen of Bichât at the apex of the fourth ventricle appeared to be closed, so that the case might perhaps be cited in support of Hilton's theory with regard to the origin of hydrocephalus. But it must be added that there was great thickening of the velum interpositum, and that it therefore seemed quite as probable

that the chronic ependymitis had arisen by direct extension from the surface. The ventricular fluid contained scarcely any albumen.

7. A man, aged thirty, a patient of Dr. Wilks, died in the hospital, in 1876, of the effects of disease of the aortic valves, and bronchitis. Toward the last he seems to have had no very marked cerebral symptoms, but when admitted he was comatose, passing urine and fæces under him, and he remained so for several days, before he gradually recovered his consciousness. His insensibility arose out of a succession of fits, which he had been having very frequently. Afterward, while in the ward, he had one or two of them, affecting the left side. He had a fairly intelligent appearance, and answered questions readily. He stated that eight years back, at which time he was a healthy young man, he had fallen from a ladder, and cut his head. He was brought home insensible, was delirious for two days, and was laid up for eleven weeks. Ever after he was unable to do any hard work. His memory failed him ; he had headache ; the sensibility of the left side of the body gradually became defective. His first fit occurred five years before his admission. Dr. Goodhart made the autopsy, and found the lateral ventricles, the third ventricle, and the aqueduct all distended, and forming a large cavity which held fourteen ounces of fluid. There was a repaired fracture at the base of the skull. The prominent parts of the brain on its under surface were discolored where they had been bruised.

I think it is clear, from these cases, even in the condensed form in which I have been obliged to record them, that the diagnosis between hydrocephalus and other cerebral affections is, in the adult, exceedingly difficult, if not impossible. In the first of them, the fact that the numb sensations appear to have been bilateral might perhaps have suggested the nature of the disease. But in the second and third cases there was hemiplegia, which seems not to have been distinguishable from that which might have been caused by a tumor.

So far as I am aware, the only writer who has hitherto attempted to give a systematic account of the hydrocephalus of adults is Huguenin. But in his article on the subject in Ziemssen's "Handbuch," the description of the symptoms by no means corresponds with the observations just cited. He speaks of the affection as bearing the closest resemblance to general paralysis (*dementia paralytica*), and he gives full reports of two cases in which such a resemblance undoubtedly existed, although there was not the *délire des grandeurs* on which so much stress used formerly to be laid in diagnosis. The apparent discrepancy is to be accounted for by the fact that neither of Huguenin's patients died in the wards of a general hospital ; one was an inmate of the lunatic asylum at Zürich, the other was nursed at home. His cases, therefore, should be taken as supplementing mine ; and they afford additional proof of the variety of the aspects that the disease may assume.

In each of my last two cases the hydrocephalus was distinctly attributable to a severe injury from a fall on the head several months or some years previously. In one of Huguenin's patients a similar origin was no less directly traced, although the accident itself appears to have been a comparatively slight railway collision. It is a very interesting and important question whether the occurrence of serious cerebral symptoms under such circumstances may not generally be taken to indicate that a chronic meningo-ependymitis with effusion into the ventricles is developing itself. There are, indeed, other possibilities to be taken into acconnt. As I have mentioned at p. 537, a tumor has sometimes been found where the supposition during life had been that the patient's symptoms were of traumatic origin ; and it may be that new growths are, in fact, sometimes set up by injuries. It has been commonly believed that an abscess is not unlikely to be met with in

cases of this kind. But I am very doubtful about such an occurrence, unless, indeed, the patient's fatal illness has gone on without any interruption from the date of the accident. I am not sure, however, whether there may not sometimes be considerable cerebral disturbance, lasting for a long time, when the only discoverable lesion is an ochery-yellow discoloration from bruising of the under surface of the brain. And once I examined the body of a man who died of some other disease, but who was said never to have recovered from the effects of an injury to the head, having been unable ever afterward to take stimulants even in moderation; and in that instance the brain, the membranes, and the bones, all appeared to be thoroughly healthy.

Huguenin, indeed, alludes to cases in which persons who had had blows or falls on the head, after suffering more or less severely for several months, or even for years, have at length partially or completely recovered. And his opinion is that they depend on a meningitis of limited extent. He remarks that the chief symptoms are headache, giddiness, and other subjective sensations of a distressing character, which often cause the patient to withdraw himself from society. The pupils may be unequal and sluggish. I have a case of this kind under observation in the person of a bank porter, who, seven years ago, received a blow on the head from the heavy door of an iron safe; ever since he has been liable to a pain in the occipital region, vertigo, and a peculiar light feeling in the head, but sometimes he is free from these symptoms for some weeks. "The least thing," he says, "seems to affect his head," so that he can take scarcely any stimulant. Reading often makes him feel giddy; and he has been obliged to go out of church on account of indescribable sensations of discomfort. Once he complained to me of a numbness in the left side of his head, and at another time of cramp round the neck, as if his collar were too tight. I may add that the optic discs are normal.

I have found the bichloride of mercury more useful than any other medicine to this patient; on one occasion it kept off his symptoms entirely for about a year. He has also taken bromide of potassium and the ammoniated tincture of valerian; and a blister has once been applied to the back of the neck. He has several times been obliged to be away from work for two or three weeks; and, no doubt, the rest which he has had has been an important part of the treatment.

In the more severe cases of hydrocephalus, similar measures appear to be the best that can be adopted whenever its presence can be determined. Huguenin advocates the continued application of a bladder of ice to the head, or of a stream of cold water, for weeks or even months together; he also recommends periodical leeching, saline purgatives, and small doses of chloral for the relief of pain. The bromides he has found useful, but not the preparations of mercury or of iodine.

5. Atrophy of the Brain.—Under this head it is at present necessary to include two groups of cases, which in some of their clinical features have to be sharply contrasted with one another. The first group consists of a somewhat special form of insanity attended with definite paralytic symptoms; it is for the most part studied in asylums, it occurs chiefly in males and at a particular period of life, and there is reason to believe that it is generally traceable to sexual excesses. It differs from all the other forms of insanity in being constantly dependent on an organic change in the brain, and yet the change in question, so far from being peculiar to it, seems to be no less marked in the second group of cases, in which there may be the utmost variety of symptoms or even no symptoms at all, and which includes adult persons of all ages poisoned by alcohol or by lead, as well as old people in whom the atrophy appears to be a mere consequence of advancing

years. Lastly, and as if to complete the confusion and to render an accurate description of these affections impossible, the morbid appearances themselves present many differences in individual cases, and I doubt whether there is a single one of them which may not sometimes be wanting.

a. General Paralysis of the Insane. *Dementia paralytica.*—With the exception of a slight reference to it in the works of Thomas Willis, the celebrated anatomist and physician, who died in 1675, the earliest mention of this disease seems to have been made by another Englishman, John Haslam, in 1798; but afterward its study was especially taken up by the French, and the first complete account of it is that which was published by Calmeil in 1826. Of late years it has attracted much attention both in England and in Germany, but even Griesinger refuses it an independent position in the nosology, and describes it in a chapter on the complications of insanity. Yet we shall find that its symptoms and course are remarkably definite. It shows little or no tendency to shade off into the other forms of mental disorder; it is not apt to come on secondarily in persons who are already the subjects of chronic insanity, nor does a relapse of mania or of melancholia ever assume its characters. It is, in fact, from the first, distinguished more or less plainly by features of its own. The impairment of muscular power, too, is of a peculiar kind. One could not make a greater mistake than to suppose that the mere association of hemiplegia or paraplegia with unsoundness of mind constitutes paralytic insanity. Thus, in Ziemssen's "Handbuch" the article on this disease, which is written by Hitzig, is placed away from those on the psychoses proper, and in one of the chapters devoted to organic affections of the brain. Perhaps the best name for it is that which he uses, namely, progressive paralysis of the insane, but in this country it is commonly called "General Paralysis of the Insane," or, more briefly, "General Paralysis," or sometimes "Dementia Paralytica." It constitutes a large proportion of the cases which in popular (but not in scientific) language are spoken of under the name of "Softening of the Brain." Its importance may be estimated from the fact that in some lunatic asylums as many as one in four of all the male patients are said to suffer from it.

The persons most apt to be attacked by general paralysis are men in the prime of life. The proportion of males to females is variously stated by writers; some give it as eight or even ten to one, others as no higher than two to one. Dr. Blandford observes that the women who fall victims to this disease almost all belong to the lower classes, whereas it is commonly seen in men who have been highly educated and who possess powerful frames and handsome faces—men who have enjoyed life and lived hard. According to Mr. Austin the subjects of it are generally fair complexioned and thin skinned, with blue or gray eyes. The time of life at which it usually occurs is between thirty and fifty years of age, and particularly about the middle of that period. In persons over sixty it is scarcely ever seen, although Mr. Austin refers to one instance in a man aged seventy-six. Those who live in towns and cities furnish a far larger proportion of cases than do rural populations.

Causes.—It is commonly said that general paralysis differs from other forms of insanity in being less frequently due to inheritance or congenital predisposition, and if this statement is correct one would rather expect that it should be traceable with corresponding facility to acquired conditions. But there are considerable differences of opinion with regard to its exciting causes. Dr. Blandford is strongly disposed to attribute it in most cases to sexual excesses. He does, indeed, admit that he has not always been able to refer it to such an origin, but then, as he says, in married men there may be great difficulty in ascertaining the fact, and an amount of sexual intercourse

which to some would be perfectly harmless may be very prejudicial to others, if they are possessed of less natural vigor or have their energies fully taxed in other ways. Dr. Blandford speaks of having seen several cases in men who had formerly been dissipated, or who when no longer young had married wives much younger than themselves. Dr. Sankey found that at Hanwell a large proportion of the women affected with general paralysis had led irregular lives. Griesinger is rather disposed to attribute the disease to excessive mental excitement, but, he adds, especially to "emotional agitations." Mr. Austin says that it commonly follows a painful moral shock, such as would be caused by bankruptcy, or destitution, or might arise from remorse. Hitzig speaks of it as probably resulting most frequently from the combination of intense work with venereal excesses and indulgence in alcoholic beverages. He and other writers also mention that it sometimes follows injuries to the head, or occurs in those who have before passed through some acute febrile disease.

Symptoms.—The course of general paralysis is commonly said to be divisible into three stages. During the first period an alteration in the character is the most striking symptom. Perhaps the man is extravagant in his expenditure, making presents to persons of whom he knows scarcely anything; or he may be dull and sulky in his demeanor, or depressed and melancholy. So far there is nothing distinctive of this rather than of other forms of insanity, but what is peculiar is an impairment of memory. Dr. Blandford —from whom I take most of the details in regard to the symptoms of the disease—says that such a patient is regardless of appointments, forgetful of the time of meals, of the hour for going to bed and the like. He comes and goes, scarcely noticing those about him, giving absurd and conflicting orders to his servants, and falling into a passion if they are not instantly executed. He neglects his business, and is careless and indifferent to things which formerly interested him. When he takes up a new scheme his interest in it soon flags. He may commit indecent actions, but if he exposes his person he often seems to be half unconscious of what he is doing; or he, perhaps, commits assaults upon women in a foolish manner, without regard to opportunity, place, or consequences. He sleeps badly, eats irregularly or voraciously, and drinks to excess, from inattention and forgetting how much he has already taken. He eats hastily, and is apt to spill his food on his dress. He is neglectful of his appearance, and his costume is often unsuitable for the occasion, or the different parts of it are incongruous with one another. At this time there is no physical change which can be detected by the eye of the physician, unless, indeed, the pupils are abnormally small and less mobile than natural, as appears sometimes to be the case.

It is of the utmost importance that the distinctive characters of incipient general paralysis should be well known to the physician and the general practitioner, since they alone are likely to see the patient at this period of his disease. The early stage is not of long duration; after a few weeks, or at the end of a month or two, further symptoms develop themselves, and the patient becomes manifestly insane. The delusions which he now exhibits are in certain respects peculiar. They are almost always connected with ideas of colossal size, or magnificent wealth, or extravagant numbers. He may say that he can walk 100 leagues in a day, or write 100 tragedies and 1000 poems in the same space of time; or that he is going to make his fortune by buying up all the joint-stock banks; or that he is about to marry the Queen and all the princesses. French writers give to this form of insanity the name of *monomanie des grandeurs,* but it is to be noted that these "large delusions" are not maintained from day to day, nor are they consistent with one another. In fact, in spite of his eager excitement,

the patient's mental condition continues to be one of progressive decay. He is extraordinarily self-satisfied, full of ideas of greatness, importance, and riches, but he takes no notice of the fact that the palace in which he believes himself to reside is really a lunatic asylum, and that the great men whom he supposes to surround him are mad people like himself. Thus he in reality cares much less about being placed under restraint than most other insane patients. In some instances, indeed, the delusions are not all of such a kind. Dr. Blandford mentions that one of his patients thought he was going to be arrested, that people were about to injure him, that they were maligning and would rob him. Yet, although his symptoms so far resembled those of melancholia, this man was often cheerful and talkative: he was very vain of his appearance and exceedingly fond of his food. Dr. Blandford also notes, as instances of ordinary delusions, that one patient believed himself given over to the devil, another thought that poison was put into his food, and a third that he had committed sins too enormous to be forgiven.

This second stage of general paralysis is further characterized by certain physical symptoms which must be carefully looked for. The earliest of them is commonly a defect of articulation, an indistinctness or thickness of speech, or a hesitation in the middle of a sentence, or a tendency to substitute for the proper word another of which some letters are the same. The tongue, when protruded, is tremulous, that is, it not merely shows a fibrillary tremor —which is often observed in persons who are in perfect health if they are nervous or anxious—but it oscillates irregularly to and fro, and cannot be held quiet. Sometimes the lips are seen to quiver, as they do in persons about to burst into tears. Mr. Austin also says that the mouth is generally closed, that the lips are compressed, and that the upper one is straight, its natural curves being obliterated. The pupils are almost always unequal, and they are sluggish. In some cases the gait becomes altered, the legs are not lifted properly, the patient walks stiffly, or straddles, or shambles along, and stumbles over any obstacle in his path. The movements of the hands and arms may also be impaired; Griesinger speaks of them as being "stiff;" objects are grasped convulsively and afterward suddenly allowed to fall. Dr. Mackenzie Bacon has pointed out that an important indication of the disease at this stage is an alteration in the character of the handwriting; it becomes tremulous and uncertain; sometimes, too, words are omitted from the sentences which the patient writes, or the same sentence may be written over and over again; or the whole may be an incoherent jumble. All these, however, are indications of mental rather than of bodily failure.

In almost all cases one or more fits occur during this period of the disease. These may be of various kinds; the patient may become profoundly comatose, with complete paralysis and anæsthesia and abolition of reflex movements, and in such an attack he may die. Or he may be less deeply insensible, with loss of power affecting one side only. The paralysis is often very transitory, disappearing in a few hours. Mr. Austin was impressed with the belief that seizures of this kind were often direct results of the accumulation of scybala in the large intestine, and that the administration of purgatives and enemata was very efficacious in removing the hemiplegia. Or the fits may be more or less distinctly epileptiform, and these, again, are often directly fatal, as in the case of a man, aged thirty-seven, who died in Guy's Hospital in 1855. Lastly, the patient may be attacked with convulsions alone, without becoming insensible or even falling to the ground.

Persons affected with general paralysis in its second stage are also exceedingly liable to paroxysms of rage and fury, surpassing in violence those that occur in any other form of insanity, except, indeed, those which are

believed to bear a more or less close relation to epilepsy. Even in an asylum such patients cause unusual anxiety and trouble to those responsible for their safety, and all writers are agreed that it is scarcely ever justifiable to allow them to remain in the hands of friends or relations at this period of the disease. Dr. Blandford remarks that these are the persons who sometimes get their ribs broken by attendants before they can be mastered. They may make the most desperate efforts to escape ; or they may tear up their bedding and clothes and go about naked. They are often horribly filthy in their habits.

But after a variable period, a week, or a month, or a longer time, these violent symptoms commonly pass off. The patient's condition may even improve so much that he is able to leave the asylum. The disease is then sometimes said to be cured, but all writers are agreed that permanent, complete recovery is the rarest possible event, and almost all of them deny that it ever occurs. Dr. Blandford says that he has known some persons affected with general paralysis who were able to live with their families, to spend their money without extravagance, and to write long letters without mistakes, detailing their travels and amusements ; but he adds that he has not met with one case in which the patient was capable of work or business. The friends and relations of such persons notice a childishness and slowness of intelligence in them, and if they attempt to resume their former occupations they break down and have again to be placed in confinement. The only exception to this rule that I have met with in my reading is a case related by Schüle (*"Allg. Ztschrift. f. Psychiatrie,"* xxxii).

There are, indeed, many cases in which no improvement takes place. The patient's mental condition gradually passes into one of dementia, his bodily state into one of complete paralysis. His notions of magnificence may continue a little longer, but his understanding and memory soon become altogether destroyed. His speech cannot now be understood ; his power of swallowing is very greatly impaired. His hands tremble so that he can scarcely hold anything in them ; and it is as much as he can do to shuffle about the garden with the aid of an attendant. Sometimes he sits and grinds his teeth for hours together, making a horrible noise.

This state of affairs leads up to a fatal termination which is very seldom long delayed. Writers are not entirely agreed as to the average duration of the disease, but all say that it is short. According to Hitzig the majority of patients die in from fifteen to thirty months after their admission into an asylum. Calmeil and Griesinger speak of the ordinary course of general paralysis as coming to an end within a period of from several months to three years. But the last-named writer alludes to exceptional cases, in which life was sustained for as long as ten years ; and Dr. Blandford mentions particularly the case of a baronet of large fortune, on whom a commission of lunacy was held in 1858, when he was suffering from this form of insanity, and who was alive in 1870. This observer says that in his experience the average duration of the disease has been considerably longer than that stated by Griesinger, the reason being that his patients have all been in a position to have the best food and nursing.

Indeed, the immediate cause of death is commonly something which may almost be termed accidental. One way in which it is often brought about is by choking ; the patient goes on filling his mouth without swallowing the food, until it gets into the larynx and trachea, or at least fills up the pharynx so as to obstruct the entrance of air. Persons suffering under general paralysis should always have their meat very finely minced, and an attendant should be present at meal-times. Another way in which death occurs is by the supervention of a low bronchitis or pneumonia ; if overlooked at its commencement, this may destroy life in a few hours. I know of one

case in which the patient, a nobleman, had been for his usual drive in the afternoon, but in the evening was noticed to be out of sorts ; on auscultation it was found that he had pneumonia, and next morning he died. Dr. Blandford remarks that persons affected with general paralysis are always more feeble in cold weather ; during the heat of summer they may regain strength to a surprising extent, but with the first frosts they fall back, and are apt to sink. Bed sores sometimes seem to bring about the fatal termination.

Anatomy.—I have already more than once insisted on the fact that general paralysis of the insane differs from the psychoses in being constantly attended with morbid changes in the nervous centres. This, indeed, is admitted by all observers, but unfortunately, when the question is that of defining accurately their nature, and of distinguishing them from those which occur under other conditions, there is by no means the same agreement. Some writers have laid much stress on the fact that the pia mater is often abnormally adherent to the convolutions, so that the cortical substance becomes torn and assumes a ragged appearance when one attempts to strip off the membrane. But in other cases there is an excess of fluid beneath the arachnoid, and the pia mater can then be removed even more easily than from the healthy brain. Thickening of the arachnoid has now and then been noted ; pachymeningitis and hæmatoma of the dura mater, and increased density and thickness of the calvaria. The absolute weight of the brain has been said to be diminished ; the ventricles are described as being of unusual size, and their ependyma as being abnormally thick ; the centrum ovale is said to be peculiarly flaccid when exposed by the knife.

Some of the earlier observers, including Calmeil, described the cortical substance of the brain as of a peculiar violet-red color ; Westphal admits that it sometimes presents this appearance, but he says that in other cases it looks remarkably pale and faded. And, as he remarks, these variations probably depend simply on the mode of death.

Again, the ganglionic cells of the brain have been found affected with pigmentary or fatty degeneration. Hitizig says that this change is almost always present in a very large number of the cells ; but he goes on to quote Westphal as having stated that similar appearances are observed in other cerebral diseases, and even under normal conditions, so that he is obliged to add that it is only the extent of the degeneration that can be regarded as peculiar to general paralysis. Less commonly, he says, cells are seen which are swollen out and sclerosed ; this seems to be in accordance with an observation of Dr. Major's, who in one case ("*West Riding Asylum Rep.*," vols. iii and iv) discovered what he describes as an hypertrophy of certain cells, these being abnormally large and furnished with an excessive number of branches. Some have thought that the neuroglia is increased both in the gray and in the white substance. In the latter, patches of degeneration have been observed, of the kind termed miliary sclerosis.

In one case Dr. Lockhart Clarke found the white substance of the convolutions full of little cavities, of round, oval, fusiform or crescentic shape, and varying in size from that of a small pea or barleycorn to that of a grain of sand, so that the cut surface looked like Gruyère cheese, or crumb of bread. These vacuoles were doubtless analogous to those which have been described by Dr. Dickinson in other diseases. Most of them are empty, but some contained the remains of vessels mixed with granules of hæmatoidin. Probably they all were originally perivascular canals. Changes in the blood vessels have indeed been described by some observers as important features in the morbid histology of general paralysis. The small arteries become dilated and tortuous, and present twists or kinks in their course. Their nuclei and those in the walls of the capillaries are found to

have undergone proliferation. Granules of hæmatoidin may be scattered upon their exterior, and they may be affected with fatty degeneration or calcification.

Moreover, other parts of the nervous system, as well as the brain, present morbid changes. The state of the spinal cord has been especially investigated by Westphal, and he has found that its posterior and lateral columns are often extensively altered. In the former, the appearances are generally those of gray degeneration, exactly like that which constantly occurs in locomotor ataxy. I shall presently have to discuss the relations between the two diseases. In the lateral columns the appearances are rather those of a chronic myelitis. It is not to be supposed that such changes arise by the extension downward of a morbid process which had commenced in the encephalon, for Westphal could not trace the affection of the posterior columns above the commencement of the fourth ventricle, nor that of the lateral columns beyond the lower end of the crura cerebri. They must, therefore, be regarded as of independent origin. Indeed, it would appear that even the peripheral nerves fail to retain their normal structure. At least, the sciatic nerves are said by Dr. Lewis ("*West Riding Reports*," vol. v) to be smaller and softer than natural, and less rounded in form. And under the microscope he was able to detect atrophy of the nerve tubules, with overgrowth of the connective-tissue elements. Again, Bonnet and Poincaré have pointed out that the great sympathetic ganglia, especially in the cervical region, constantly present morbid changes. The nerve cells in them are sclerosed and pigmented; they may even undergo destruction to a considerable extent, their place being taken by adipose and connective-tissue cells. The discoverers of these appearances believe that they constitute the starting point of the disease, and that all its phenomena depend upon the resulting vasomotor disturbances. But such a conclusion seems to me most unlikely to be correct. I should rather regard the changes in question as affording a further illustration of the fact that in general paralysis the nervous structures throughout the body simultaneously undergo degeneration.

Diagnosis.—This must be considered from two points of view. In some cases it is a question between general paralysis and the psychoses; in other cases between it and the various forms of paralysis dependent upon chronic alcoholism, brain wasting, or other diffused changes in the nervous centres. It is not generally difficult to say whether a patient is suffering from general paralysis or from mania, even at an early stage. But mistakes have sometimes been committed, from its being supposed that the presence of large delusions is characteristic of the former disease. Thus Dr. Blandford mentions the case of a gentleman who wanted to make a tunnel through the earth to the antipodes, and who thought that people might live a thousand years if they would bathe in beef tea and beer, and that he should be able to pay off all the mortgages on his estate by assembling 10,000 persons in his park, having them photographed, and selling the photographs at five pounds apiece. But it was clear that he was not laboring under general paralysis, for he did not stutter, his memory was perfect, and (above all) he had had a similar attack some years before. In cases of this kind a correct diagnosis is of the greatest importance, because the prognosis depends upon it. The opinion was given that the case was one of mania and that it might pass off; and the sequel proved the correctness of this judgment.

The exceptional cases in which general paralysis occurs in persons at an advanced age may be very difficult to distinguish from those of senile dementia, unless, indeed, the more characteristic symptoms of the former disease are well pronounced. The childish nature of the delusions in those who are

suffering from senile dementia commonly lends aid to the diagnosis; that complaint is slower and more uniform in its course; it is less apt to be complicated with epileptiform fits, and any apoplectic attacks that may occur are much more likely to be followed by permanent hemiplegia.

Chronic alcoholism sometimes gives rise to a chain of symptoms so like those of the less marked forms of general paralysis that it may be impossible to make a diagnosis between the two diseases until one has watched the case for a little time. Dr. Blandford says that the muscular weakness does not, in alcoholism, extend to the muscles of articulation, but this is inconsistent with the statements of most other authorities.

Lastly, there may be a difficulty in distinguishing general paralysis from some affections of the spinal cord. There are cases in which an impairment of muscular power shows itself some months before any psychical symptoms develop themselves. It has even been a question whether the disease may not sometimes run its course without being attended at any time with impairment of the mental powers. This question is answered in the negative by alienist physicians; but one may fairly reply that those observers would have no chance of seeing such cases, supposing them to occur. It is in the wards of a general hospital that they must be looked for. But, so far as I am aware, there is no such case on record. Dr. Wilks has published in the "*Guy's Hospital Reports*" (xvi, p. 194) at least one instance in which the patient's mind was unaffected while he was under his care, the paralytic symptoms being well marked. But he had only been ill nine or ten months, and I think that, in all probability, the mental symptoms afterward developed themselves.

There is sometimes a special difficulty in distinguishing general paralysis in its early stage from locomotor ataxy. Dr. Wilks relates that two physicians—one an authority with regard to the former disease, the other with regard to the latter—had the same case shown to them, and that each of them pronounced it to be an example of the malady with which he was the more familiar. The explanation seems to lie in the fact that the two diseases are not infrequently present in the same individual at the same time. I have already quoted Westphal's observations as to the occurrence of gray degeneration of the posterior columns of the cord in cases of general paralysis; and that writer expressly states that they had presented the ordinary symptoms of locomotor ataxy. As far back as 1862, Baillarger published clinical reports of five cases of a similar kind.

Treatment.—With regard to this there is unfortunately not very much to be said. I have already stated that in the early stage of the disease confinement in an asylum is absolutely necessary. It is, indeed, a question whether it might not sometimes run a more favorable course if the patient were secluded at an earlier period than is usually the case. His friends and relations are but too apt to insist on his having amusement, to take him to the seaside, or even to make him travel about from place to place; whereas, what his brain really needs is absolute rest. Among medicinal agents Dr. Blandford speaks very highly of tincture of digitalis in doses of ♏xv to ♏xxx every four hours. He says that it often soothes such patients wonderfully, and restores them from a state of noisy turbulence to one of comparative rationality. According to Hitzig the application of galvanism to the medulla oblongata is sometimes of marked temporary utility; he also speaks favorably of iodide of potassium. The bichloride of mercury has been largely used, but without any good result. Dr. Blandford lays stress upon the importance of withholding stimulants, such as brandy, while there is excitement; but he says that opium and morphia and chloral are often useful both in the early and, still more, in the later stages of the disease. The same writer lays stress on the value of

tonics when the more acute symptoms have passed off, the tincture ferri perchloridi and the other preparations of iron being particularly serviceable. Dr. Crichton Browne has recently ("*Journal of Mental Science*," 1875) published two cases in which the extract of Calabar bean in doses of a quarter to a third of a grain, continued for nine or twelve months, appeared to cure the disease, although it was in an advanced stage.

β. Other Forms of Atrophy of the Brain.—I have already remarked that, in sharp contrast with the definite clinical course of general paralysis, which disease is chiefly seen in asylums for the insane, there occurs in ordinary medical practice a precisely similar atrophy of the brain, attended with the greatest possible variety of symptoms, or even with no symptoms at all. This affection has been very generally ignored, although Dr. Wilks published some observations in reference to it in the "*Journal of Mental Science*" for 1864. But that it is not very infrequent is, I think, evident from the fact that I have before me notes of no less than fifty cases in which it has been found in the post-mortem room of Guy's Hospital within the last twenty years; and that number might be considerably augmented, for in making my abstracts I selected chiefly the more striking examples.

The wasting of the cerebral substance is characterized, not only by loss of weight, but also by an obvious shrinking. When the dura mater is turned aside, after the calvaria has been removed, the surface of the hemispheres may appear to be covered with a gelatinous substance, which, however, is in reality merely an accumulation of serous fluid in the meshes of the pia mater, that runs out as soon as the arachnoid is punctured, leaving the membranes collapsed and wrinkled. The convolutions are small and rounded, and they are separated by deep, broad sulci; the ventricles may be wide; the ependyma is often granular; the choroid plexuses have undergone more or less of cystic degeneration. The cerebral substance itself is in some cases firm and natural looking. But in other instances it presents patches of softening, either thickly scattered throughout the white matter beneath the convolutions or limited to the basal ganglia. In an old man of seventy-six, whose body was examined in 1876, the *centrum ovale* showed smooth-walled cavities round the vessels, especially in the neighborhood of the gray matter, so that it had an appearance like that of Gruyère cheese—exactly such as was described by Lockhart Clarke in cases of general paralysis. When morbid changes of this kind are present, the minute arteries are, as a rule, themselves greatly diseased. Not unfrequently they are so thickened and even calcified, that their cut ends stick up out of the cut surface of the brain as if they were so many little bristles embedded in it. The membranes, too, are often thickened and opaque, especially over the hemispheres; and the ependyma of the ventricles is granular. It would be difficult to deny that these last appearances are indicative of a chronic inflammatory change, rather than of a mere atrophy; but they certainly seem to occur in cases in which there is no other evidence of inflammation.

The causes of atrophy of the brain vary in different cases. Sometimes it seems to be a *senile change*, and it may then give rise to no obvious symptoms; the old man whose hemispheres resembled Gruyère cheese,* was said to have been perfectly clear in his intellect. In other persons of advanced years, however, there is a more or less marked failure of both mind and body. They are eccentric or odd in manner, or even demented. Of one patient, a woman, aged sixty-eight, who was brought to the hospital for a fractured thigh, it is noted that she got out of bed, splint and all, and tried to walk about, with the result that the limb became gangrenous.

* [See remarks on this condition of brain by Drs. Savage and W. H. White ("*Path. Tr.*," vol. xxxiv, p. 1, 1883).—ED.]

Another, a man, aged seventy-four, who was taken in for bronchitis, showed no special cerebral symptoms, except that he persisted in passing his evacuations under him. Others, again, have been admitted for injuries due to attempts at suicide; a woman had thrown herself out of the window and fractured her spine; a man had cut his throat. Probably many cases of this kind are undistinguishable from those of ordinary insanity, in which the brain remains perfect, so far as our means of investigation enable us to determine its state. Nor is the affection confined to old people; I have notes of several cases which occured in persons between forty and fifty years of age. Thus, in 1867, there died in Guy's a man, aged forty-nine, who had been an accountant, but who for some time had been unable to do his work. His memory had failed him, his speech had become uncertain, he wrote badly, and he spelled his entries in a check-book all wrong. Afterward, however, his writing was correct, but he formed his words very slowly. When speaking he would lose the thread of his discourse, and would vainly clasp his head with his hands in search of what he wanted to say. For two or three days before his death he lay in a doze. At the autopsy patches of brown discoloration, breaking down in their centres, were found in each corpus striatum; the cerebral arteries were very rigid and calcareous; the kidneys were granular.

In some cases of this kind the disease advances to a fatal termination within a few months of its commencement; its diagnosis from other organic affections of the brain may then be impossible. There is often a more or less complete paralysis of some or all of the limbs; sometimes convulsive movements or jactitations are present; sometimes epileptiform seizures occur. Headache appears not to be generally a marked symptom; but in one patient it was exceedingly severe for the last six years of his life, and unintermittent for three months at a time.

Again, cerebral atrophy is often the result of chronic *alcoholic poisoning*. A large proportion of those who die of delirium tremens have wasted brains; but that disease cannot be described as being merely an effect of the morbid change in question, since in some cases the brain is found to be perfectly healthy.

Yet another cause of it is *poisoning by lead*. In 1863, a compositor, aged thirty-four, died in Guy's who had been admitted for colicky symptoms, and who afterward became almost totally paralyzed; the convolutions of the brain were shrunken, the sulci were deep and contained an excess of fluid, the ventricles were unduly large. Chronic plumbism, as is well known, sometimes gives rise to epileptiform convulsions, which may be repeated at intervals, and followed by delirium and by a fatal coma. I believe that in cases of this kind the brain is always found wasted.

INFLAMMATION OF THE MEMBRANES OF THE BRAIN.

Tubercular Meningitis—HISTORY—MORBID ANATOMY—RELATION OF THE TUBERCLES TO THE INFLAMMATION—THEIR OCCASIONAL ABSENCE IN BASAL MENINGITIS — ÆTIOLOGY — ONSET — COURSE—LATER STAGE—TUBERCULAR MENINGITIS IN ADULTS—DIAGNOSIS—PROGNOSIS.

Epidemic Cerebro-spinal Meningitis—HISTORY—MORBID ANATOMY—SYMPTOMS AND COURSE—ÆTIOLOGY—DIAGNOSIS.

Acute Non-tubercular Meningitis—ANATOMY—VARIED CAUSES—SYMPTOMS—DIAGNOSIS—PROGNOSIS.

TREATMENT OF MENINGITIS GENERALLY.

ADHESIVE THROMBOSIS OF CEREBRAL SINUSES.

HÆMATOMA OF DURA MATER.

Among the most frequent and the most fatal of all cerebral diseases are those which depend on inflammation of the membranes of the brain. Their study has been greatly simplified of late years. Until recently, it was usual to describe separately an "acute inflammation of the dura mater," an "arachnitis," and a "meningitis" seated in the pia mater. It is true that various diseases of the bones of the skull—such as, for instance, caries of the temporal—may give rise to ulceration or sloughing of the corresponding part of the dura mater. But that local affection requires no special notice; it is only preliminary to the development of a diffused inflammation of the other membranes or of an abscess within the brain itself; and upon these conditions all the clinical and pathological interest of the case is centred. Again, there is not, so far as I am aware, any disease that can be said to be strictly an "arachnitis." Pus or purulent lymph is sometimes found in large quantity within the cavity which lies immediately internal to the dura mater; and I shall have to describe its presence or absence in that space as constituting one of the distinctions between two different forms of meningitis. But the limitation of suppuration to a supposed serous sac, without implication of the pia mater, is not, I believe, known even to the surgeon, as a result of any kind of injury to the skull; and certainly the physician never meets with such an affection. If the arachnoid were perfectly analogous with the pleura or the peritoneum, we should doubtless expect it to be liable to inflammatory processes attended with the exudation of lymph and serum, and resembling those that occur so commonly in the other great serous cavities. But the modern anatomical doctrine is that no such analogy exists; the "parietal layer" is now regarded as merely the epithelial lining of the dura mater; and the "visceral layer" as nothing but an outermost condensed stratum of the pia mater. The subdural space—as the supposed arachnoid cavity is now called—is believed to be continued outward along the optic nerve, just like that which is termed the subarachnoid or "subpial" space. Formerly, it was, of course, supposed to be cut off by a reflexion of the arachnoid membrane.

Thus, the diseases now to be described are, all of them, forms of *meningitis*. They all are seated mainly in the loose tissue of the pia mater, over which they diffuse themselves more or less widely; but they differ much in their intensity, and still more in their causes. One of them is attended

with the development of tubercles; another occurs epidemically, and ought in strictness to be placed with the fevers rather than with local lesions of the brain; a third is made up of a group of affections themselves referable to a variety of conditions. The most common of them, at least in this country, is "tubercular meningitis;" it is, indeed, absolutely the most frequent of all organic diseases of the nervous centres. I shall, therefore, describe it first; and afterward I shall take up, in succession, the "epidemic" and that which may be termed the "simple" form.

1. TUBERCULAR MENINGITIS.—It is a curious circumstance that, although this disease has been known for more than a century, its real pathology has been recognized only during the last fifty years. The earliest detailed account of its symptoms was published in 1768, by Dr. Robert Whytt (a man of great eminence in his day in Edinburgh), under the name of dropsy of the brain; and in the early part of the present century it became generally known as "acute hydrocephalus"—a designation which was retained by Sir Thomas Watson as recently as 1857, as being "so established, both among medical men and with the public, that he could not venture to propose any change." He was, however, well aware of the fact, first pointed out by Papavoine in 1830, that the essential morbid change is in reality the presence of tubercles in the membranes, the fluid within the ventricles being quite of subordinate importance. And as inflammatory products also are generally found in large quantity, the name of tubercular meningitis, originally suggested by Bricheteau, is obviously a very appropriate one, and has now met with universal acceptance.

Morbid Anatomy.—The appearances found after death vary considerably in different cases, and in different parts of the brain. As a rule, the most marked lesions are found at the base. The diamond-shaped space bounded by the optic tracts and the crura cerebri is perhaps filled with gelatinous or puriform lymph, and a similar material envelops the great arteries arising from the circle of Willis and their branches. The inflammatory process may extend along the Sylvian fissures until it occupies a great part of the convex surface of the hemisphere. Generally, however, the membranes covering the convolutions show no obvious changes; their surface may be dry and the sulci reduced in size; but these are merely effects of pressure. A large quantity of lymph is often spread out over the pons and medulla oblongata, and it may be traceable in the subarachnoid space as far as the lower end of the spinal cord. Almost invariably there is a yellowish nodule upon the upper surface of the cerebellum, close to the opening of the veins of Galen; and sometimes the vellum interpositum and even the choroid plexuses are infiltrated and thickened. The fluid within the ventricles is increased in quantity, and is generally rather turbid. Its specific gravity may be raised to 1010; it yields a more or less distinct coagulum on boiling, and exhibits leucocytes under the microscope. The ependyma is often granular. The adjacent parts of the brain are usually much softened. The fornix and the septum lucidum may be almost diffluent, and even the great basal ganglia may have their consistence so much reduced that they fall into a shapeless pulp as soon as their removal from the skull deprives them of the support of surrounding structures.

These changes, however, are common to other forms of meningitis as well as that now under consideration. What is special to the latter is the presence of more or less numerous tubercles. If there are but few of them, they are most easily recognized in the Sylvian fissures or in the folds of pia mater dipping into sulci; they then appear as minute gray dots, adherent to the smaller arteries or to delicate filaments of connective tissue. When they are abundant, they may become fused together so as to ensheath an

artery in a granular mass. Some are generally found caseating, and their opaque, yellowish color enables them to be more easily recognized. Cheesy aggregations of considerable size may be found embedded in puriform lymph. If the tubercles are present in very large numbers, they may be seen thickly scattered as milk-white spots beneath the visceral arachnoid, especially on the under surfaces of the cerebral lobes. They may also be developed in the form of very minute transparent granules upon the inner aspect of the dura mater lining the fossæ of the base of the skull; and (according to Huguenin) between the two layers of that membrane, close to small twigs of the middle meningeal artery. In the vertebral canal they are found both on the smooth side of the spinal dura mater and in the arachnoid space. Within the ventricles I believe that they occur only in the choroid plexuses. They sometimes grow from the summits of the processes of the pia mater that pass down between the convolutions, appearing as whitish-yellow streaks along the small arterial branches within the cineritious substance of the cortex. Dr. Gee mentions a case in which the capillary vessels throughout the whole of one hemisphere were everywhere studded with miliary tubercles, which remained after the softened cerebral substance had been all washed away.

In one instance I found the right hemisphere affected on its convexity with a meningitis which appeared to be clearly associated with the presence of tubercles, but the left side of the brain and its base seemed to be entirely free. Huguenin describes two similar cases, in each of which the affection was limited to the territory of one of the Sylvian arteries.

The histological characters of meningeal tubercles have already been given at p. 83; and in discussing the theory of acute tuberculosis, I have advanced the reasons which lead me to think that the disease is not the result of any infective process, nor due to the absorption of matter from caseous foci, as is believed by so many modern pathologists. I have also remarked that even if one leaves out of consideration the scrofulous glands and similar affections, to which those observers attach so much importance, one still finds that tubercular meningitis scarcely ever occurs as an independent malady. In almost every instance it is either secondary to some chronic tubercular disease, for which the patient is already under treatment, or else it is (as Dr. Gee says) "merely a fragment" of a general acute tuberculosis. Not rarely, it is both consecutive to another chronic lesion and associated with the simultaneous development of recent tubercles throughout the body generally.

I have analyzed one hundred and twenty-four cases of tubercular meningitis that have occurred in succession at Guy's Hospital since 1856. Of these sixty-five were in persons under the age of twenty; fifty-nine in persons between the ages of twenty-one and sixty. Among the former there were thirteen in which the cerebral affection was secondary to hip joint disease or spinal disease, or some other malady capable of clinical recognition; among the latter there were twenty-eight such cases, mostly of pulmonary phthisis. In eighty-six of the one hundred and twenty four cases there were miliary tubercles in the viscera or in the serous membranes, sometimes, indeed, in very small numbers, but often in such abundance that the lungs or other organs were said to be "stuffed" with them. On the other hand, there are a good many instances in which the brain alone was examined, or in which the state of other parts is not mentioned. In only nine or ten of the whole number is it distinctly stated that recent tubercles were present in the pia mater and nowhere else.

Another question—which is of more importance in reference to tubercular meningitis than to any other form of acute tuberculosis—concerns the relation of the tubercles to the lymph and other products of common

inflammation that are so constantly found associated with them. The opinion expressed by Huguenin (in Ziemssen's "Cyclopædia") is that the miliary granulations are first developed, and that the pia mater tolerates their presence for a time, but that they afterward excite a process of reaction which passes on into inflammation. I believe that Rilliet was the first to propound this doctrine, and he went so far as to say that the so-called prodromata of the disease were effects of the actual "deposition" of the tubercular material. But as Wilks and Moxon have pointed out if such a view were correct, one ought sometimes to discover meningeal tubercles in small quantities, unmixed with inflammatory products, in those cases in which acute tuberculosis destroys life by invading the lungs or other organs; and to find it unattended with the characteristic cerebral symptoms. In this, they say, they have always failed, although they carefully examined the membranes in many cases. Moreover, inflammatory changes are always present where miliary tubercles are found in large numbers in the lungs; but it is certain that the development of the tubercles themselves is, in such cases, the direct cause of the patient's illness and death. On the other hand, one sometimes finds tubercles in the pia mater unattended with the exudation of lymph or pus in any appreciable amount. But I believe that in such cases symptoms resembling those of tubercular meningitis have always been observed during life.

I have met with two instances of this kind. One occurred in a woman, aged thirty-two, who was admitted under the obstetric physician for ulceration of the os uteri and for an abdominal tumor that turned out to be the omentum indurated by tuberculous matter. She was attacked by hemiplegia, coma, ptosis, and delirium, and died in seven days. In making the autopsy, I found that the brain and its membranes looked healthy, except for the presence of a single minute granule on a fold dipping into one of the sulci, and for that of a little filmy material round one Sylvian artery, which was absent on the opposite side. So slight was this change, that if it had been observable on both sides I should certainly have passed it by as unworthy of further investigation. But the microscope showed that even the apparently healthy artery which I had set apart for the sake of comparison had a distinct growth of tubercle about it; and the one little white grain in the pia mater was actually caseating in the centre, its periphery being made up of lymphoid tissue. The case shows that by minute investigation it is sometimes possible to trace the cause of fatal cerebral symptoms to a lesion that might readily be overlooked.

The other instance is that of a man, aged fifty, who became suddenly unconscious on May 2d, 1876, and was brought to the hospital in a state of coma, with stertor and right hemiplegia. He afterward partially recovered, and on May 5th he was sensible enough to answer questions that were put to him; but in the following night he was attacked with another fit, of which he died in twelve hours. I found tubercles thickly scattered about the Sylvian arteries and in the adjacent parts of the pia mater, but without any lymph being present; both lungs contained many gray clusters. In that case, indeed, it is probable that the tubercles had been formed more slowly than usual, for it was stated that a fit had occurred as far back as twenty-two days before the man's death, and that afterward he was always drowsy and stupid. It ought, perhaps, to be added that he was a gouty subject, and had Bright's disease of the kidneys, so that one was obliged to take into consideration the question of uræmia; but I came to the conclusion that the meningeal tubercles were the cause of his convulsive seizures, believing that tubercles are never found in the subarachnoid tissues without having given rise to some cerebral symptoms.

About a year later this very point was raised in a trial for murder.

Harriet Staunton, the wife of one of the prisoners, had died in a state of neglect and emaciation, which led to suspicions that she had been starved or poisoned. The medical men who made the post-mortem examination expressed it as their opinion that death had been due to deprivation of food, and yet stated that they had discovered in the membranes of the brain bodies which they believed to be tubercles, but to which they attached no importance. What were the exact appearances is uncertain, for one report speaks of "small patches of rough, millet-seed like deposit in the meshes of the pia mater," and another of "a small, recent patch of tubercular deposit upon the arachnoid membrane on the upper part of one hemisphere, about the size of a fourpenny piece." The husband and three other persons were convicted of murder. But before the time fixed for their execution the leading pathologists of London addressed a memorial to the Home Secretary and the lives of the prisoners were spared. If there really were tubercles in the meninges, their presence ought certainly to have been regarded as an indication that the immediate cause of the woman's death was disease. But it is to be observed that a similar conclusion would not necessarily be tenable if applied to a small "solitary" tubercle growing in the brain substance even though it should be adherent to the under surface of the pia mater. I have already, at p. 540, mentioned that such tubercles are sometimes, though very rarely, found in the bodies of those who have had no cerebral symptoms, but have died of some other disease.

The reader may perhaps be disposed to doubt whether tubercles that are only discovered by the aid of the microscope really possess the importance attributed to them in the last paragraph; but, just as in a case of concussion of the brain the presence of obvious ecchymoses of one or two convolutions (which cannot be regarded as themselves the cause of the fatal symptoms) is nevertheless of the highest significance as showing that the injury was sufficiently severe to have produced extensive though invisible damage to the texture of the organ, so it would seem that the development of even the smallest tubercles in the pia mater is attended with changes in the cerebral tissues that are incompatible with the maintenance of life, although our knowledge of those changes themselves is as yet very imperfect. Rindfleisch speaks of the superficial layer of the cortex—patches of which often remain sticking to the pia mater when it is stripped off—as infiltrated with leucocytes, and Huguenin says that inflammatory products may even be found in the white substance of the hemisphere. Indeed, some one part of the brain is now and then found in a state of well-marked yellow or red softening. I have notes of five instances of this that have occurred at Guy's Hospital, and others are mentioned by Rindfleisch and Huguenin. Sometimes the softened part has been the island of Reil on one side, sometimes the temporo-sphenoidal lobe, or the front of a lateral half of the brain. In one case, examined by Dr. Goodhart, no granule masses could be detected in the softened parts, but "the tissue was very fatty and granular, and the nerve fibres seemed to have undergone destruction, scarcely any of them being visible." The "white softening" of the central parts described at p. 596 appears likewise to be attended with a mere disintegration of the neuroglial elements and of the nerve fibres, no exudation cells being discoverable. I may take this opportunity of remarking that in some cases no ventricular effusion occurs, and that the fornix and septum lucidum then retain their normal consistence. Certain pathologists have, therefore, supposed that white softening is a mere result of post-mortem maceration of the cerebral substance, but such an opinion is untenable.

Basal Meningitis Without Tubercles.—In cases in which the symptoms and course have been altogether like those of tubercular meningitis, the membranes at the base of the brain and in the Sylvian fissures are sometimes

found after death with well-marked signs of inflammation, but no evident tubercles. Six or seven cases of this kind are to be found in the records of Guy's Hospital, but in all of them, with one exception, the lungs (and often other viscera as well) contained miliary tubercles; in the one exceptional instance the bronchial glands were caseous. A sufficiently careful microscopical observation might perhaps have cleared up the difficulty, but even without this the changes in other parts seem to me to be conclusive as to the real nature of the disease. Such cases are exactly analogous with those of the so-called "pneumonic phthisis," in which characteristic tubercular lesions are present in the larynx, in the intestine, or elsewhere.

There is, however, a form of acute basal meningitis in which neither tubercles nor scrofulous changes of any kind are to be found in any part of the body. This disease is described by Huguénin as "*leptomeningitis infantum.*" He relates as a typical case that of a female child, aged eleven months, who died in the fourth day of an attack of measles, attended with convulsions and other cerebral symptoms. Flattening of the convolutions, injection of the choroid plexuses, distention of the ventricles and softening of the central parts were the only obvious morbid appearances, and I must confess that these would not have deterred me from attributing the affection of the brain to the measles, but the microscope showed that leucocytes were present everywhere in the pia mater, especially at the base, though not in such numbers as to render the membrane cloudy or opaque.

A striking instance occurred at Guy's Hospital in 1859, when I was clinical clerk to the late Dr. Barlow. A boy, aged nine and a half years, died after an illness of twelve days' duration, which began with intense headache and ran its course with convulsions, grinding of the teeth, strabismus, and coma. That the disease was tubercular meningitis was doubted by no one who saw the child, and when the skull was opened the brain looked flattened, as if by effusion; but, except that its tissue was soft and that there seemed to be a slight increase of the fluid in the ventricles, no morbid changes were discovered. There were no tubercles in other organs. The real pathology of such cases appears to me to be still doubtful, but they make it necessary that one should always be cautious in asserting positively that a patient is suffering from tubercular meningitis, and perhaps they should especially deter one from giving an absolutely unfavorable prognosis.

Ætiology.—The causes of tubercular meningitis are those of scrofulous affections in general. Impure air, want of exercise, scanty food, are among the chief of them. An inherited predisposition is an important factor in its ætiology; it often attacks in succession several children of the same parents at about the same ages. When this is the case one is apt to suppose that there must be a special morbid susceptibility of the nervous centres, but it is to be borne in mind that, although clinically the cerebral symptoms mask all others, yet the disease is almost always a general tuberculosis. This fact also seems to render it very doubtful whether emotional excitement, overstudy, mental shocks, alcoholic intoxication, or blows upon the head can be capable of giving rise to the disease; at least it would seem that they can only act indirectly by lowering the general health, and not directly by disturbing the functions of the brain. Huguenin alludes to two cases in which intense emotional depression was followed by tubercular meningitis; in one there was an interval of fourteen days before the complaint set in. The patient had before enjoyed tolerably good health, but at the post-mortem examination it was found that there was latent pulmonary phthisis of old date, and that the lungs also contained miliary tubercles. Several years ago, a little boy who was in one of the surgical wards at Guy's Hospital suffering under disease of the hip joint was placed under chloroform; soon

afterward he was seized with sickness, which caused no alarm, but next day he was very ill, and he at length became comatose and died at the end of a fortnight; tubercular meningitis was found to be the cause of death. This was no doubt an accidental coincidence, and so was another case, that of a policeman, who attributed his illness to over-fatigue in attending a review in Hyde Park in June, 1860, but who afterward admitted that he had previously been complaining of pain in the head. Three children have died in Guy's Hospital in whom the exciting cause of the disease has been supposed to be a blow or fall upon the head from one to three months before. Thus, unless I am wrong in rejecting the doctrine which would refer tubercular meningitis to infection of the blood by absorption of caseous matters, I can only conclude that we are as yet ignorant of its having any special exciting cause.

Sex and Age.—It is a curious fact that the disease is much more frequent in males than in females. At Guy's Hospital the proportion has been as eighty to thirty-seven, and it does not seem to have varied very much at different periods of life, although Huguenin says that below fifteen years of age the preponderance of males is much more marked than in adults. The relative frequency with which persons of different ages are attacked is not yet accurately known. There are no hospitals to which children and adults are brought in numbers corresponding with their ratios to the population as a whole, and until post-mortem examinations become universal the Registrar-General's "*Reports*" will fail to do justice to the liability of grown-up persons to tubercular meningitis. It is stated by all writers to be much less common in infants under two years than in older children, but Guersant met with one case in an infant only six weeks old. Three cases have occurred since 1854 at Guy's Hospital in infants aged six months, ten months, and one year respectively. Of ages between two and four years there have been fourteen cases; between five and seven and a half, nine cases; between eight and ten, twelve cases; between eleven and fifteen, eleven cases; between sixteen and twenty, sixteen cases; altogether sixty-five cases in persons under twenty. Between twenty-one and thirty there have been thirty-one cases; between thirty-one and forty, fourteen cases; between forty-one and fifty, eleven cases; between fifty-one and fifty-six, three cases; altogether fifty-nine cases in persons above twenty years of age.

Clinical Course.—The symptoms of tubercular meningitis are not essentially different in patients at different ages, but as the best writers have based their descriptions mainly upon observations of cases occurring at an early period of life, and as such cases present certain minor clinical peculiarities, I will first limit my remarks to the disease as it is seen in *children*. Afterward I shall point out what features undergo modifications in adults.

In the first place, before any definite indications of cerebral mischief develop themselves, a general failure of health is often observed, which may last for several weeks, or even for two or three months. The symptoms which are manifested during this period are termed "premonitory" or "prodromal." In the well-known work of Rilliet and Barthez on the diseases of children, an admirable sketch of them is given, which has afforded materials for all subsequent writers. Probably they may be due to two different causes, sometimes to a scanty early formation of tubercles in the lungs or other organs, which is afterward followed by a more abundant crop, so that at the autopsy a few of them are found caseating while the rest are still gray; sometimes to the slow progress of a chronic scrofulous affection of the mesenteric or bronchial glands. Foremost among them is emaciation; the limbs waste and lose their roundness, the ribs and the bony processes stick out beneath the skin, the muscles feel soft, the skin is lax and flabby. The cheeks often retain their plumpness of outline, so that

the loss of flesh is first observed by the nurse who dresses and undresses the child; but the face becomes pale, the eyes are dull, and there is a want of animation in the countenance. The appetite is diminished or capricious; there is no thirst. The bowels are disordered, being generally constipated, but with intervals of diarrhœa; the evacuations may be pale and offensive. The disposition and the temper often become changed. The child is dull, apathetic, and slow in its movements; it is easily fatigued by lessons, and quickly tired of toys. It may be fretful and peevish, or it may exhibit a morbid tenderness and affection, breaking off in the midst of a game to throw itself into the mother's arms, and bursting into tears if she should check the unexpected display of emotion. Headache is sometimes present, but more frequently the complaint is rather of being sleepy and tired and wanting to lie down. At night it is restless, lying with the eyes half closed, rousing at the slightest noise, and being unable to sleep with a candle in the room; or it may grind its teeth, and start or cry out with alarm at its dreams. Whether fever is commonly present is doubtful. Dr. Gee speaks of feverishness in the evening, but he adds that he knows of no thermometric observations, and that the most careful mother often fails to observe any heat of skin until the period of prodromata has passed. He suggests that the presence of a remittent elevation of temperature may perhaps serve to indicate that the formation of tubercles has actually commenced, and that the child is not merely in a state of depressed health such as might be antecedent to their development; but the difficulty seems to me to be that trifling disorders of the stomach and bowels so easily excite feverishness in children.

I have already implied that premonitory symptoms do not always occur, but Dr. Gee says that among twenty-six cases collected by him there were only two in which they were not noticed. Their duration is very variable, sometimes not more than a fortnight but generally longer, and now and then even as much as four or six months. They may subside, and the child's health appear to improve, before the disease breaks out in a characteristic form. What is peculiar to the actual "invasion" of tubercular meningitis is that its further course is limited. In marked contrast to the uncertain length of the prodromata, I believe that life is never prolonged much beyond twenty-one days from the occurrence of the earliest invasion symptom. The only apparent exception to this rule that I know of is in cases in which one or more "solitary" tubercles (of the kind described at p. 535) had been developing themselves in the brain before the commencement of the meningeal affection.

The symptom that first excites serious alarm is most often the occurrence of repeated vomiting. Sometimes the child is sick only when it takes food, sometimes it brings up bilious matters even though it may have swallowed nothing. The sickness generally lasts for two or three days only, but it may go on for a week. If it once ceases for twenty-four hours, it seldom returns. In some cases there is no vomiting for the first day or two; now and then it is altogether absent. The symptom which then most frequently ushers in the disease is a convulsive seizure, more or less completely epileptiform in character. In one instance mentioned by Dr. Gee there was general rigidity which recurred several times; in another the attack took the form of temporary unconsciousness. In a single one of his cases (twenty-five in number) the invasion was marked neither by vomiting nor by convulsions, but by a rather sudden increase of the headache, drowsiness, and wasting which had existed during the premonitory stage.

Severe pain in the head is now almost invariably present; the child keeps its hands pressed against the forehead, or may go on rubbing the scalp, first in one place, then in another. From time to time, when exacer-

bations occur, he perhaps calls out, "Oh, my head!" Trousseau lays stress on a peculiar inarticulate "hydrocephalic cry," which is sometimes heard from the very beginning of the disease, sometimes only toward the end; he describes it as a single, sharp, loud sound, like that of a person exposed to some sudden danger; it may be repeated every hour, or every five minutes, for several days together. Rilliet, on the other hand, declares that this symptom is neither frequent nor special to meningitis, and Dr. Gee agrees with him. For several days there may be no marked impairment of the intelligence; the child, perhaps, continues to take part in rational conversation, but his answers may be rather slow and his memory and power of perception somewhat impaired. He is apt to lie in a drowsy state, half asleep, with his eyes staring vacantly, and then he may go on talking senselessly to himself, or repeating some particular phrase over and over again, or singing, whistling, shouting. The pupils are often sluggish, and very commonly one of them is larger than the other. A tendency to squint is another early symptom of very frequent occurrence. Trousseau relates one case in which there was a transient unilateral hemiopia; the child was sitting near a window when it called out, "Oh, mamma, look at that little boy; he has only half a blouse and half a face!"

Such are, as a rule, the symptoms of tubercular meningitis for, perhaps, eight days after its invasion. During this period—which is sometimes not very aptly called the "stage of brain irritation"—there is more or less fever, the evening temperature rising, perhaps, to 102° or 103°, while in the mornings it may be 101° or 100°, or even not above normal. The pulse may be a little quickened, or natural, or slower than natural; sometimes it is irregular, being easily made more rapid by the slightest excitement, or even becoming so without any obvious cause. The frequency of the respirations is generally but little altered. Constipation of the bowels is almost always present. The tongue may be furred, but it is sometimes almost clean.

When about eight days have passed, the condition of the little patient undergoes a change, which now and then appears to be almost sudden, but more often takes place gradually; and its most striking feature is a loss of consciousness; and the period which follows has been called the "stage of pressure." The child now ceases to take notice of anything that goes on in the room. It commonly lies on one side, curled up with the knees drawn close to the abdomen, and the hands folded over the genital organs. Sometimes the head is drawn backward and the muscles of the nape of the neck may then be felt to be hard and rigid. It may keep grinding its teeth every few minutes, making a noise which has a most disagreeable, jarring effect upon one's ears. The pupils now become dilated and insensible. One or more of the cranial nerves may be paralyzed; the third, for instance, so that the eyelid drops; or more rarely the facial nerve. There may be loss of power in the limbs. Tickling the soles of the feet may cause only one leg to be drawn up, the other remaining extended. The evacuations are often passed into the bed without the patient's knowledge.

The order in which the various symptoms make their appearance is uncertain; some of them may commence during the earlier period of the disease, before coma sets in. This is particularly the case with certain changes in the patient's aspect, which have now to be mentioned. One is the presence of a frown upon the brows, and deep lines often seem to be drawn around the nose and mouth. There is generally flushing of one cheek, sometimes of both of them; or the whole of the face may be suffused with blood. If the countenance is pale, it may, perhaps, become reddened when the child is disturbed, or when anything is given to it to drink. So, also, any part of the body which has been pressed upon shows a marked injection of its

capillary blood vessels. A particular instance of this was made into a leading symptom of tubercular meningitis by Trousseau, under the name of the *tache cérébrale*. He pointed out that if one draws one's finger nail gently over the patient's thigh, or abdomen, or face, a bright red line is produced, and that this differs from the effect of an equally slight scratch in a healthy person, by appearing earlier (within thirty seconds), by lasting longer (eight, ten, or fifteen minutes), and by being broader and of a deeper color. With regard to the diagnostic value of this sign, I shall have some remarks to make further on.

The ophthalmoscope may show ischæmia of the optic discs, or descending neuro-retinitis, or both in succession. Dr. Allbutt found some affection of the retina in twenty-nine out of thirty-eight cases examined by him.

Another symptom which may be observed in most cases at this period is retraction of the abdominal walls. Vomiting is generally absent, and the bowels may still remain obstinately constipated; but for some unknown reason the intestines no longer contain the usual quantity of gas; and the belly, therefore, becomes deeply hollowed, or (to use a common expression) "boat-shaped," the rib cartilages, the iliac crests, and the pubic symphysis appearing unduly prominent. The tongue may now be red and dry, but it often still remains moist. The temperature seldom rises above 101°; Dr. Gee remarks that it may for days together remain at between 96° and 98°. Thus, if the case is advanced when the child is first brought to one, the disease may appear altogether non-febrile. The pulse during this period is generally infrequent, 60 or even 50 per minute; the number of its beats may each day be less than before; it is still apt to be irregular and unequal in force. A similar irregularity and inequality of the respiratory movements are very commonly present; the child, perhaps, breathes rapidly three or four times in succession, and then the chest may remain motionless for some little time. Trousseau laid great stress on this symptom, and has recorded a case in which the breath was held for as long as fifty-seven seconds. Typical "Cheyne-Stokes respiration" is sometimes observed.

The "stage of pressure" may continue with but little alteration until it terminates in the patient's death; this being, perhaps, immediately preceded by a convulsive seizure. But in certain cases the symptoms during the last twenty-four or forty-eight hours are to some extent peculiar. One remarkable change is that there may be a brief return of consciousness for a short time before the end. I once saw a striking instance of this myself. Dr. West relates how a girl, aged seven, who had been in a state of stupor for six days, and profoundly comatose for two days, became conscious, swallowed some drink, spoke sensibly, and said she knew her father; in the course of an hour and a half, however, she became worse again, and a little later she died. The pulse often becomes rapid during the last two or three days; and as Dr. Gee points out, the temperature may steadily rise until it is above 107°. But in some cases the beats of the heart remain infrequent up to the time of death; and the temperature even in the rectum may fall, until it is very low indeed. In one case, three days before the fatal termination, the thermometer registered 97.8° and 96°; next day the highest temperature was 96.2°, the lowest 93°; the day after they were 82.8° and 82.1° respectively; and on the day of death 80.5° and 79.4°. In other cases the face and limbs are livid and cold, and covered with a clammy sweat, while the thermometer shows that fever is still present. Toward the last a peculiar fetid, earthy smell is often perceptible, which I do not remember to have noticed in any other disease. I am surprised to find that Rilliet and Barthez, after alluding to Whytt having mentioned this symptom, go on to say that they have not themselves recognized it.

Tubercular Meningitis in Adults.—The onset is comparatively seldom

preceded by marked prodromal symptoms in older persons. As I have already observed, it is often secondary to phthisis, and, when this is the case, the pulmonary affection is, of course, rightly regarded as the cause of any general failure of health that may have been noticed. Then, again, few grown-up persons are watched as carefully as children are by their parents and nurses; and in adult life a loss of flesh may be due to so many other conditions that tubercular meningitis is very unlikely to be thought of until its symptoms are actually developed. But apart from all these considerations, I must state it as my impression that most of the patients whom I have seen have been fairly well nourished at the time of death. I have no recollection of having ever observed a very marked degree of wasting, unless when it was obviously referable to some co-existing visceral disease; and in the case of Harriet Staunton, already referred to at p. 598, I should have been reluctant to regard the extreme emaciation, which must have been going on for several weeks, as dependent upon the presence of meningeal tubercles. The actual invasion is, I believe, far less often ushered in suddenly by vomiting in adults than in children. Dr. Gee, indeed, speaks of vomiting as an early symptom, occurring in almost every instance; but neither of the last two patients whom I saw in consultation had any sickness, at least up to a time when other characteristic symptoms had developed themselves, so that the nature of the disease could hardly be misunderstood. What particularly impressed the fact on my mind was that in each case the medical man in attendance had been mainly led to a diagnosis of enteric fever by finding that the stomach did not reject its contents. Again, I do not find that in a grown-up person an epileptiform seizure is of frequent occurrence as a mere invasion symptom, followed by the slow development of the disease in regular stages. In adults the disease seems generally to begin very gradually and insidiously. On the other hand, we have had at Guy's Hospital more than one case in which the sudden occurrence of convulsions has been the precursor of a fatal termination within a day or two, and in which an autopsy has shown that the disease was tubercular meningitis. It is not at all uncommon for death to take place after an illness of twenty-four or forty-eight hours only. In 1868, a man, aged thirty-two, a patient of Dr. Wilks, died within two days of having been about his business, which was that of a draper; on admission he was so restless that he had to be held down in bed, but he quickly became comatose. In one instance there was violent delirium as late as two days before death.

In some cases of tubercular meningitis, in adults, the first symptom is a well-marked local paralysis; I do not know that the same thing ever occurs in children. In 1871, a woman, discharged from a surgical ward because she had advanced phthisis, came the same morning to the taking-in room to seek readmission as a medical patient. No symptoms of cerebral disease had been observed, but I found that she had paralysis of the left facial nerve, an affection which must have developed itself within a few hours. I therefore took her into the clinical ward; she seemed rather stupid, but was intelligent enough to be able to tell us that she could not hear the ticking of a watch until it was placed in contact with the left ear, whereas on the right side her hearing was perfect. However, she quickly became drowsy, and then comatose and unable to swallow; and at the end of six days she died. At the autopsy I failed to discover any special affection of the facial nerve, either in the petrous bone or elsewhere. So far as I know, Huguenin is the only observer who has attempted to trace the affections of special cranial nerves occurring in tubercular meningitis to definite local changes. In one instance, in which the face was paralyzed, he found the portio dura thinned, of a yellow color, and covered with a large quantity of pus; cellular

elements were present in numbers in the nerve sheath, but the fibres appeared to be intact. In another similar case, however, no change could be found in the nerve. In one instance, in which there had been convergent strabismus, the abducens was greatly diseased, diffuse suppuration having occurred between its fibres. Similar appearances were twice discovered in the third nerve, when the muscles supplied by it had been paralyzed during life; but in some other cases, in which the same symptoms had been incompletely developed, the nerve seemed to be normal.

Lastly, tubercular meningitis may have hemiplegia for its earliest, and even for its principal, symptom. Three instances of this have been observed at Guy's Hospital within the last few years. One patient was a man, aged thirty-three, who was admitted on account of an abdominal tumor, which proved afterward to be a tuberculous omentum. Some days before his death he was attacked with loss of power in the left arm and leg; he was sensible to the last. In the other two cases the right limbs were affected, and, as the superficial seat of the lesion might have led one to anticipate, the paralysis was accompanied by well-marked aphasia. One patient was a woman, aged twenty-six, who came under Dr. Wilks's care in 1867; I well remember that, until the real nature of the affection was revealed at the autopsy, there was not the slightest suspicion of its being due to any cause other than some disease of the Sylvian artery. The other, also a woman, aged forty-one, had been attending as an out-patient for phthisis, when she was seized with right hemiplegia and loss of speech; afterward she became semi-delirious, and her paralysis changed sides. In none of these three cases was any well-marked change found in Broca's convolution or in the adjacent parts of the brain; but in a boy, aged nine, in whom right hemiplegia and aphasia were combined with the more ordinary symptoms of tubercular meningitis, there is said to have been red softening of the left third frontal, and of the inner ends of the two left ascending (or central) gyri. Huguenin relates three instances in which paralysis of the right limbs and loss of speech were the chief symptoms; in two of them many tubercles were present in the left Sylvian fissure, but none in the opposite one, and in the third case the pia mater in the left fissure was more thickened than anywhere else. In one of the cases in which there was red softening of the left island of Reil and of Broca's convolution, the first symptom after headache was speechlessness, but this lasted only about seven minutes, and for two days afterward the patient (a man, aged fifty) was able to go to business as usual. One can only suppose that the loss of speech was due to functional disturbance of the brain; or, in other words, that it was a neurosis, and analogous to that which has been observed in other cases, referred to at p. 516.

Huguenin raises the not very important question whether it is generally possible from the symptoms to determine the presence of ventricular effusion, or the extent to which tubercles are developed on the convexity of the brain and at the base respectively. His statements and the observations which have been made at Guy's Hospital seem to show that when there are no inflammatory changes in addition to the tubercles there is apt to be no persistent coma. Violent delirium is sometimes associated with the presence of very numerous tubercles between the convolutions of the upper surface of the brain. Choked discs are probably an indication of a general increase of pressure within the cranial cavity, whereas neuro-retinitis points more definitely to the existence of an inflammatory process at the base of the brain, in the neighborhood of the optic tracts or nerves. In one case in which there was impairment of power in all four limbs, Huguenin found changes in the superficial layers of the crura cerebri. The extension of meningitis to the spinal canal does not seem to give rise to any very marked

special symptoms. It probably gives rise to a rigidity of the muscles of the neck, which (in children, at any rate) is very common, for this is one of the chief symptoms of an epidemic form of meningitis which, until recently, was supposed to be peculiar in affecting the spinal as well as the cerebral membranes; and sometimes the occurrence of painful cramps or spasms in the limbs may be attributed to the same cause.

Diagnosis.—This may either be very easy or very difficult. During the first few days of a case which is to last two or three weeks one is often unable to speak positively, but there is seldom much uncertainty when its fatal character is once fully developed. All writers lay stress on the importance of distinguishing it from enteric fever, and the fact is indisputable that many cases, which for a week or ten days are supposed to be examples of that disease, afterward become attended with such well-marked cerebral symptoms that they are forthwith transferred to the category of tubercular meningitis. Thus Rilliet and Barthez describe a special form as having a *début typhoide*. But although to the clinical observer the meningeal affection thus masks all the other features of the case, yet the pathologist almost always finds it to be only a part of a general miliary tuberculosis, which in reality constituted the patient's illness from its commencement, and to which the early febrile symptoms were due. In our reports at Guy's Hospital I find only two recorded instances in which the first diagnosis was that of fever, and in which tubercles were found in the meninges and nowhere else. Sometimes, indeed, the opposite error is committed, enteric fever being attended with strabismus and irregularity of pupils, vomiting and constipation, as well as with headache, delirium and coma, so as to be taken for meningitis. So far as I know, the *tache cérébrale* of Trousseau affords very little help in the diagnosis of cases that would otherwise be doubtful. Dr. Gee says that in tubercular meningitis he has often been unable to bring it out except by using an unfairly hard stroke, such as would cause reddening of the skin in any child; and I have seen instances in which this symptom has been present, but which a review of the whole course of the disease, after the recovery of the patient, has led to the conclusion that it was enteric fever. The clinical value of ophthalmoscopic changes in the optic discs is still somewhat doubtful. It is certain that a normal state of the retinæ is no proof of the absence of tubercular meningitis, but I believe that the time has not yet arrived for a dogmatic expression of opinion as to the positive significance of ischæmia (or even of retinitis) as between that disease and some less severe affection of the brain, such as might be attended with great vascular congestion of its tissue. One appearance, indeed, is conclusive, namely, the presence of tubercles in the choroid. It is true that they belong not to the meningeal affection itself, but rather to a general acute tuberculosis, but this fact in no degree diminishes their diagnostic importance. For, on the one hand, the membranes of the brain are very seldom the sole seat of miliary tubercles, and, on the other hand, whenever there are any tubercles in the membranes the case always assumes clinically the aspect of a cerebral disease, even though they may be infinitely more numerous elsewhere. In fact, in all cases of suspected tubular meningitis one should carefully search the lungs and other parts for the very slight indications of acute tuberculosis which they sometimes yield; one ought even to examine the testes, the lymphatic glands, and other organs, for chronic lesions of the same nature.

Another morbid state, that formerly used often to be mistaken for tubercular meningitis, is one which is of frequent occurrence in young children, and which has been rendered classical by the descriptions given of it by Marshall Hall (1825), Abercrombie (1828), and Gooch (1829). The first of these writers called it "the hydrocephaloid disease;" Sir Thomas

Watson gave it the name of "spurious hydrocephalus;" and Dr. Gooch stated that he had "invariably found it attributed to, and treated as, congestion or inflammation of the brain." But at the present day I do not think that a skilled practitioner is likely to fall into the error of diagnosis which fifty years ago was thus universally committed; and at any rate the mistake is not now likely to be attended with any serious results. Among the chief causes of the affection was one to which little children are no longer liable, namely, the free withdrawal of blood, under medical authority, in spite of existing depression of the vital process. A case related by Dr. Hall is that of a girl, aged two and three-quarter years, who had had sixteen leeches applied for an attack of influenza; and he states that all his patients were in a state of exhaustion before they were attacked by the cerebral symptoms; many of them had had protracted diarrhœa after weaning. On the other hand, Dr. Gooch says that in most of his cases there had been no previous illness; but perhaps he was hardly keen enough in the appreciation of the gradual effects of insufficient or improper food. The child's aspect is characteristic; it lies on its nurse's lap, unwilling to raise its head, drowsy or even comatose, with sunken, half-closed eyes, dilated, insensible pupils, irregular and sighing respiration. The face is pale and the skin cold. Dr. Gooch, indeed, speaks of a slight and transient flush as being sometimes present, and Dr. Hall, of an early stage in which the little patient is irritable, restless, feverish; but such symptoms very seldom, if ever, last long enough to mislead a careful observer into the diagnosis of meningitis. Depression of the fontanelle is an important symptom of exhaustion in children, although it seems formerly to have escaped notice. The proper treatment is to give ammonia and brandy, but above all, to take care that suitable food is supplied. For an infant recently weaned a wet nurse should generally be procured; and a return to the breast is often advisable, even when the child is some months old and has long been fed with the bottle. At least, it ought to have asses' milk, or goats' milk, if cows' milk should appear not to be readily digested. To prescribe leeches and calomel would, of course, be fatal; but this kind of practice is now obsolete in tubercular meningitis.

At Guy's Hospital we have had cases which during life were supposed to be examples of mania, delirium tremens, or epilepsy, and in which the disease has turned out to be tubercular meningitis. Huguenin remarks upon the possibility of mistaking the latter disease for uræmia, when a chronic renal affection, in reality tuberculous, is attributed to morbus Brightii. A curious case occurred some years ago at Guy's, that of a painter, who came saying that he had "lead colic," and that his bowels had not been open for a fortnight. In the evening, after his admission, he had convulsions and became insensible, and on the morrow he died. There was strumous disease of two of the lower dorsal vertebræ, with a large abscess in front of the spine; and the membranes of the brain and cord showed the characteristic appearance of tubercular meningitis. The same disease has been found on post-mortem examination in two cases of boys admitted into the surgical wards for symptoms supposed due to vesical calculi; no cause could be discovered for the irritability of bladder from which they had been suffering before any obvious signs of cerebral mischief appeared. A very similar case occurred in a woman who was taken into a medical ward for trouble with her bladder; but, in addition to the affection of the membranes of the brain, there was a tumor in the spinal cord, and this may possibly have given rise to the vesical symptoms.

In adults, however, the most serious error of all is to mistake tubercular meningitis for hysteria. I shall never forget a case which I saw many years ago, that of a clergyman's daughter who had been unfortunate in love, and

who was attacked with delirium; until within a few hours of her death no one was able to speak confidently as to the nature of the disease. Two similar instances have occurred at Guy's Hospital. One patient, a woman, aged twenty-five, had long been "odd in her mind and scarcely to be trusted;" she was attending as an out-patient, with hysterical symptoms, when she sought admission, and died three days later. There were several yellow tubercles, of the size of peas, in the brain, as well as the usual appearances of tubercular meningitis.

Prognosis.—The reason why it is of so great consequence that one should not mistake tubercular meningitis for hysteria or enteric fever is that the prognosis of these affections is so different. Tubercular meningitis is one of the most fatal of all diseases. Rilliet has, indeed, recorded an instance of recovery, in which the child died five and a half years afterward, of a second attack, and at the autopsy the remains of the former mischief at the base of the brain were clearly recognized; and a similar case is mentioned by Trousseau. Among all the post-mortem examinations which I have made, I have only twice found the membranes on the under surface of the encephalon matted together in such a way as to suggest that inflammation had formerly occurred there. In neither instance was any caseous matter present; so that, if meningitis was really the cause of the adhesions, it may probably have been a simple, rather than the tubercular, form of the affection.

However, it is by no means a very rare circumstance for recovery to take place in cases in which tubercular meningitis had been diagnosed more or less positively, and for the nature of the disease to remain uncertain, as it must necessarily remain in such cases, unless an accident should lead to an autopsy being made at a future time. Most observers are disposed to think that these are generally instances of enteric fever complicated by cerebral symptoms. But it is surely fair to remark that in some fatal cases the usual morbid changes are not discoverable, while the absence of tubercles in the other organs deprives us of all warrant for the supposition that the microscope might reveal their presence in the membranes, even in the most minute quantity. I would not venture to assert that the disease is then merely a neurosis. But even if some as yet unrecognized lesion should hereafter be demonstrated, one might at least suppose that it need not necessarily prove fatal. How closely a case which terminates in recovery may resemble tubercular meningitis is strikingly illustrated by a clinical history which we owe to Dr. West. A child, aged three years and a half, a member of a phthisical family, was attacked by a disease which ran the ordinary course of acute hydrocephalus, unchecked by the customary treatment. Convulsions took place, coma succeeded them, deglutition was very difficult, the pupils were dilated and almost motionless, the pulse was very feeble and very frequent, and everything portended a speedy death. A younger brother had died a year before, of the same disease. Food was still given, as the power of swallowing was not entirely lost, and ammonia and ether were administered, and after a time quinine. For days the child remained unconscious, but at length she began to raise her hands to steady the cup that was put to her lips. Next she recovered her sight, after some weeks she became able to speak, and after many months she began to walk with a tottering step. Three years afterward, although her intellect was not defective, she still had a vacant smile, and had never regained flesh, nor recovered the look of health; her gait, also, remained unsteady. Whatever the real nature of this case may have been, one cannot be wrong in deducing from it the necessity of being very cautious in asserting that it is impossible for a patient to get over an attack of what appears to be tubercular meningitis. However confident we may be in our diagnosis, and however threatening may be the symptoms, our opinion should be given with some reserve.

The diagnosis between tubercular meningitis and other organic affections of the brain is of comparatively little practical importance. The prognosis is scarcely, if at all, affected by it; and there are very few cases in which the treatment would be different. The points which distinguish this kind of inflammation of the membranes of the brain from the other kinds will be indicated hereafter when they have been described. I believe that many cases have been recorded as examples of sporadic "cerebro-spinal fever" in England which have really been tubercular. Particular cases of abscess or tumor of the brain may sometimes resemble those of meningitis very closely; but it can hardly be said that a mistake in this direction is of serious consequence. The single exception is that in grown-up patients one must always be alive to the possibility that syphilis may be the cause of the most varied cerebral symptoms.

The *treatment* of tubercular meningitis will be discussed when the simple forms of the disease shall have been described.

2. Epidemic Meningitis or Cerebro-Spinal Fever.—From the earliest years of the present century there have been recorded, from time to time, in various parts of the northern hemisphere, epidemics of a disease characterized anatomically by inflammation of the membranes of the brain and cord, and clinically by fever, various eruptions, and a number of cerebral and spinal symptoms, especially rigidity of the neck, or of the whole vertebral column. So striking is the symptom last mentioned that in Germany it has given to the affection the popular names of "Genickkrampf" and "Nackenstarre." In medical works it has hitherto been called "epidemic cerebro-spinal meningitis," or "cerebro-spinal fever." But I think that it is preferable to term it simply "epidemic meningitis," since the epithet "cerebro-spinal" is likely to encourage the notion that an extension of the inflammatory process to the membranes round the cord is more or less distinctive of it as compared with the other forms of meningitis.

The first well-ascertained epidemic of this disease seems to have been in 1805, at Geneva. In 1806 it appeared in the United States and continued to prevail there for ten years. During this time, and indeed throughout the past half of the century, it was observed in different towns of France and of Italy, in Algeria, Spain, Denmark, etc. In 1854, and for seven years afterward, it raged in Sweden, destroying more than 4000 persons in that country. From 1861 to 1864 it showed itself in various parts of the United States. In 1863 it broke out in Germany; the northeastern provinces of Prussia were the first to suffer from it; but within the next year or two it appeared in Erlangen, in Nuremburg, and in other South German towns, and in the country districts of Franconia. From that time it has never ceased to show itself at intervals of a few months or longer, now in one part of the German Empire, now in another. Writing in 1874, Ziemssen said that it seemed to be naturalized.

The British islands have hitherto been remarkably free from this disease. In 1846 it appeared in many of the workhouses of Ireland; and in 1866–1868 a very fatal type of it prevailed in Dublin, and to some extent in other parts of the country. Scotland, I believe, has been altogether spared by it. And in England only a very few isolated and small epidemics of it have been observed in certain provincial towns and villages. Its occurrence in London has not yet, I think, been satisfactorily established. A few cases have, from time to time, been recorded as sporadic examples of it by writers who seem to have thought that the fact that a meningitis was cerebro-spinal was of itself sufficient to justify a presumption that it was related to the epidemic disease. But now that we know that all forms of inflammation of the base of the brain are apt to extend to the cord, this conclusion is, of course, un-

tenable. And the symptoms observed in the various cases in question have not seemed to me to support such an hypothesis.

Anatomy.—The morbid anatomy of epidemic meningitis has, in fact, little or nothing to distinguish it from that of the sporadic form of the disease. Pus and lymph are found both at the base and on the convexity of the brain, especially between the pons and the chiasma, along the large vessels, and in the various depressions and furrows on its surface. It rarely happens that the hemispheres are uniformly covered by it. In the spinal canal the exudation is generally most abundant at the lower part, and on the posterior surface of the cord, having perhaps accumulated there by gravitation. The ventricles of the brain mostly contain turbid serum or pus; the choroid plexuses and even the whole of the ependyma may be coated with puriform lymph; in one case Fronmüller is said to have found the central canal of the cord dilated and full of pus. Punctiform hemorrhages or small spots of softening may be seen in the cerebral substance; or it may be simply œdematous and flattened. The cord presents similar changes, but less marked.

Among the appearances presented by other organs are congestion and œdema of the lungs (especially of their lower lobes), engorgement of the liver and spleen (the spleen, however, is not very often much enlarged), a relaxed state of the heart, congestion of the kidneys with fatty epithelium and fibrinous casts in the tubes, and a fine granular degeneration of the fibres of the voluntary muscles in general, and especially of those which lie along the spine. Rigor mortis is said to be of long duration. The cadaveric stainings of the surface appear early, and are not always limited to the dependent parts of the body.

The pericardium and the pleuræ have sometimes been found ecchymosed, or even lined with puriform exudation. Ziemssen once saw the large intestine inflamed, as in dysentery. The joints often contain pus, and sometimes there are scattered abscesses in the connective tissues and muscles. All these changes must be regarded as complications dependent on a secondary blood poisoning.

Symptoms.—As a rule, cerebro-spinal fever sets in suddenly; the patient may be at work, or (if a child) at play or at school, when he is seized with shivering and violent pain in the head, and feels so ill that he is obliged to go to bed at once. But sometimes—among Ziemssen's cases in five out of forty-three—there are slight prodromata, consisting of headache, malaise, nausea, loss of appetite and wandering pains. In several instances these passed off, and there was an interval, in which the patient felt perfectly well, before the disease began with its usual violence.

Vomiting is almost always an early symptom, being repeated whenever an attempt is made to sit up. After a day or two it generally ceases, but the headache mostly continues throughout the whole course of the disease, although it may sometimes subside for a time. It varies in character and in seat, being sometimes frontal, sometimes occipital, sometimes diffused over the whole of the head. Giddiness is often present with it. In cases of moderate severity the patient lies in a state of stupor, tossing restlessly about, but rousing when spoken to and trying to answer. He is often very irritable to light and sound. The pupils may at first be normal or contracted; ultimately they become dilated. In the more dangerous cases he quickly becomes delirious, with or without convulsions, or passes into a condition of insensibility. Even when he is deeply comatose he often still feels the pain in the head, and groans or cries out, or grasps the temples between his hands. Aphasia, hemiplegia, and other paralytic symptoms, are sometimes observed; but in the most severe cases of all their presence can hardly be determined.

The rigidity of the neck already referred to is scarcely ever entirely

absent, but is not commonly a marked symptom during the first day or two. It varies in degree, from a slight stiffness, noticed only when an attempt is made to bend the head forward, up to a forcible retraction, bringing the occiput almost to a right angle with the spine. Dr. Sanderson has suggested that it is due to a half voluntary effort on the part of the patient for the relief of pain in the muscles; but it may be present when there is no pain at all, either in the neck or in the back. It would seem, however, that the muscles below the occipital bone do not feel as hard as might be expected; for Dr. Sanderson could not detect any tightness of them so long as the head was thrown back, and Ziemssen probably means little more when he says that the tetanic spasm is "limited to the deeper muscles, the trapezii almost invariably escaping." In about half the cases this symptom is accompanied by a contraction of the extensor muscles of the dorsal and the lumbar vertebræ. Sometimes the back is arched so as to be in a state of opisthotonos, but more often it is simply straightened, or (as Ziemssen expresses it) in *orthotonus*. If an attempt is made to raise the patient, he either slips down to the foot of the bed without bending his back at all, or allows his body to be lifted a very little way, at the cost of so much pain that he is very soon put back into the recumbent posture. Almost always he lies on one side with his knees drawn up. He may then be nearly free from pain in the back, but sometimes its intensity is hardly at all affected by the position of the body; it is apt to be particularly severe in the sacrum. Pains in the limbs, and especially in the legs, are often complained of; some writers lay stress on the frequency of an acute pain in the knee. The joints may become hot, red, and painful, as is the case in other spinal affections. An extreme cutaneous hyperæsthesia is another common symptom; the patient, though he may be comatose, will perhaps scream out at the slightest touch, or even if his bed is shaken. Tetanic rigidity of the limbs is seldom present, and so also trismus is of infrequent occurrence.

The degree of fever in epidemic meningitis is exceedingly variable, and its course is very irregular, so that even those observers who lay most stress on the typical fever courses of many other specific diseases admit that nothing of the kind can be traced here. The temperature usually ranges from 100° to 103°, but it may fall and remain normal, or nearly so, for a day or two at a time; sometimes it rises to 105° or 107°, especially toward the last. The rate of the pulse may be natural or slightly increased; it is liable to frequent fluctuations; in bad cases it may be very rapid. The face is generally pale. The spleen is sometimes, but rarely, found enlarged. As a rule, the abdomen is retracted, but it may be greatly distended. Ziemssen observed several cases in which complaints were made of oppression at the epigastrium, of constriction of the chest, and even of severe paroxysms of dyspnœa. An abundant secretion of urine has been noticed by several German physicians, even when the fever was high; in exceptional cases a small quantity of albumen or of sugar has been present.

An important symptom in this, as compared with other forms of meningitis, is the occurrence of certain cutaneous eruptions. Chief among them is herpes of the face. This generally begins on the lips and spreads to the cheek, nose, ear, eyelids; it is often bilateral; it may cover the whole side of the face, and thus it is far more marked than in any other acute disease. It first appears between the third and the sixth day, but fresh outbreaks of it may take place as late as the sixth or the seventh week. It may be seen even on the trunk or on the limbs; those parts, however, are more often the seat of a roseolous or an erythematous rash, an urticaria, or a diffused and extensive purpura. There appears to be nothing specific in the characters of any of these eruptions; they are often mixed together in the same case. Ziemssen lays stress on the symmetry with which they are distributed on opposite

sides of the median plane—herpes on each wrist, urticaria on each leg, or petechiæ on each shoulder. Dr. Collins, of Dublin, saw purpuric spots suppurate and scab over, so that they finally left pitted cicatrices. Sometimes large patches of hemorrhage coalesce and give a uniform black appearance to a considerable part of the body.

The organs of sight and hearing are affected in many cases of cerebro-spinal fever. There is often an intense conjunctivitis, attended with extreme chemosis. Ulceration of the cornea, irido-choroiditis, optic neuritis may each of them develop itself, leading to the usual consequences of these several lesions. Vision may be suddenly lost at an early period of the disease; in cases which recover, the patient is apt to be left more or less completely blind. The ears were found by Ziemssen to suffer in eight cases out of forty-two. Pain, tinnitus, impairment of hearing were generally complained of soon after the patient fell ill; they either passed off or ended in a partial or total deafness. Such symptoms sometimes depend upon suppuration of the tympanum, leading to perforation and discharge through the meatus. It has been suggested that in other instances they may be direct results of inflammation of the floor of the fourth ventricle, involving the *striæ acusticæ*, or that they may be due to the presence of purulent exudation about the seventh pair of nerves. Ziemssen, however, remarks that he has often found both these lesions in cases in which there was no affection of the auditory functions during life. In certain cases, on the other hand, in which deafness had been present, Heller discovered after death a suppurative process in the labyrinth, besides an infiltration of the portio mollis with pus. It then became a question whether these morbid changes were caused by an extension of mischief from the pia mater, or whether they began simultaneously with the meningitis. Heller was disposed to adopt the former opinion. It is curious that in cases of this kind the portio dura constantly escapes and that facial paralysis is not observed. Severe inflammation of the labyrinth usually leads to an absolute loss of hearing; and in most cases both ears are affected. The consequence is that the patient, if very young, never learns to speak. Even children two or three years old, who were able to talk before they fell ill with the meningitis, and whose articulation after their recovery was at first tolerably distinct, soon begin to lose the power of speech, and ultimately become unintelligible. Hirsch, indeed, says that there is sometimes an aphasia, as an immediate result of the cerebral affection; but this was never observed by Ziemssen. How important a part may sometimes be played by epidemic meningitis in the production of deaf-mutism is shown by the fact that in 1874 every one of the inmates of an asylum at Bamberg owed the defect to an attack of that disease.

Cerebro-spinal fever exhibits great variations in its severity, so that systematic writers describe several distinct forms of it. Some cases are called *foudroyant* or fulminant, the patient dying within a few hours from the commencement of his illness. Thus Dr. Gordon recorded one instance in Ireland in which the disease ran a fatal course in less than five hours. There was a dark, purplish eruption of spots of various sizes and shapes, especially upon the lower limbs. Ziemssen says that among forty-three cases he met with four in which the duration was from twelve to thirty hours. He relates a curious instance of a girl who was attacked one afternoon with headache and vomiting, but who got up in the middle of the following day feeling perfectly well, and went out of doors to fetch some beer; at about 2 o'clock she was again seized with violent headache and fell into convulsions, and at 6.30 she died. This form of the disease is seen chiefly at the beginning of an epidemic. According to Ziemssen, the presence of exudation into the membranes is discoverable only with the aid of

a microscope, which reveals an infiltration of cells, principally along the blood vessels. But in Dr. Gordon's case, greenish lymph had already been poured out both on the surface and at the base of the brain, and in places along the cord.

On the other hand, there are cases which are termed *abortive*, in which the patient may not be confined to bed for more than a day or two, or may even go on with his work as usual from the beginning to the end of his illness. Ziemssen has recorded in detail three examples of this form of the disease; in each of them headache, a painful stiffness of the neck, and vomiting (or at least nausea) were present; in one there was also rigidity of the upper dorsal vertebræ, and in another herpes and partial deafness were observed. In every instance recovery took place within four or five days. Abortive cases are said to be especially numerous when an epidemic is declining. Their proportionate frequency is very variable. According to Hirsch it sometimes happens that the greater part of the population of a district in which cerebro-spinal fever is prevalent are affected by this mild variety of the disease. Or it may be seen in the adults and the old people, while the severe form is raging among the children. To what extent the morbid changes in the membranes are developed in cases of this kind is as yet altogether unknown.

As a rule, however, the severity of epidemic meningitis is intermediate between the two extremes just mentioned. If it terminates in recovery, it does not begin to subside before the end of a week or a fortnight. There are certain minor modifications of the disease to which brief reference must be made. One is an "intermittent" form, in which there are regular paroxysms of fever, recurring after a quotidian or tertian type, with aggravation of all the other symptoms, the intervals being more or less completely apyretic. There are two ways in which cases of this kind have been explained; one supposition has been that they depend on the combined action of the marsh poison and of the specific poison of epidemic meningitis; the other, that the last-named disease is itself really of malarial origin. But it is evident that neither of these opinions is correct, for the intermittent variety has been observed in districts where ague does not occur. Moreover, Ziemssen has shown that when measured by the thermometer the fever is far from exhibiting the regular gradations which are characteristic of that disease. The remissions or intermissions often last over several days, and the temperature chart is most indefinite, and is not at all modified by the administration of quinine. Another so-called variety is the "typhoid." This arises in protracted cases, and is marked by muttering delirium, a dry, brown tongue, sordes on the lips, involuntary evacuations, and bed sores.

Again, several observers have found that during or just after an epidemic of cerebro-spinal fever, meningitis has presented itself with unusual frequency as a *complication* of other acute diseases. This is especially apt to be the case with acute (croupous) pneumonia; no less than fourteen instances of it are recorded as having occurred in Erlangen between 1866 and 1872. It has also been noticed with pleurisy, acute tonsillitis, and scarlatinal nephritis. Many of the patients recover perfectly well, the headache and stiffness of the neck passing off as the symptoms of the primary malady subside; it may then be said that the meningitis is of the "abortive" variety. But not a few such cases terminate fatally; and the usual morbid changes in the membranes are then found at the autopsy.

The mortality of epidemic meningitis seems to vary in different epidemics from 30 to 70 per cent.; the mean mortality is estimated at 40 per cent.

Relapses are not very uncommon, even in cases of moderate severity, in which the disease began to subside after a week or two of illness. And if

the patient's recovery should be uninterrupted it is often very slow. The headache sometimes continues throughout the convalescence, and may even persist for years afterward, undergoing aggravation when he stoops, or makes any mental or bodily effort. Ziemssen says he has seen several instances in which the spine remained stiff during several weeks. It seems natural to refer these symptoms to a cicatricial thickening of the pia mater, which has, indeed, been found present in certain cases where there has been an opportunity of making an autopsy, at an interval after recovery from epidemic meningitis, in consequence of chronic hydrocephalus having developed itself as a sequela of the disease. Recent observations seem to show that this is not an infrequent occurrence. Ziemssen has recorded three instances in which death happened ten weeks, twenty-seven weeks, and thirty weeks respectively from the commencement of the patient's illness; and one in which it took place as late as seven years after. In all of them the membranes were thickened and opaque in different places; and twice cheesy masses were found upon the convexity as well as at the base of the brain. The quantity of fluid in the ventricles was often very great; in the case which proved fatal at the end of thirty weeks—that of a boy, two years old—the thickness of the hemisphere (white and gray matter together) was only about an inch. A sufficient explanation of the occurrence of hydrocephalus under such circumstances may, perhaps, be found in the persistence of the inflammation of the ependyma which exists during the acute stage of the disease. But, as Ziemssen remarks, the fact that between the meningitis itself and the commencement of the symptoms of the ventricular effusion there is often a clear interval, during which convalescence appears to be going on favorably, tends to support the opinion that the secondary affection is in some way dependent on the cicatricial changes in the membranes. Niemeyer has suggested that the outflow of blood through the veins of Galen is interfered with by pressure. Dr. Collins, of Dublin, in a case which he examined on the sixty-sixth day, found the cerebro-spinal opening between the cerebellum and the bulb occluded, and attributed to this the hydrocephalus, in accordance with the well-known views of Mr. Hilton. But in a series of cases recorded by Dr. Merkel, of Nuremberg, in vol. i of the "*Deutsches Archiv*," it was noticed that there was always, also, a large quantity of fluid beneath the arachnoid around the *cauda equina*. This, it is obvious, is altogether inexplicable on the mechanical theories just referred to. And on the whole it seems best to fall back upon the idea of a chronic ependymal inflammation, which, indeed, is directly supported by the histological investigations of Merkel, who found the ventricular lining and its vessels thickly set with nuclei.

The symptoms which indicate the supervention of hydrocephalus are said by Ziemssen to be chiefly severe headache, and pains in the back and limbs, occurring only in paroxysms, and attended with vomiting, loss of consciousness, convulsions, and the involuntary discharge of fæces and urine. During the intervals, which may last for weeks at a time, the patient may appear to be in good health, mental and bodily; but very often he is dull and stupid, or he is affected with a general cutaneous hyperæsthesia, or with paralysis, or contraction of one or more of the limbs. Progressive emaciation appears to be another marked symptom. Whether it is possible for recovery to take place from the hydrocephalus is as yet uncertain. It is to be noted that failure of memory and weakness of intelligence, when they immediately follow an attack of epidemic meningitis, do not point to the presence of ventricular effusion, and are not of evil omen, since they generally pass off in the course of a few months. And the same thing may be said of various paralytic affections which are now and then observed during convalescence from the disease.

Ætiology.—The causes of cerebro-spinal fever and its relations to other so-called blood diseases are still very obscure. Those who are attacked by it are comparatively seldom over forty, and generally less than twenty years of age; in some epidemics almost all the cases have been in children under fifteen. But two of Ziemssen's patients were old people, aged seventy and seventy-seven respectively Males appear to be more often affected than females. Ziemssen says that it chiefly seizes upon strong, healthy subjects, but he and other observers are agreed that it is apt to be especially prevalent among the poor, who are ill fed, and who live crowded together in dirty, damp, ill-ventilated dwellings. When it occurred in France, at the beginning of the century, it was often entirely limited to the soldiers in barracks, the civil population in the same towns escaping entirely. In Ireland, in 1846, it affected principally the inmates of the workhouses; in the United States, it fell with especial severity upon the negroes.

Some of the facts just mentioned would seem to indicate that epidemic meningitis resembles typhus in its distribution, and may, therefore, be infectious. But of its being contagious in a narrow sense there is no evidence whatever. All observers are agreed that it does not pass from the sick to the healthy directly, under ordinary circumstances. Hirsch, however, has collected a series of cases which seem to show that a man going from an infected to a healthy place, may sometimes carry with him the germs of the disease, so that not only he himself afterward falls ill with it, but others are attacked in their turn. One suggestion is that a contagious principle is given off by the sick, but that it has to undergo some transformation or intermediate stage of its development, possibly in another animal, before it can infect a human being. I find it stated, on the authority of Mr. Ferguson, Veterinary Officer to the Privy Council in Ireland, that on each occasion when the disease has prevailed in that country, it has co-existed with an epizoötic of the same nature among pigs and dogs.

On the other hand, many observers think that epidemic meningitis is dependent upon some mysterious atmospheric or telluric influence, capable of manifesting itself simultaneously in places far apart from one another. Thus Dr. Sanderson reports that in 1865 it broke out on or about January 15th in two districts of the department of Dantzic, distant at least thirty miles from each other. So, again, Stillé insists on its having repeatedly prevailed in Europe and in America in the same years, and in the way in which it has made its appearance within the United States at places hundreds of miles apart. In this respect he compares it with influenza, and defines it as a pandemic disease. But I certainly think he goes too far when he hints that the very small local epidemics which have occurred in England, and even some scattered cases of meningitis that happen to have been recorded in London at a time when cerebro-spinal fever existed on the continent of Europe, may have been due to one and the same widely diffused cause.

I have already mentioned some of the facts which show that marsh miasm is not the cause of epidemic meningitis. Ziemssen further remarks that the districts in which this disease has prevailed have often been dry, sandy, elevated plateaus; and Hirsch, that it differs from ague in being especially apt to occur in the winter and spring. Yet another point of distinction is its tendency to attack children rather than adults.

Diagnosis.—This is seldom difficult. At the commencement of an outbreak, however, it would seem that enteric fever may sometimes be mistaken for it. Leyden says that among the German troops before Paris, in 1870, a series of cases occurred in which marked rigidity of the neck, severe headache, and hyperæsthesia were present, while the abdomen was flat, the temperature was low, and the bowels were confined. At first it was

doubtful whether the disease was not meningitis, but the autopsies showed that it was really typhoid fever, with but slight implication of the intestine. Epidemics of the two diseases may also prevail simultaneously. Leyden has seen this several times; he insists on the facial herpes as distinctive, since it is never observed in enteric fever.

When occurring as a complication of acute pneumonia, epidemic meningitis may be far from easy of diagnosis. Ziemssen remarks that rigidity of the neck is often entirely absent in such cases. In very young children, too, this symptom possesses very little significance even when it is present; and convulsions, coma, and other signs of cerebral disturbance, are equally without value. Maurer maintains that a tense, projecting fontanelle affords great help, since it proves that the intracranial pressure is excessive, and this is not likely to be the result of simple pneumonia.

Again, if an isolated case of meningitis should occur, it is often impossible to say whether it belongs to the epidemic, the simple, or to the tubercular form. The question may be of the gravest importance, if cerebro-spinal fever should happen to be raging in a neighboring town or district. It would seem that the spinal symptoms—the painful stiffness of the neck, the rigidity of the vertebræ, the hyperæsthesia, and pains in the limbs—are generally less marked in the non-epidemic varieties. Tubercular meningitis may often be distinguished by its prodromata, by its gradual onset, by its slow and interrupted course. The presence of a roseolous or purpuric eruption would probably be a conclusive proof that the case was one of cerebro-spinal fever.

In giving a *prognosis*, it is important to bear in mind the treacherous character of the disease. A case which at first appears to be of but little severity may afterward develop dangerous symptoms and prove rapidly fatal; and, on the other hand, patients whose condition had seemed hopeless sometimes recover.

3. Acute Simple Meningitis.—Some modern writers describe several forms of acute simple inflammation of the membranes of the brain under distinct names, according as the convexity or the base is affected, and according as the disease is more or less distinctly traceable to different causes. And, undoubtedly, a meningitis in which the whole surface of the brain becomes covered with a thick layer of pus is attended with much more violent symptoms, and destroys life far more quickly than one which is limited to a particular region, and in which only a small quantity of lymph is effused. But no complete division into separate categories is possible; as indeed one might have expected, from the analogy afforded by the similar affections of other parts.

Anatomy.—When the most intense degree of inflammation has been present, one finds, on turning back the dura mater, that the cerebral convolutions are completely hidden by a green, purulent material. This looks as if it were spread out over the hemisphere in the *sub-dural* space; but by scraping the arachnoid surface one generally discovers that little, if any, of it is really free. Dr. Moxon says that even when some of the exudation had appeared to come off upon the edge of the scalpel, he often found only epithelium and detritus on examining it with the microscope. Sometimes, however, a considerable quantity of pus is sub-dural; this, I believe, proves that the disease was due to extension from without. The converse, however, is very far from being true; namely, that meningitis is always attended with exudation into the "arachnoid cavity," when it is set up by such causes as caries of the petrous bone or necrosis of the calvaria. For, on the contrary, in the vast majority of these cases no sub-dural pus is found; and whenever it is widely diffused over the hemispheres I believe that it is

always present in great abundance within the meshes of the pia mater as well.

The investing membrane of the brain, with all its processes into sulci, may, in fact, be swollen to many times the normal thickness, appearing either soft, green, and gelatinous, or firm, yellow, and felt-like, according to the consistence of the exudation infiltrated into its substance. And from such a condition there may be every gradation, down to a point at which only slight traces of pus can be discovered, along some of the principal vessels, or at which the inflammatory process is limited to the base or even to one Sylvian fissure, or to a single lobe of the cerebellum.

In some of the more severe cases the convolutions themselves are softened. When the pia mater is stripped off, it carries with it portions of the cerebral tissue, leaving a ragged surface behind. Huguenin says that numerous leucocytes may be found throughout all the layers of the cortex, and that suppuration of the brain substance may actually reach such a point as to give rise to a diffused, yellow-gray maceration visible to the naked eye. The vessels often contain but little blood, in consequence of the increased pressure which precedes death. The ventricles may either be empty or contain a turbid liquid, or even pus. The choroid plexuses are sometimes infiltrated with inflammatory products.

Some of the sinuses in the dura mater are now and then found plugged with ante-mortem clots, even when the meningitis is not secondary to any disease of the bones which could itself have set up the thrombosis by extension. In a little girl, aged three, for example, who died in Guy's Hospital, and in whom both hemispheres were covered with a thick layer of yellow lymph, each lateral sinus, but especially the left, was filled with a greenish, softening material.

How often an acute simple meningitis of the brain spreads to the membranes of the spinal cord, it is probably impossible at the present time to say. In the majority of recorded cases the vertebral canal has not been opened, but we have had at Guy's Hospital at least eleven cases in which inflammatory products have been found in greater or less quantity beneath the spinal arachnoid. In none of them was there any reason to believe that the disease was of epidemic origin; in four it was directly caused by fracture of the skull or severe injury to the brain, or arose by extension of mischief from the cranial bones.

Ætiology.—The causes of simple acute meningitis are various. Most frequent among them are injuries to the head. These generally come under the notice of the surgeon, but the physician must remember that the disease sometimes follows a blow or fall which may not have produced any external bruise, and about which the patient may say nothing, particularly if he should have been intoxicated at the time. Some years ago a man was admitted into Guy's Hospital for a fractured thigh, caused by his having fallen into a cellar while drunk. He died at the end of five weeks, having been delirious all the time. He was believed to have delirium tremens; nothing was known of any injury to the head; he had been able to get out of bed and stand upright. At the autopsy it was found that there was general acute meningitis affecting the base as well as the surface; a small part of one parietal bone, over an area an inch in diameter, was of a greenish color, and its diploë was reddened. Both the periosteum and the dura mater seemed quite healthy, but it was thought probable that the bone had been injured at the time of the accident. Another common cause of meningitis is the extension of inflammation from chronic disease of some part of the skull. Syphilitic caries or necrosis of the calvaria sometimes kills in that way, and such a result may be altogether unexpected, the patient's progress having apparently been favorable until a very short time

before his death. So, again, with lesions of the temporal bone secondary to affections of the ear. Six cases of this kind have been observed at Guy's Hospital within the last few years, a much smaller number than that of the cases in which cerebral abscesses were due to the same cause, and in two or three of them the pathology is, perhaps, still open to doubt. One patient, a man, aged twenty-two, was said to have had sunstroke seven days before being attacked, and another man, aged twenty, had been struck on the head with a bolster immediately before dangerous cerebral symptoms set in. In each instance it was clear that the inflammation started from a diseased temporal bone, but there was a question whether an independent "exciting" cause was not in operation. On the other hand, there was the case of a woman servant, aged twenty-seven, of whose illness no history could be obtained; as no cause for the meningitis of which she died was discoverable, the ears were specially examined by the late Mr. Hinton, who found pus in the labyrinth on one side. To this, accordingly, the disease was attributed, but it must be remembered that in epidemic cerebro-spinal fever inflammation often spreads to the ears from the membranes, and there seems to be no reason why the same thing should not occur in the simple form of the disease.

Numerous other local affections may be mentioned as occasionally giving rise to meningitis. I once made an autopsy in a case in which an epithelioma, commencing in the lip, extended with ulceration along the pterygoid region until it passed through the foramen ovale into the interior of the skull, and another example occurred in a little girl who had necrosis of the upper jaw after measles, and in whom the cavernous sinus was full of a dirty-brown fluid. Or, the disease may be set up by suppuration of the eyeball, traveling, in all probability, along the sheath of the optic nerve. Trousseau relates a case in which its starting point was a chronic affection of the first two cervical vertebræ.

Next in frequency to the cases of meningitis that are traceable to local causes come those which are secondary to some other acute disease, and which are, therefore, classified by Huguenin as *metastatic*. It is, indeed, possible that more careful research may hereafter show that these have themselves a *local* starting point in some lesion affecting the cerebral vessels. Thus we have at Guy's Hospital had two cases in which inflammation of the pia mater accompanied an attack of erysipelas of the scalp; in one it is noted that the calvaria was discolored yellow. In four instances it appeared to be part of a common pyæmia; once there was a gray patch of cerebritis reaching the surface; once pericarditis also was present, so that one might regard the disease as specially attacking the serous membranes if the arachnoid could still be called by that name; once the meningitis was the only evidence of blood poisoning, but such seemed to be the most probable explanation of its occurrence, as it came on six days after an operation for imperforate anus in a child a year old; once it was associated with an abscess in the lung, and with suppuration in the mediastinal connective tissue and in the substance of one leg, there being, however, no obvious primary lesion. In a single case inflammation of the cerebral membranes was believed to be a complication of typhus; no affection of the vessels in the pia mater was discovered, but the spleen contained infarcts, and there was thrombosis of the renal veins. Four times it seemed to be secondary to acute pneumonia or pleurisy; all but one of these cases, however, presented peculiar features. One occurred in a man, aged twenty-two, who had been in the hospital six weeks for pleuro-pneumonia, and was convalescent and able to go into the grounds, when he was again attacked with dyspnœa and high fever, and died in three days; the spinal membranes, as well as those of the brain, were inflamed. Another

was in a woman, aged twenty-seven, who had been ill for a week with pleurisy, and then was attacked with an acute diphtheritic affection of the fauces, and died a day later, having been very restless toward the last, and having one pupil wider than the other; in her, also, the affection was cerebro-spinal. The third case was that of a man, aged twenty, who had acute meningitis involving both hemispheres of the brain, and pleuro-pneumonia of the lower lobe of the left lung; he was suffering from gonorrhœa, and Dr. Wilks was inclined to regard this as the cause of his fatal illness, but it was further said that he had had a blow on the head a few days before he was attacked. In the remaining case the patient was a temperate man, aged fifty-seven, who died, after sixteen days' illness, of acute pleuro-pneumonia affecting the left lower lobe; no cerebral symptoms appear to have been observed beyond delirium before death. There was much recent lymph both at the base of the brain and on the vertex. The only other morbid conditions that were discovered were chronic renal disease (apparently in moderate degree) and hypertrophy of the heart.

Huguenin says that at Zürich inflammation of the cerebral membranes is an exceedingly frequent complication of acute pneumonia. He quotes Chvostek as having found it four times in 220 cases in Vienna, and adds that in the Swiss city the percentage is higher.

Dr. Moxon has laid special stress on the fact that syphilis sometimes seems to be the cause of an acute simple meningitis, and I find five cases of this kind in our records, in addition to those in which inflammation of the membranes was set up by disease of the calvaria. Once a gumma was discovered in the brain substance. In no instance was any disease noticed in the cerebral arteries, but it seems very probable that such a lesion may hereafter be discovered in similar cases. In 1871, a man, already in the hospital for disease of the aortic valves with regurgitation, died after two days' illness of cerebro-spinal meningitis; no embolism was detected, but the spleen contained infarcts. In 1874, a woman, aged forty-eight, who had been admitted for chronic jaundice caused by biliary calculi, was attacked by a fatal inflammation of the cerebral membranes; this, however, was doubtless consequent upon ulcerative endocarditis of the aortic valves, which was discovered at the autopsy. It is remarkable that I do not find in our records a single case in which Bright's disease appeared to be the cause of meningitis. Huguenin, however, mentions it as having occurred consecutively to acute pleurisy in a girl, aged fourteen, who had "acute fatty degeneration of the kidneys."

There remain a few cases in which meningitis can be traced to none of the causes hitherto mentioned. In our records at Guy's Hospital I find nine cases of this kind. In three of them pus or lymph was spread out in large quantities over the whole surface of the brain, equally over the hemispheres and at the base; in three the amount at the base was much less than upon the convexity; in one the latter region was alone affected, the material being, in this instance, lymph. Once the under surface of the brain and the subarachnoid space of the spinal cord were all bathed in pus, there being but little upon the hemispheres. In the two remaining cases the distribution of the inflammatory products was the same as in ordinary examples of tubercular meningitis In every instance the cause of the disease remained a mystery, but sometimes meningitis of the convexity is set up by the action of the sun's rays upon the head. Huguenin speaks of laborers who have been attacked while working bareheaded in the fields, and he gives full details of the case of a sailor who fell ill the day after he had been rowing without anything upon his head in the hottest weather of July, and who died in five days. Another case related by the same writer is that of a student who was foolish enough to spend three hours, from 1 to

4 P.M., during a hot summer's afternoon, paddling about the lake of Zürich, lying flat on his belly on a board. All the exposed parts of his body became severely sunburned; at 6 P.M. he was seized with headache, and an hour later with shivering, and all the symptoms of meningitis rapidly developed themselves; his illness, however, terminated in recovery. Guersant met with a similar instance which ended fatally, and was verified by an autopsy, in an infant, aged six months, whose cradle had been left in the sunshine in the middle of a garden. Rilliet and Barthez mention the case of a child who manifested alarming cerebral symptoms after reading a book, the pages of which were exposed to the sun.

Course.—The symptoms of acute meningitis bear a general resemblance to those of the tubercular affection; in the typical forms of both there is a stage of "irritation," which is succeeded by a stage of "pressure." But the more severe cases of the one present a peculiarity that is, I believe, without parallel in the other disease, namely, that each of the two periods, although marked by the most characteristic symptoms, may yet terminate within two or three days, or even last no longer than a few hours. Thus an effective contrast can be drawn if one describes several distinct non-tubercular varieties of meningitis, but, as I have already stated, I think that such an argument is artificial.

Simple meningitis commonly begins quite suddenly: it has no prodromata. The patient, if an adult, may be seized with a rigor; in children this is less common. Headache is generally present from the first; it may either be referred to the forehead, or affect all parts alike. There may be exacerbations from time to time, in which piercing cries are uttered; the agony appears to be altogether intolerable. Giddiness is frequently an early symptom, the ground seems to give way beneath the feet, and the legs may feel so weak that to stand upright is almost impossible. Vomiting, perhaps, occurs once or oftener. There is an extreme irritability to light and sound. The eyes are brilliant and injected, the face is flushed, the head is hot, the carotids are felt to throb violently. The temperature rises, and may reach 104° by the third day; there may be great complaint of thirst. The pulse is quickened, as a rule, but this is not always the case. Epileptiform convulsions are not uncommon and sometimes mark the commencement of the disease. The back of the neck is often rigid, so that the patient seems to bore his head into the pillow. Sometimes his limbs are stiff. He generally lies coiled up in bed, anxiously avoiding notice, and most unwilling to be disturbed in any way. His mind may for a time be perfectly clear, but afterward he becomes delirious. In some cases there is violent maniacal excitement from the very first. Sleep is altogether wanting, or very broken and disturbed. The pupils are, as a rule, contracted; the ophthalmoscope may show either ischæmia or neuro-retinitis.

The second stage of the disease is characterized by stupor, which more or less quickly passes into coma. The pupils are sluggish, or even dilated and insensible; they are often unequal in size. There may be loss of power in the limbs of one side, with or without a similar affection of the corresponding half of the face. Sometimes the patient squints, but even when the base is involved one can seldom make out a definite paralysis of any of the cranial nerves; they, of course, remain unaffected when the inflammation is limited to the convexity of the brain. Epileptiform convulsions may return again and again until one of them proves fatal, or they may be altogether absent, or, again, there may be attacks of spasm confined to certain muscles or to one arm and one leg. The temperature generally remains high, ranging from 102° upward, but the face is now pale, and the extremities may be cold to the touch and bathed in a profuse sweat. Toward the last, the evacuations are passed involuntarily.

The duration of simple meningitis varies with its intensity. Those cases in which the whole surface of the brain, including the convexity, is inflamed scarcely ever last more than a week and generally terminate within three or four days. When the base alone is affected the patient sometimes lives as long as in the tubercular form of the disease. But this is by no means always the case; he may die in forty-eight hours, and the only discoverable morbid change may be on the under surface of the brain. In such cases, however, there is no successive development of the symptoms in regular order. Sometimes the patient is attacked with epileptiform convulsions and becomes quickly insensible, or he may be violently delirious. In certain cases loss of spirits is the earliest symptom, as was noticed long ago by Abercrombie. There may be little or no pain in the head, and thus the real nature of the disease may be altogether overlooked.

Diagnosis.—Simple inflammation of the membranes of the brain may be difficult to recognize, as we have already seen to be the case with tubercular meningitis; but the diseases most likely to lead one into error are not quite the same. When it is impossible to make out the presence of any of the recognized causes of the affection, and when the symptoms are obscure, one may be unable to distinguish it from alcoholism, mania, or epilepsy. On the other hand, when the patient is known to be suffering from acute pneumonia or erysipelas it may be very hard to determine whether there is more than the ordinary symptomatic delirium. Cases in which alarming indications of cerebral disturbance are traceable to otorrhœa seem to be peculiarly liable to be wrongly interpreted. In 1877, I made an autopsy in the case of a patient of Dr. Frederick Taylor's, who had died with what appeared to be clear symptoms of meningitis secondary to disease of the ear, including convergent strabismus, a swollen, œdematous condition of both optic discs, delirium, and coma. The only sign of any disease of the encephalon was a blackened state of the arachnoid over a small part of one lobe of the cerebellum, but there was a putrid thrombosis of the lateral sinus and jugular vein, and this had set up numerous pyæmic abscesses in the lungs. In the "*Med. Times and Gazette*" for 1877 will be found a precisely similar case, under Dr. Wilson Fox, in which the patient had all her limbs flexed, and suffered from headache, photophobia and hyperæthesia of the surface, but in which the brain and its membranes were perfectly healthy. It would, therefore, appear that pyæmia dependent upon thrombosis of a lateral sinus is capable of simulating meningitis rather closely. The point is of the more importance in that the former disease seems not to be always fatal; at least this appears to me to be the most probable interpretation of two cases recorded by Dr. Andrew in the "*Med. Times and Gazette*" for 1875. One was that of a youth, aged sixteen, who had had a discharge from the ear, and who became drowsy and heavy, with frontal headache, giddiness, sickness, and blurring and œdema of the optic discs. The other occurred in a girl, aged nine, who had also had ear disease, and who was attacked with pain in the head, vomiting, and delirium, so that she screamed and started in her sleep. Meningitis was diagnosed, but each patient recovered after an illness of some weeks' duration, in the course of which there was distinct evidence of pyæmia, one of them having pneumonia, the other having an abscess in the thigh.

Lastly, I believe that there is always an element of uncertainty in regard to the diagnosis of those cases in which the typical symptoms of meningitis develop themselves in regular order and with such rapidity as to threaten a fatal termination within three or four days, or even still earlier. I have seen more than one instance of this kind, in which the gravest prognosis was given, but in which a complete recovery took place. A case which comes forcibly to my recollection is that of a young lady, the daughter of a

medical man. Her education had been rather neglected during childhood, and she had been for some weeks straining all her powers to keep pace with her schoolfellows in her studies. I have, unfortunately, no notes of her symptoms, but I know that they appeared to point clearly to the presence of meningitis; yet the attack passed off, and she is now in good health. I have cited a case of Huguenin's, in which the diagnosis was adhered to, in spite of the fact that the patient recovered.

Prognosis.—The question as to the possibility of recovery from acute simple meningitis is still more definitely raised by certain cases which come under the notice of ophthalmic or of aural surgeons. In 1866, Mr. Hutchinson recorded in the "*Ophthalmic Hospital Reports*," a series of cases of children who were brought to him for blindness, which he found to be due to optic neuritis, and nearly all of whom had had a severe illness, attended with delirium and other cerebral symptoms, and supposed to be fever. Dr. Allbutt suggests that the disease was really meningitis. This observer also states that he has seen several instances in which a condition of defective mental development (or idiocy as it would be called) has been associated with atrophy of the optic discs, and has, in all probability, been referable to a long past inflammation of the cerebral membranes. On the other hand, certain cases in which a permanent deafness has resulted from an acute illness, have been interpreted in a very different way by Voltolini (who first drew attention to them in 1867) as well as by some other writers. According to Brunner (of Zürich) the affection in question commonly sets in suddenly; the child suffers from fever; it may vomit; its head is hot; it becomes excited and delirious, tossing about in bed and screaming violently; within the first twenty-four hours it becomes partially unconscious, and after two to four days it passes into a state of coma. At the end of another period of two to four days, however its regains its senses very rapidly; but when it first tries to walk it is found to stagger, and it quickly becomes deaf and (as a consequence) remains dumb for the rest of its life. Voltolini's theory is that the disease in such cases is an acute inflammation of the labyrinth; the cerebral symptoms (as in Ménière's disease) being supposed to be caused in some way by the aural affection. Direct evidence from the post-mortem room is as yet altogether wanting. But, as Brunner remarks, the fact that the deafness is constantly bilateral is opposed to such an interpretation. He thinks that there may be some morbid change on the floor of the fourth ventricle, implicating the *striæ acusticæ*. But it seems to me that Voltolini's cases and those of Mr. Hutchinson and Dr. Allbutt must be taken together, and in connection with the other cases in which recovery takes place after an illness resembling meningitis, but in which there is not left either deafness or blindness or idiocy.

What makes me hesitate in coming to any conclusion as to the real nature of such cases, is the circumstance that one scarcely ever finds in the post-mortem room adhesions of the membranes at the base of the brain or other clear evidences of a former meningitis. It has, however, been shown that in epidemic meningitis recovery is not infrequent, and that the disease often leaves behind it deafness or blindness; in several cases the remains of inflammatory exudation have been discovered when the disease has run into chronic hydrocephalus, which has proved fatal a few months later. These observations might at first sight appear to settle the question, but it may be objected that there is a fundamental difference in the fact that simple meningitis does not give rise to any similar form of chronic hydrocephalus, and further, that at present nothing is really known as to the morbid anatomy of those cases of epidemic meningitis in which complete recovery takes place. Such cases are undoubtedly due to the same cause which gives rise to "cerebro-spinal fever" in

general, and so are properly included under the synonymous term of "epidemic meningitis." But it has not yet been ascertained that they are attended with an actual visible inflammatory change in the membranes, and as regards simple meningitis it is impossible for us to take an equally broad view, and to waive the production of direct pathological evidence.

After all, however, the really important point is the practical one, namely, that cases which appear to be well-marked examples of inflammation of the membranes of the brain do not always terminate fatally. Until the advance of clinical experience or the introduction of new methods of investigation shall have rendered this statement incorrect, no explanation of the real nature of the cases in question, however complete and however convincing, can deprive it of its significance.

Thus, in a case of meningitis, one should never entirely lose heart; scarcely any is so hopeless as to justify one's abandoning it altogether—particularly if the patient is a child.

Treatment.—At an early stage an important object is to relieve the headache and to diminish the cerebral excitement. The patient should be placed in a cool, dark, well-ventilated room, and should be kept perfectly quiet. The hair should be cut short, or even shaved close to the scalp. Cold should be applied to the head, and for this purpose a large bladder, containing some small pieces of ice and a little water, is more serviceable than anything else; evaporating lotions are far less effective, and require to he very frequently changed. In 1878, I saw a case which appeared clearly to demonstrate the usefulness of the local application of ice. A man, aged forty-three, a baker, was taken with a kind of fit on the evening of the 26th of June, and was seen the same night by Dr. Churchward, with a temperature of 104.6°, a pulse of 160, a very flushed, hot face, vomiting, and great pain in the head and neck, as well as in the back and in the limbs. He had been driving about all day in a hot sun; three days previously he had stood for some hours in water up to his knees, in consequence of his cellars having been flooded by a heavy rainfall; for more than a month he had been unusually irritable in his temper. For a day or two he seemed to improve, but during the night of the 28th he became collapsed and almost pulseless, with a cold, clammy sweat. After this he remained sleepless and was very delirious, with contracted pupils and constant twitching of the hands. On the 30th I saw him; his symptoms were then of the most alarming character; I found the optic discs normal. We prescribed a mixture containing the iodide and the bromide of potassium, and a draught of chloral and morphia, in the hope of inducing sleep. Next day (July 1st) he appeared to be rather worse than better; he had not slept for more than five minutes; he had been sick again; he was constantly talking and picking the bed clothes. Dr. Churchward, therefore, ordered a towel to be wrung out of iced water, and to be kept applied all over his head and neck, with lumps of ice between the folds. The effect seemed to be magical; in a few hours he became quiet and fell asleep; the sickness and the clammy perspirations ceased. He liked the cold to his head, and during the night he slept for six hours. On the following day (July 2d) he was rational, and told Dr. Churchward that he remembered nothing since the 29th June, except that another doctor had examined his eyes. The ice was continued until July 4th. His recovery was uninterrupted; on the 7th he got upon the sofa, and by the 11th he was able to be drawn out in a carriage. This patient died in the spring of 1880, of acute pneumonia. Dr. Churchward obtained for me permission to examine his brain. There was no conspicuous change, nothing, I think, that would have attracted my notice if I had not known of his former illness. But the arachnoid and pia mater at the base of the brain appeared to me to be thicker and crisper than natural, especially over

the right Sylvian fissure; the olfactory lobes were more firmly bound down; and there was more connective tissue on the under surface of the pons. The velum interpositum also seemed to be increased in density, although the lateral ventricles were not dilated.

In epidemic meningitis, Ziemssen and other German physicians keep ice bags to the head, back and neck for weeks together, and find that they afford marked relief to the patient's sufferings, soothing him and enabling him to sleep.

Most physicians abstain from administering anodynes in cases of meningitis, as being likely to mask the symptoms; but at an early period of the disease this objection seems unfounded, and Dr. Bristowe says that he has frequently given opium with manifest relief. Huguenin recommends the subcutaneous injection of morphia in small doses, and the use of enemata containing fifteen to forty-five grains of chloral. Ziemssen says that in epidemic meningitis remedies of this class are indispensable. To check vomiting, the patient may have little pieces of ice to suck; or bismuth, hydrocyanic acid, and similar remedies may be prescribed.

There is, perhaps, no reason to suppose that leeches, or venesection, or the most active purging could cut short a meningitis which is going on to the effusion of lymph and pus. But is it equally certain that some such measures may not be useful in those cases in which recovery is most likely to occur? In epidemic meningitis, at any rate, recent German authorities adopt all those "antiphlogistic" measures which were in vogue in this country thirty years ago. They not only apply leeches behind the ears and cupping glasses to the spine; in fulminant cases, or when the patient is very restless, they employ venesection; and they administer calomel, or rub in blue ointment, at the same time washing out the mouth with solution of chlorate of potass, to prevent the gums from being affected by the mercury.

So, again, in simple meningitis, the older physicians believed that they had seen successful results from energetic treatment. Some of Abercrombie's cases will perhaps still bear quotation. "A girl, aged eleven" (Case 69), "had violent headache and vomiting, with great obstinacy of the bowels, and these symptoms were followed by dilated pupils and a degree of stupor bordering on perfect coma; pulse 130. She had been ill five or six days; purgatives, blistering, and mercury to salivation had been employed without benefit. One bleeding from the arm gave an immediate turn to this case; the headache was relieved; the pulse came down; the vomiting ceased; the bowels were freely acted upon by the medicines which they had previously resisted; and in a few days she was quite well." "A gentleman, aged twenty-one" (Case 72), "was first affected with confusion of thought and very considerable loss of recollection. He then complained of headache, and after a day or two had double vision. At this time he was out of bed the greater part of the day, but was restless and confused, and at times incoherent. He was then confined to bed, and had constant headache, much incoherence and oppression, the double vision continuing. The pulse was at first frequent, but fell gradually and sank below the natural standard; and the symptoms went through a course exactly similar to that which has been described in many of the fatal cases. As the pulse fell in frequency, he became more and more oppressed, until he sank into a state of stupor, from which he could scarcely be roused to answer a question of the most simple kind. The case went on in this manner for eight or ten days, during which time he was treated by repeated general and topical bleeding, cold applications, blistering, etc. The bowels were very obstinate, and large doses of the most active purgatives were given with little effect. The case was considered desperate, when he began to take the castor oil in full doses, repeated every two or three hours. In a few hours he was purged very actively nine or

ten times; the same evening he was relieved from every alarming symptom, and in a few days he was free from complaint." "A girl, aged seven" (Case 75), "had severe headache, impatience of light, fever, and slight delirium, followed by stupor, squinting, and great obstinacy of the bowels. The tongue was at first foul, but became clean after a day or two. She was considered as being in a hopeless state of hydrocephalus. At the end of a week, strong purging being produced, she recovered rapidly, and in a few days was free from complaint." It is, no doubt, possible that the favorable issue of these cases was spontaneous, and would have occurred independently of all active treatment." But, on the other hand, it may be that under the negative practice of the present day they would have terminated fatally.

Of course, it is essential that our treatment should do no harm. In those cases in which it is difficult to distinguish between enteric fever and tubercular meningitis it would be very wrong to purge. And another class of cases, in which uncertainty of diagnosis ought to influence our practice, is that of the meningitis secondary to disease of the ear. This form of the disease is probably altogether hopeless, but I have already shown that pyæmia may simulate it very closely.

Again, in adult patients it is of the utmost importance that we should not overlook the possibility that syphilis may be the cause of symptoms resembling those of meningitis. We have seen that it may give rise to inflammation of the membranes of the brain, but I am not sure whether that affection would be especially amenable to treatment. What I am now thinking of is rather that one may be called in to see a man who is insensible, and that the history of the case may seen to point to meningitis, but that the lesion may really be one of the more direct results of syphilis. It would then be cruel to ask of a wife, or of a mother, questions which might leave the most painful impressions, and which, if answered negatively, could never decide the matter. But the most careful search should be made for evidence of syphilis, such as the presence of nodes or gummata, and the recurrence of miscarriages or sterility on the part of the wife. If there be any reason to suppose that such a cause may be in operation, full doses of the bichloride of mercury or of the iodide of potassium should be given. If this treatment is unsuccessful it can do no harm, and its success is sometimes very great. Whether it is advisable to prescribe mercurials (except as purgatives), or iodide of potassium, in cases which are believed to be examples of idiopathic meningitis, is very doubtful.

When coma sets in, the question arises as to the application of blisters. Sir Thomas Watson says that in his experience the patient has sometimes emerged from that condition after a cap of blistering plaster has been put upon his head. He does not tell us whether the improvement was permanent. Huguenin speaks in high terms of the value of energetic douches (of cold water) for the same purpose. But it seems to me that to excite a merely temporary return of consciousness might be injurious instead of beneficial. The application of mustard plasters to the calves of the legs must surely be altogether futile. Our endeavor should rather be to sustain life from hour to hour, in the hope that the tide may turn. Sir Thomas Watson remarks that "patients apparently moribund are occasionally saved by the judicious administration of stimulants and restoratives, of ammonia, Hoffmann's anodyne, beef tea, wine, and (it may be) of well-timed opiates."

In those cases in which recovery takes place it is probably advisable to give iodide of potassium during convalescence, and a good supply of nourishing food is then essential.

Adhesive Thrombosis of Cerebral Sinuses.—Another affection of the

membranes of the brain—thrombosis of the sinuses—may conveniently be described in this place, although to the pathologist it seems remote from meningitis. I have already spoken of cases in which pyæmia results from a putrid inflammation of the great intracranial venous channels, itself secondary to disease of the temporal bone, facial carbuncle, or injury to the skull, and attended with the formation of a thrombus that rapidly softens down into a puriform liquid, But the affection now to be discussed has no local starting point, gives rise to no general infection of the blood, and consists in a simple plugging of one or more of the sinuses with a firm clot, which becomes closely adherent, has sometimes a laminated structure, and tends to undergo organization into permanent tissue. Von Dusch and Gerhardt are the writers to whom we are most indebted for our knowledge of it. It is, I believe, of very infrequent occurrence ; at least, I find only one example of it in our pathological records at Guy's Hospital. Its most usual seat is the longitudinal sinus ; sometimes it extends into the tributary veins, so that the hemispheres appear to be covered with coiling, worm-like bodies ; sometimes it is prolonged into one of the lateral sinuses. In some instances hemorrhage beneath the arachnoid seems to have occurred as a consequence of this affection ; in others, the substance of the brain is said to have been found ecchymosed or softened, but it appears to me doubtful whether those cases have been correctly interpreted.

The clinical recognition of plastic or adhesive thrombosis of a cerebral sinus is exceedingly difficult. One might have thought that the distention of collateral channels would serve to indicate its presence. And some writers have described an engorgement of veins running from the anterior fontanelle of an infant to the neighborhood of the temples and ears, and the occurrence of epistaxis, as signs of plugging of the longitudinal sinus ; while they have supposed that œdema over the mastoid process pointed to obstruction in the corresponding lateral sinus. But surely one often sees veins ramifying over the scalp of a child without any such affection being present.

There are, however, certain conditions under which this form of thrombosis is especially apt to occur, and which, therefore, may suggest a suspicion of its presence. It has been observed in ill-nourished infants, six months or a year old, who have suffered severely from diarrhœa for some weeks before their death. Such cases generally resemble those of the "spurious hydrocephalus," described at p. 591, but with the addition of some more definite cerebral symptoms, such as nystagmus, squinting, ptosis, facial paralysis, and especially rigidity of the neck, back, or limbs. The bones of the skull may be felt to overlap one another, and the fontanelle is generally sunken, but in one of Gerhardt's cases it was said to have filled out and to have become tense toward the last. In adults an adhesive thrombosis seems sometimes to occur spontaneously ; sometimes it is consecutive to enteric fever, or a sequel of parturition, especially when much blood has been lost.

Its symptoms are very vague. There may be little more than a general apathy and depression, or the patient, after complaining of headache, or being delirious, may become comatose, or his face or limbs may be affected with convulsions or with some form of paralysis. Thus this rare disease is liable to be mistaken for several others, each of which is comparatively common ; namely, for the less acute form of meningitis, for tumors and other lesions limited to the upper parts of the hemispheres, and for the diffused affections of the brain which were described in the last chapter.

The duration of the thrombosis is variable ; it may last several weeks, or death may occur a few days after its commencement, so far as this can be determined from the clinical history of the case. Its treatment would probably consist in keeping the patient in a state of perfect quietude, feeding him well and administering ammonia and stimulants.

HÆMATOMA OF THE DURA MATER.—There still remains to be described an affection of the cerebral membranes which, having for many years been a puzzle to morbid anatomists, has recently become a subject of clinical interest also. It consists in the presence of one or more membranous layers on the inner surface of the dura mater, within the so-called arachnoid cavity. When recent they are soft and vascular, but in course of time they may become tough, white, and fibrous, so as to resemble in appearance the dura mater itself. Virchow says that in one instance he counted no less than six or seven of them lying one upon the other. They commonly extend over the greater part of a hemisphere, and at their margins they adhere together so as to form a closed sac, or a series of sacs, which are generally attached much more firmly to the inner face of the dura mater than to the arachnoid surface of the pia mater. In consequence, they were formerly supposed to be formed by a separation of the imaginary parietal layer of the arachnoid from the dura mater, but this notion has long ago been refuted. Indeed, the cyst is sometimes perfectly unattached, so that it falls out as soon as the fibrous covering of the brain is cut through. In a large proportion of cases (50 per cent. according to Huguenin) the affection is bilateral, a separate hæmatoma concealing and flattening the convolutions of each hemisphere. A considerable quantity of blood, either recent or tawny-brown with age, is commonly found between the layers, which themselves are often deeply stained with hæmatoidin. But sometimes that which fills the adventitious cavity, or cavities, is a thin, serous fluid which may contain a quantity of cholesterine; it is believed that such cases afford the only examples of what was formerly described as a variety of hydrocephalus, in which the effusion was supposed to be in the arachnoid space, instead of being in the ventricles (*Hydrocephalus externus*).

In 1845, Mr. Prescott Hewett, in a paper read before the Royal Medical and Chirurgical Soc., maintained the opinion, which had before been promulgated by Houssard and Baillarger in France, that the starting point of this disease is an effusion of blood into the space between the dura mater and the arachnoid surface of the pia mater. But a few years afterward Virchow gave the powerful support of his authority to a very different doctrine, namely, that the earliest morbid change is an inflammation of the dura mater; this, he supposed, becomes hyperæmic, and exudes upon its under surface a delicate material, richly supplied with wide, thin-walled vessels, which rupture and yield the blood that is so commonly found extravasated in cases which have proved fatal. Virchow's view has been adopted by most of those who have since written on the subject; and his name "pachymeningitis hæmorrhagica" has met with very general acceptance. Quite recently, however, Huguenin has reverted to the theory that the disease begins as a hemorrhage. If his observations are correct, one can hardly escape from the conclusions which he draws from them; and in any case the best designation for it seems to be the old one of "hæmatoma of the dura mater," which leaves the question of its origin open. I should mention that in a large majority of instances the affection is merely a complication of cerebral atrophy, whether of that form which is simply senile, or of that which is due to chronic alcoholism, or of that which constitutes general paralysis of the insane. Now, Huguenin states that in a number of cases of general paralysis he has been able to trace what he believes to have been the earliest stage of a hæmatoma, in the presence of a soft layer of blood clot, spread out over the convolutions, having its greatest thickness (2 mm.) opposite the parietal eminence, and gradually thinning off toward its margins. This substance comes away from the dura mater only in small shreds, but they have no vascular connection whatever with that membrane, which is itself perfectly intact, pale, and free from hyperæmia. Moreover, the

microscope shows that they really contain at this period no elements but those of coagulated blood—a network of coagulated fibrin, blood discs, and leucocytes. Afterward, indeed, the clot undergoes organization, vessels are developed in it, and become continuous with those belonging to the dura mater, which now looks more or less injected. Any one versed in the ordinary pathological doctrines of the day will have little difficulty in forming a conception of the nature of the subsequent changes, which lead to the formation of massive membranous layers, or to the accumulation of collections of blood, or of serous fluid in their midst.

As to the original source of the hemorrhage, Huguenin thinks that it comes from the veins which open into the longitudinal sinus; he has noticed that these vessels are often varicose and thinned, that their coats are affected with fatty degeneration, and that they are sometimes filled with thrombi; once he actually discovered a laceration in the wall of one of them in a case in which a layer of blood was spread out over the brain.

Hæmatoma of the dura mater occurs more often in males than in females. It is met with chiefly in those who are advanced in life, but exceptional cases of it may be seen at all ages; in 1864, Dr. Wilks exhibited to the Pathological Society a specimen taken from a young man. I have already shown how frequently this affection is associated with atrophy of the brain. It is scarcely ever observed in the post-mortem room of a general hospital. At Guy's I do not know of a single well-marked example of it within the last twenty years. Dr. Wilks obtained his preparation from the dissecting room, the young man, who was "half-witted," having died in a workhouse. Thus I am unable to understand Huguenin's statement that among the causes of hæmatoma are chronic affections of the lungs and heart, kidney diseases, blood diseases (such as "pernicious" anæmia, scurvy, and hæmophilia), typhus, recurrent fever, smallpox, acute rheumatism. When they end fatally, these complaints are, no doubt, sometimes attended with hemorrhage into the membranes, but it by no means follows that in cases which recover the same thing may occur to such an extent as to lead to the formation of membranous layers. Injuries to the head, however, seem occasionally to give rise to the affection. In 1855, Dr. Quain showed to the Pathological Society a specimen taken from a farmer, aged fifty-eight. He had for three years suffered from various cerebral symptoms, which dated from a fall from his cart, when he was temporarily rendered insensible and received a large scalp wound on the right side.

The clinical course of hæmatoma of the dura mater varies widely in different cases. Sometimes the only recognizable symptoms are those of a fatal apoplectic seizure consequent on the sudden outpouring of a large quantity of fresh blood in or between membranes formed out of a coagulum of old date, which itself had in no way disturbed the patient. Very often the affection is found on post-mortem examinations of those who have died of general paralysis of the insane, unattended with any unusual symptoms such as to suggest the presence of a hæmatoma. It would be a great mistake to suppose that the epileptiform and other attacks which are of frequent occurrence in general paralysis are usually, if ever, traceable to that condition. Indeed, as Huguenin remarks, the wasted brains of these patients allow of the accumulation of a large quantity of blood, or of products derived from it, on their surface, without pressure. Even headache seems to be very generally absent in such cases.

However, it would seem that in some exceptional instances the affection gives rise to symptoms that may enable it to be diagnosed. Huguenin lays stress on the occurrence of two or more apoplectiform attacks separated by an interval during which the patient remains fairly well. He relates in

detail the case of an intemperate man, aged thirty-one, who about two years before his death began to complain of severe headache, and who one day fell unconscious, with a slow, full pulse, contracted pupils, transitory convulsive movements of the right side, and partial paralysis of the right side of the face. After twenty-four hours he gradually recovered his senses and got up, but he continued to suffer from pain in the head, and slowly lost his memory and intelligence, and finally he had another seizure, which proved fatal in four days. The brain was found atrophied, with dilated ventricles, and with a hæmatoma on each side consisting of a complete membranous sac divided into compartments. But I cannot endorse Huguenin's opinion that such a history is any way characteristic of this affection.

Attempts have also been made to diagnose hæmatoma of the dura mater in cases which have ended in recovery. That it is possible for the affection to subside after giving rise to well-marked symptoms appears to be established by a case recorded by Bouillon Lagrange. An old man, aged seventy-five, who was suffering from drowsiness, and who was already failing in intelligence and memory and bodily activity, had a fall from his horse. He was not injured outwardly, but he now became more deeply unconscious and had right hemiplegia. At the end of two months he was perfectly comatose and almost totally paralyzed ; he was unable to speak, and passed his motions under him. After this, however, he gradually began to improve, he regained his senses and his memory, he recovered the use of his limbs, and when two more months had elapsed he was considered to be cured. He remained quite well for six months, and then he was murdered. The back part of the right hemisphere was found to be flattened by a cyst, which adhered to the arachnoid and contained three or four spoonfuls of a bloody liquid. Griesinger has even ventured to infer the presence of this affection in a man, aged fifty-seven, who recovered and was in good health at the time when his case was published. He had been a spirit drinker, and for about five months had suffered from severe headache, extreme drowsiness, and confusion of ideas. His gait then became unsteady, his pupils were equal and contracted, his pulse was rather irregular, he seemed to have a little loss of power in the left side of the face. He would sleep all day long, and pass his urine in his bed, but at the end of a month he began to improve, and in about ten days from that time he was perfectly well and was discharged from the hospital. Griesinger confesses not only that he expected a fatal issue, but that he originally diagnosed a rapidly-growing cerebral tumor. It is perhaps worthy of notice that among the symptoms of which the patient complained was one which I have myself seen in a case of tumor, namely, a sensation of something moving to and fro within the skull, but so little is positively known of recoveries from any of the organic diseases of the brain that the observations of Griesinger and Bouillon Lagrange are well worthy of being remembered.

The best treatment for a case supposed to be one of hæmatoma would probably consist in the application of cold to the head, in the administration of purgatives, and perhaps in the abstraction of blood by leeches or even by venesection.

NEUROSES,

OR

FUNCTIONAL DISORDERS OF THE NERVOUS SYSTEM OF WHICH THE ANATOMICAL SEAT IS UNKNOWN.

THE SPASMODIC NEUROSES.

HISTRIONIC SPASM OR SPASMODIC TIC—SPASMODIC WRY NECK—"FUNCTION SPASMS" EXCITED BY MOVEMENTS—WRITERS' CRAMP AND OTHER "PROFESSIONAL" SPASMS—PARALYSIS AGITANS—CHOREA—TETANY—TETANUS—HYDROPHOBIA.

We have now to enter upon the consideration of a group of diseases which may be termed the "spasmodic neuroses," and in all of which the chief symptoms are spasmodic movements in the voluntary muscles. Some of these are very closely connected together, being often found at once, or in succession, in the same patient; others are not more intimately related to one another than to any of the other neuroses. The order in which I shall take them will be to begin with those which are limited to a single muscle, or to a few muscles, and afterward to pass to those which involve an entire limb, or the whole body. The following enumeration of them, with brief definitions, will, I think, be found useful, since they are apt to be confounded with one another, particularly when affecting the upper limbs.

1. *Spasmodic Tic or Histrionic Spasm.*—A jerking movement, sometimes limited to a single muscle, repeated at considerable intervals without alteration for a great length of time; sometimes suddenly replaced by a similar movement of an entirely different part.

2. *Spasmodic Wry Neck or Torticollis.*—Paroxysms of clonic spasms in one side of the neck, rapidly succeeding one another, and leading to great distortion of the head; sometimes extending to the side of the face or to the corresponding upper limb; sometimes passing into tonic contraction of certain cervical muscles; occasionally subsiding for a time, and recurring after the lapse of some years.

3. *Writers' Cramp* and the analogous affection of musicians, milkmaids, and others; consisting mainly in an incapacity to perform some particular action, in consequence of a spasmodic movement in the fingers, hands, or wrist; tending to progress and to involve other muscles, even those of the opposite limb, if these are called into play in the continued performance of the action in question.

4. *Paralysis Agitans.*—An oscillatory movement, generally beginning in one of the upper limbs, but afterward affecting the corresponding lower

limb, or the opposite upper limb, or the neck, or tongue, or all these parts together; for a time paroxysmal, but afterward continuous so long as the patient is awake; at first ceasing when the affected muscles are called into voluntary action, but at a later peried increased by all attempts to use the limbs; attended, in advanced cases, with some degree of rigid flexion of the trunk, arms and legs, and with a tendency to hurry in walking or to fall forward.

5. *Chorea or St. Vitus's Dance.*—A disorderly succession of more or less perfectly co-ordinated movements, occurring altogether involuntarily, and interrupting and frustrating the progressive motions of the limbs and body; often limited to the arm and leg of one side, or more severe in them than in the opposite limbs; occasionally replaced by incomplete paralysis, commonly attended with an altered mental state, and sometimes even with mania.

6. *Tetanus.*—Tonic spasms, occurring in paroxysms; beginning in the jaw or neck, and extending to the whole body; accompanied by extreme pain; in the great majority of cases, traumatic in origin, and fatal in result.

7. *Tetany.*—A tonic spasm, occurring in paroxysms lasting some minutes, or even longer; generally limited to the distal parts of the limbs; but in some instances extending to the face and trunk.

8. *Hydrophobia.*—Spasms chiefly affecting the pharynx and larynx; excited by attempts to swallow liquids; the result of infection by the bite of a hydrophobic animal; ending in death.

Spasmodic Tic.—*Histrionic Spasm.*—This is a disorder for which we have in English no very good designation. The French term it *tic convulsif*, while the Germans employ the name of *mimischer Kramf* for that form of it which affects the muscles of expression, or those supplied by the facial nerve. Dr. Sieveking, in translating Romberg, introduced the term *histrionic spasm* as an equivalent for the German name, but it has not been generally accepted, and it is liable to be misunderstood, and taken as meaning that actors are especially subject to the complaint. *Spasmodic tic*, therefore, is the designation which I shall employ.

Its essential feature is the occurrence, at longer or shorter intervals, of a sudden rapid involuntary contraction in a single muscle or group of muscles. One eye may be instantaneously closed and opened, or the forehead may be wrinkled on one side, and as quickly made smooth again; or the angle of the mouth may be twitched to one side, giving to the face the aspect of a meaningless grin. Or, again, the head may be made to nod forward, or the neck may be distorted to one side, or the shoulder shrugged, or the diaphragm may contract and cause the utterance of a sharp cry. Trousseau mentions that after the lapse of twenty years he recognized a former schoolfellow, who happened to be walking behind him, by a sort of barking noise that he made. The person himself is often unaware of the complaint; which is, indeed, scarcely more than a morbid habit or trick. Trousseau says that he was consulted by a lady whose three daughters had muscles in different parts of the body affected with spasmodic tic, for which she bitterly reproached them, but who did not know that she herself had it in her face. Another point which this writer mentions is that the movements sometimes shift from one part and attack another. Thus a patient came to him for violent spasms of the head and one shoulder; methodical gymnastic exercises were prescribed; and after some time the affection disappeared from the right shoulder (where it had been located) and presently showed itself in the left shoulder. A similar instance is related, by Sir Thomas Watson, of a gentleman who when young used to give an involuntary shake of his

head; a blister, applied to his throat for some affection of the air passages, made this movement painful, and the movement ceased; but to use his expression, it broke out in his nose, so that he was ever afterward in the habit of wrinkling it. I lately saw a boy, aged twelve, who four years before had acquired a trick of sniffing with his nose, and of coughing as though he would be choked; this was followed after two years by a habit of "making faces;" and a month before he was brought to me he began to toss his head over to the right side at intervals of a few minutes; whereupon the sniffing and coughing were given up, Sometimes, however, an affection of this kind lasts for a whole lifetime unchanged.

I do not know that it is ever influenced by medicinal treatment.

Spasmodic Wry Neck or Torticollis.—In describing spasmodic tic, I just now mentioned a form of it which consists in twitching of the patient's head to one side, this contraction (as is usual in that complaint) being sudden and passing off instantaneously, so as to leave the neck perfectly free in all its motions. But the cervical muscles are liable to another and a very different kind of spasm, which is called "wry neck" or "torticollis." This is characterized by a rapid succession of jerking movements, that draw the head with great force toward one shoulder, and give rise to extreme deformity, of which, however, the exact description differs somewhat, according as one or another muscle is chiefly involved. When it is the sterno-mastoid, the corresponding ear is pulled down toward the clavicle, while the chin is pushed upward, and the whole face is thrown to the opposite side. When it is the trapezius, the head is simply drawn backward, without rotation of the chin; the shoulder being at the same time raised. The fact that the larger half of the head lies across the median line on the unaffected side has often led to mistakes as to which side is really the seat of the spasm. But this is a matter of direct observation, and not merely of inference; one or more muscles in a state of powerful contraction can generally be felt, or even seen through the skin; and writers have asserted that they tend to undergo hypertrophy, while the opposite ones feel soft and are apt to become wasted. Sometimes, however, no such difference can be made out; one is then obliged to suppose that some of the deep-seated muscles of the neck are affected.

Spasmodic wry neck occurs chiefly in adults. Dr. Reynolds says that all but one of the patients whom he has seen have been more than thirty years of age, and the majority more than forty. However, one well-marked case has recently been observed at Guy's Hospital in a girl, aged eleven, who had had it ever since she was a baby twelve months old. Men and women appear to be affected in about equal numbers.

Origin.—This disease is often directly traceable to cold. Thus Dr. Golding Bird ("*Guy's Hosp. Rep.*," vol. vi) had a patient who was attacked shortly after having been thrown into a state of partial stupor by driving across an open country in a gig one severe winter's night. And Dr. Bright relates the case of a woman, who was sitting exposed to a draught of cold air, when she suddenly felt what she thought was "a nerve giving away" on the left side of her neck, whereupon her head was drawn to the right. I know of two instances in each of which the complaint appeared clearly to be the result of a fall upon the head. Very often, however, no exciting cause can be discovered. The patient, if a woman, may have been pregnant when the complaint began; or it may have followed a severe mental shock, continued worry, or (according to Erb) an attack of enteric fever; but none of these conditions precede it often enough to be definitely associated with its ætiology. Dr. Reynolds notes, as a significant fact, that he has not been able to trace it to overwork. But Dr. John Harley has reported, in vol. lvii

of the "*Med.-Chir. Trans.*," two cases in which this appeared to be the cause of wry neck, one being that of an engraver whose head was habitually kept raised and slightly rotated to the right, so that he might have an oblique view of the plate on which he was engaged. And, in confirmation of his opinion that it was this which gave rise to the affection, Dr. Harley mentions the case of a governess who spent much of her time in ruling the pupils' copy-books, and who used to rotate her head rhythmically and with emphasis from left to right; she, in her declining years, lost her control over this movement, and her face was twisted every second to the right shoulder, unless her attention was strongly engaged in some other matter.

Symptoms.—In cases not directly traceable to cold, the development of the complaint is generally very gradual. Dr. Reynolds describes the patient as at first feeling uneasy in the neck, and as thinking that something is wrong with his cravats or with the pillow, until after some months he discovers, or is told by a friend, that his head is not straight. During the early progress of the case, an ordinary observer might suppose that the man's shirt-collar was uncomfortable, and that he was trying to ease it by moving his neck to one side; or that he was looking over his shoulder, and endeavoring to get his head further round. Pain is at first absent, but after a time he complains of a dull, aching sensation, which extends to the shoulder, or even down the arm. At first, by an exercise of the will, he can overcome the spasm, so as to look straight before him, or toward the affected side; even at an advanced stage he can sometimes, by a very powerful effort, restore the equilibrium of the two sides for a moment, but at the cost of much distress, and with the risk of aggravating the severity of the spasms afterward. As the disease progresses, he generally gets into the habit of bringing his head into the proper position with his hands. Thus, a person to whose case I have already alluded, used to walk about with his arm raised, and ready to seize hold of his nose, which he employed as a kind of lever to control the movements. The spasms are always arrested during sleep, and they often cease when the patient is in the recumbent posture, and when he supports his head with his hands. They are at once brought on by any excitement, and by talking, or by attempting to walk. In the more severe cases the head is jerked about with extreme violence, and this goes on for hours or even for days without any interval. Sleep may be rendered impossible, and the state is altogether one of the utmost misery.

Spasmodic wry neck may remain stationary for years; neither advancing nor receding. But sometimes it extends from the parts first affected to the side of the face and the eye, or to the shoulder and arm. I know of two cases in which the movements began in the upper limb and attacked the neck secondarily. This and the fact that the deep cervical muscles are sometimes the ones mainly concerned, prove that the disease is not specially associated with the distribution of the spinal accessory nerve, as would appear from the description given by Erb and others. In some cases it passes off within a few months under some plan of treatment, and a long while afterward returns with its characters unaltered. Thus, in a case of Dr. Golding Bird's, already referred to, there was an interval of about seven years between the first and the second attack; and I have notes of another case in which the patient got well, returned to his work as a gas-fitter for thirteen years, and at the end of that time was again seized with the disease, but on the opposite side. I do not know that this neurosis ever, like spasmodic tic, deserts one part of the body in order to attack another immediately afterward.

In the advanced stages of the disease it sometimes, but not always, happens that the muscles which are the seat of the spasms fall into a state of tonic contraction, so that for hours together the head remains obliquely

drawn down to the shoulder but perfectly motionless. Even then, however, any kind of emotional excitement, or the slightest attempt at voluntary movement, generally leads to the development of clonic spasms. Some writers have also described tonic wry neck as a separate form, and it is the fact that in some cases there is no history of the occurrence of the ordinary jerking movements at any period. Thus a girl, aged seventeen, who was lately in Guy's Hospital under the care of Dr. Habershon, had her head drawn down so that the chin rested on the second left costal cartilage, and with the right side of the lower jaw so close to the sternum that it was difficult to introduce the finger between them; very slight twitching movements were all that had been noticed. But the affection was said to have developed itself while the patient lay paralyzed in bed, unable to move a limb, or even to feed herself, and this, perhaps, accounts for the absence of clonic spasms. It is, however, to be noted that the girl was the subject of well-marked hysteria, for it is possible that the wry neck was really an expression of that neurosis, and analogous to the contractions of the limbs, which (so far as I know) occur only in hysterical women.

Another cause of this form of wry neck is irritation from disease of the teeth. Two such instances are on record, in both of which the spasm seems to have been tonic. One is a case of Mr. Hancock's ("*Lancet*," 1859, i, p. 80), of a girl who for more than six months had her head drawn down nearly to the left shoulder; extraction of a stump and of a decayed tooth in the left side of the lower jaw completely cured her in a few days. The other case, which occurred in 1813, is related by Mr. Mitchell in the fourth volume of the "*Med.-Chir. Transactions.*" It is so far peculiar that the spasms began in the tongue and side of the face, and only affected the neck after the lapse of some days, but we have seen that precisely analogous modifications, in the extent to which different muscles are involved, occur in the ordinary clonic form of wry neck. The patient was a woman, aged fifty; during the attacks her neck was drawn round to the left shoulder, her arm was rigidly extended, her eyelids were widely opened, and the two eyes directed to the left, her mouth was opened and distorted to the left side, showing the clinched teeth; her tongue felt as hard as a board and was curved to the left side; her forehead was wrinkled, and all the muscles of her face were thrown into a state of rigid distortion. After three months the tonic spasm was succeeded by a rapid tremulous motion of the affected parts, and the attack passed off, to be repeated at intervals which rapidly grew shorter, so that a fortnight from the commencement of the disease the spasms became almost continuous. Extraction of some carious teeth and fangs in the left upper jaw was quickly followed by a cure. I am not aware that the ordinary clonic form of wry neck has ever been traced to local irritation of any kind.

Results.—One curious effect of wry neck, when it occurs in childhood, is the permanent deformity in the bones of the face to which it gives rise. In the girl to whose case I have just alluded, the left side of the face was considerably larger than the right. Thus, when the head was just straight, the right eye lay at a higher level than the left one, the median line of the face, instead of being upright, formed an obvious curve, with its concavity toward the left, and the left side of the upper jaw projected beyond that of the lower jaw, while on the right side the relation between them was reversed. The vertebræ of the neck were also distorted, there being a projection on the left side, apparently caused by the transverse process of the atlas. Precisely similar deformities were noted in the little girl, aged eleven, who had been in the hospital a few years before. They are of importance, because they might be regarded as indications of a primary disease of the bones, such as does, in fact, often cause a prominence of the cervical spine on

one side, and moreover is constantly attended with impairment of the movements of the head.

Treatment.—This is very unsatisfactory; on the one hand, many cases are altogether intractable and are not benefited by any curative measures; on the other hand, those in which relief is afforded, or which get perfectly well, yield to the most diverse remedies, which must differ altogether in their *modus operandi*, and each of which in turn fails more often than it succeeds. Dr. Reynolds speaks of galvanism as being very useful in cases which are not already of too long standing. He finds that a continuous current of moderate intensity, passed through the muscles which are the seat of spasm, causes them to relax, at least for a time. But he adds that even when by this means the head has been maintained in equilibrio for many minutes, day after day, he has often observed that, as soon as the poles are withdrawn, the spasms continue instantly to return. And sometimes it has been obvious that the ultimate effect of the treatment has been to increase the movements. In a patient, who some years ago came under Dr. Habershon's care at Guy's, a considerable amount of relief was afforded by the plan of fastening the sponges upon different parts of the neck, and leaving them for some hours at a time. Faradization of the muscles on the opposite side of the neck is sometimes useful, by increasing their power of resistance. On the affected side there is an exalted sensibility to interrupted currents; the patient may be altogether unable to bear the application of one which is so weak as to cause no pain whatever on the healthy side.

Rest is, of course, essential; and it would seem desirable that some mechanical support should be provided which may keep the head in its proper position. Dr. Reynolds, however, says that he has not yet seen any apparatus which a patient with confirmed wry neck could habitually wear, but that the appliance suggested by Dr. Hearne, of Southampton, is useful in recent cases, and may at least control the movements for an hour or two; as, for instance, when a clergyman wants to get through a particular service.

Another measure of which he speaks highly is the hypodermic injection of morphia. Beginning with a tenth of a grain, he recommends that one should, if possible, increase the dose until two or even three grains are administered twice daily.

In one case recorded by Dr. Radcliffe ("*System of Med.*," ii, p. 133), arsenic was injected subcutaneously with very striking results. From five to fourteen minims of Fowler's solution were thrown into the connective tissue over the affected muscles at intervals of three or four days or longer; they caused much local irritation and inflammation; but this was afterward diminished by diluting the solution with water. After the fourteenth injection, at the end of about nine weeks, the patient left the hospital almost well. This method of treatment has, however, been tried at Guy's by Dr. Wilks and by Dr. Moxon, but without success.

Dr. John Harley has recently ("*Med.-Chir. Trans.*," lvii) related two cases in which the administration of large doses of succus conii proved very useful. Beginning with an ounce of the liquid, he rapidly pushed the dose in each case until one patient took three and a half ounces once daily, and the other four ounces twice daily. The effect aimed at was the production of a general relaxation of the muscles, so that at the end, the second hour after swallowing the hemlock juice, the patient should be unable to rise from the sitting posture or to walk unassisted, and that there should be ptosis, impairment of power to masticate and swallow food, and slowness of speech. In the first case the spasm became greatly diminished; but the drug had ultimately to be discontinued on account of the mental depression to which it gave rise. In the second case the affection was almost cured, and the

patient resumed his occupation, working half time, with only an occasional tendency to twist of the head, if he became over-tired. Dr. Moxon has followed Dr. Harley's plan at Guy's with a decided measure of success.

Another plan of treatment, by which Busch succeeded in curing permanently three cases out of four, is the application of the actual cautery in lines, five or six inches long, to the neck on both sides of the spine, and maintaining suppuration for some weeks after the separation of the eschars.

Some years ago ("*Med.-Chir. Rev.*," 1866) Mr. Campbell de Morgan excised an inch of the trunk of the spinal accessory nerve, above the origin of the branches to the sterno-mastoid muscle, in one very severe case, with permanent relief to the spasm, but with equally permanent paralysis of the affected muscles. Another operation, which is performed several times with good results, is the subcutaneous section of the sterno-mastoid muscle.

In those cases in which the spasm is tonic, it is always advisable to straighten the head under chloroform, and to endeavor, by mere mechanical appliances, to prevent the return of the contraction.

FUNCTION SPASMS.—Within the last few years several observers have devoted attention to cases in which spasmodic contractions of various kinds are brought about by voluntary movement, the exciting cause in each instance being generally limited to some one particular action, so that the patient remains quite quiet when merely sitting or lying down, and is able to use all his muscles for other purposes perfectly well. The best paper on the subject is, I think, one by Dr. Weir Mitchell in the "*American Journal*" for 1876. His name for this group of affections is that of "functional spasms;" but this appears to be so liable to be taken as meaning "spasms which are functional and not organic," in which sense it would include all the diseases described in the last few sections, that I have thought it better to substitute for it the term "function spasms" which, if less euphonious, is also less misleading.

Many of Dr. Mitchell's cases are exceedingly curious. Two are examples of what he terms "lock spasm." A watchmaker, who often had to pick up and adjust very small screws, would find, ten or twelve times a day, that his thumb and forefinger suddenly became locked upon one of them, so that he had to release it with a loop of twine, not always without wounding the finger; even then the cramp would last for some considerable time longer. At a later period, when he was turning over the pages of a book, the finger and thumb would sometimes close with violence, so as to tear the leaf. Another man, a sawyer, was liable once, or even twice, a day to have the arm arrested, and fixed in a state of flexion, at the moment when his saw was drawn back to prepare for the downward movement. By no effort that Dr. Mitchell could exert was he able to overcome the spasm; on one occasion he made the patient bend over, so as to bring the forearm into a horizontal position, and he then found that for five minutes the biceps supported a weight of eighty pounds suspended from the wrist.

In other cases, the attempt to walk was accompanied by a tonic spasm of certain muscles, so that a kind of string halt was produced. Yet another patient, a journeyman tailor, had an extraordinary seizure whenever he jumped up suddenly from the prone or the sitting posture. The right leg was then bent at the knee, the left was thrown on it in violent flexion, the body and the head were twisted to the right; the right arm was extended and raised; the left was thrown outward and backward in extreme pronation. Then there was a general writhing of the whole frame, the face muscles twitching here and there; and the attack passed off with a groan of relief. This man could make every possible movement, slowly or abruptly as he pleased, so long as he remained seated or lying down; he was even

able to get up; and, after cautiously standing still for a moment, could walk away as well as any one else.

In other cases, again, spasms accompanied the act of laughing or talking, or, above all, chewing. One patient, as soon as he began to masticate his food, had his mouth jerked open, so that he had to keep shutting it with his hand. When he wanted to swallow, his face assumed an aspect of terror. "He would suddenly muster courage, and swallow the contents of his mouth at a gulph. Then instantly the jaw flew open, the head was drawn back and down upon the left shoulder, the face was convulsed, and sweat ran from the forehead." In another case of the same kind the gastrocnemius were involved in the spasm, so that the patient was jerked into a squatting position. I have myself seen an instance in which the chewing of food brought on a kind of wry neck, the head being twisted over to one shoulder; and another, in which the effort of speaking was attended with an extraordinary series of grimaces.

But the strangest of all are, perhaps, certain cases in which any attempt to stand excites violent convulsions. The patient is thrown off his feet, his legs pass into a state of clonic spasm, and he begins to execute a succession of the most rapid and irregular jumping and skipping movements, even though he may be supported on each side by another person. So great are his efforts that his face flushes, his pulse is quickened, and he breaks out into a profuse sweat. Yet, when he is lying or sitting, he remains quiet, and can move his legs perfectly well in all directions. In this affection a special name, that of "saltatorial spasms" was invented in 1859 by Bamberger, who recorded two examples of it. A few similar instances have since been observed by other German observers, and in England by Dr. Gowers, who has written on the subject in the "*Lancet*" for 1877. In their details these cases differ to some extent from one another, and this affords one an additional reason for purposing to include them in the wider group of "function spasms." Sometimes it has been possible for the patient to excite the movements by pressing with his feet against the foot board of the bed, even while he is lying down; in one instance, tickling the skin of the soles would elicit them; in another, pressure upon a tender part of the spine had the same effect. They have often been much augmented by emotional excitement; and this, I think, has sometimes led to doubts as to their genuineness, it being found that they are much less violent when no one was looking on; but the same thing may be said of many other affections belonging to the same class. Erb suggests that saltatorial spasms will ultimately be found to be exaggerated tendon reflex; but in one of Dr. Gower's cases traction on the *tendo-Achillis*, after the method described at p. 392, produced no ankle clonus. Dr. Gowers is no doubt right in thinking that the movements are due to the combined and simultaneous action of peripheral and voluntary stimuli upon irritable centres in the cord; but as the same stimuli are in action in all healthy persons whenever they stand upright, this really throws little light upon the question. In Bamberger's first case, that of a youth of nineteen, the affection came on during convalescence from acute pneumonia; recovery took place in about a month, under the internal administration of morphia. Other cases have been much more protracted; in one instance the spasm had not subsided when the patient died of fever, at the end of five years. There have been two cases in women, both of whom were affected with hysteria; one had several attacks of the spasm, each lasting some months; her recovery was once quite sudden, exactly as so often happens in hysterical contractures.

In some of his cases of function spasm, Dr. Mitchell found benefit from the injection of solution of atropine into the substance of the affected muscles.

WRITERS' CRAMP.—I have still to describe a class of cases which evidently belong to the "function spasms," but which differ from the rest in the circumstance that the involuntary motions that attend them are generally very slight and simple, being, in fact, only just sufficient to disturb the due execution of some highly specialized action, involving numerous and complicated muscular movements. A musician, for example, becomes unable to play the piano or the violin, whichever happens to be the instrument he may have chosen to excel in; a tailor or shoemaker no longer has the power to sew; a milkmaid ceases to be able to press the milk from the cow's teats; a smith, to bring down his hammer upon the anvil. I have even seen a man, whose whole occupation was to clean knives and boots and shoes for a large house, and who became incapacitated for this work, although he could still do everything else. Each of these cases, however, is exceptional; infinitely more numerous are those in which the complaint consists in an inability to write. And since "writers' cramp" has been studied much more thoroughly than the rest, it will be convenient that we should in the main confine our attention to it, premising that the description of it is generally applicable to them also, *mutatis mutandis*. Indeed, there is no one name which would conveniently include them all; that of "co-ordinated business neuroses," proposed by Benedikt, being too unwieldy to meet with acceptance.

The exact nature of the impairment in the power of writing varies widely in different cases of writers' cramp; hardly any two are alike. Sometimes the patient's complaint is that after he has been writing for a few minutes his hand and forearm feel wearied; sometimes that the fingers become suddenly extended so that the pen drops from his grasp; sometimes that the index finger is straightened and drawn off the pen handle; sometimes that the fingers are pressed too tightly upon it, so as to impede its movements. In many cases there is no visible cramp or spasm; all that can be seen by a looker-on is that the hand has its motions arrested in the act of writing. But in some instances there is obvious tremor, so that the point of the pen is shaken.

The handwriting is often altered in character; in some instances the change consists in a reduction of the size of the letters, the power to make free strokes failing, and the writing becoming "cramped;" other patients become unable to accomplish a "running hand," and are obliged to confine themselves to a slow "round text." I have had one patient who could do anything but short hand perfectly well, but unfortunately he was a law reporter. In the immense majority of cases, however, the peculiarity consists in an uncertain tremulous formation of the strokes which make up the letters. Indeed, when the affection is severe the handwriting becomes altogether unintelligible, consisting of a meaningless succession of shaky lines and curves.

Persons affected with writers' cramp adopt all sorts of odd devices to diminish the inconvenience which the complaint causes them. Some will write only with a quill, and, so far as it goes, this is undoubtedly a good plan, for the muscular effort required is very much less than with a steel pen. Others employ a large, thick pen holder, or fix a nib in a broad piece of cork. Dr. Poore ('*Practitioner*,' xi) mentions the case of a man who used a rounded mass of wood of the shape of a boy's top, which he held tightly within the hollow of the palm of his hand. Others grasp a pen holder with the closed fist, or fasten it to one finger by means of a ring.

Sometimes, however, the fault seems to be not so much in the hand as in the wrist and forearm. A patient of Dr. Poore's said that the first thing he noticed was a difficulty in bringing down the hand upon the paper, and for three days he forced himself to write by holding down the right wrist with the

left hand, but "at the end of this time that resource failed him." Indeed, one of the most important characteristics of "writers' cramp" is that it is a progressive disease. For a time a person affected with it may succeed in accomplishing the art of writing by bringing different sets of muscles into exercise. Thus, instead of employing the weak muscles of the thumb and fingers, he may use those of the forearm; or he may place the arm in some extraordinary attitude as soon as he takes up the pen, resting, perhaps, his wrist upon the table, and raising his elbow at an angle; or pressing the limb close to the side of his body; or, again, straightening the elbow, and holding the instrument at arm's length. But if he persists in writing much all these resources soon fail him. The muscles which he has called in to his assistance "give out" in their turn. Even if he should have taken the pains to learn to use the pen with the other hand, that, too, is after a time attacked. At advanced periods of the disease, the muscles are often affected with spasmodic contractions of considerable violence, independently of all efforts at writing. Dr. Poore's patient, to whom I have referred, complained that his hand was always poking about; it would sometimes bounce suddenly out of the side-pocket of his coat while he was walking in the street, and he once broke a jug in consequence of an unexpected spasm while he was pouring out some water. I had a patient who told me that his shoulder invariably gave a jerk when he put his hand up to his head. Lastly, in some cases, the spasm extends to the muscles of the neck and body. Dr. Reynolds mentions an instance in which the effort to write caused the head to be drawn downward to the right shoulder and the trunk to be contorted so as to be concave on the right side. The same writer also states that he has seen torticollis, occasional strabismus, stammering, and palpitation of the heart, associated with writers' cramp.

There is not generally any impairment of sensation. Sometimes the patient complains of "numbness," or "tightness," or "coldness," or of some peculiar sensation which he cannot define, in one particular finger, or running from some part of the hand up toward the arm. In some rare cases actual anæsthesia is said to be present. Very commonly the act of writing, if continued for any length of time, causes the hand and wrist to ache, or even the shoulder or the spine. These abnormal feelings distress the patient to an extent which seems altogether disproportionate to that of the actual pain which he has to endure. Indeed, a peculiarity is often observable in the psychical state of those who are affected with writers' cramp in its more advanced stages; they are highly irritable and excitable, and very despondding about their complaint. Dr. Poore speaks of one man as having been apparently on the verge of suicide, and of another as having actually taken up a knife with his left hand and stabbed himself on the wrist. Such patients are generally nervous, and they are less than ever able to write when others are looking on, or when the subject matter is of special importance. Dr. Poore was told by one person that he got on better when copying than when composing what he wrote, and another patient had to watch the point of his pen, for if he looked away from it his handwriting at once became unsteady. Those who are affected with this disease are also said to sleep badly, and to be generally ill nourished, with soft, flabby muscles. Dr. Poore attaches some importance to a thin, brittle state of the nails which he has often noticed.

It is generally assumed that the cause of writers' cramp, and of the affections allied to it, is overuse of the muscles, or working too hard and too long at that particular kind of labor, which becomes accompanied with the spasm. And it is undoubtedly true that most of those who are attacked are clerks, or accountants, or in some way professionally engaged in writing. Moreover, as Dr. Poore points out, the commencement of the disease often

dates from the time at which some heavy task has been accomplished, as when an architect has worked against time to complete the details of a plan, or a lady to finish within two or three days an etching for a bazaar. Still, it is also the fact that some individuals strain their powers to the utmost, and get through extraordinary amounts of writing, without ever suffering from it; and others are attacked who have at no time over-exerted themselves in any way, or have actually done less writing than is safely accomplished by the majority of persons. I have seen two such cases; one in a boy, aged fifteen, whose education seemed to have been rather neglected than otherwise; and one in a lady of fashion, who had at most to write a few letters and notes to her friends and relatives. The cause of the disease must then lie mainly in the nervous organization of the individual; indeed, Dr. Poore speaks of an instance as having come under his observation in which three generations in a direct line were affected. One of his patients, too, referred the complaint to the severe shock caused by an alarm of fire.

Pathology.—The seat of this neurosis is doubtful, and there are two different theories with regard to it. One, which is supported by Reynolds and by Erb—and which seems to me the more probable—is that it lies in the central ganglia which effect the association and co-ordination of muscular movements for the more complex actions. The nutrition of these structures is supposed to be impaired, as the result of over-exertion, and the consequence is a perversion of their functions, which expresses itself in irregular spasmodic movements and in the other symptoms of the disease. A strong point in favor of this view is the fact that when one arm has been affected the other is often afterward attacked; and the relations which writers' cramp bears to torticollis and other spasmodic neuroses afford a further argument in the same direction. The other theory, which was first proposed by Zuradelli, and which is ably advocated by Dr. Poore, is that the starting point of the morbid change is exhaustion of some particular muscle or set of muscles—generally one of the small intrinsic muscles of the thumb or fingers—muscles which are kept constantly in action throughout the whole time during which a pen is held in the hand. One or more of these, it is suggested, begins after a time to respond sluggishly, or not at all, to the stimulus of the will. The patient then unconsciously calls into play other muscles, generally those of the forearm. In their turn, these, too, become worn out; and the process of substitution is carried on indefinitely, and always with the same result. Dr. Poore has in a large number of cases of writers' cramp tested the electric reactions of the muscles which are specially engaged in the act of holding a pen, and has found that one or more of them often showed a marked impairment of irritability as compared with those of the opposite limb. It might be thought that a powerful argument against the theory in question is afforded by the well-known and remarkable fact that a patient who is severely affected with the disease is often perfectly well able to perform all other actions. But Dr. Poore argues that the utility of the affected muscles is only apparent; the hand and forearm, he says, are so bountifully furnished that for most movements there are more muscles than are required. If a man finds some of them to be weak, he unconsciously substitutes others in their place, to do the work required to be done. This writer maintains that certain among the more delicate actions of the hand are generally interfered with besides those which give to the case its principal character. Thus one of his patients could no longer hold a knitting needle; another was unable to take up her dress between her thumb and her forefinger; and a third could not toss over a letter into a letter-box. So, Erb states that the sufferers from writers' cramp may be unable to button their clothes, to work embroidery, or to

play upon the piano. One of Dr. Poore's patients had great difficulty in feeding and in dressing himself. Benedikt has proposed to distinguish different forms of the disease, namely, a spasmodic, a tremulous, and a paralytic form; but, so far as I can judge, such divisions are altogether arbitrary.

Diagnosis.—The diagnosis of writers' cramp, or of the allied forms of "function spasm," is tolerably easy when they have reached an advanced stage. But one must always recollect that impairment of the power of writing may result from a number of other affections of the nerves and muscles of the upper limbs. Progressive muscular atrophy, neuritis of the ulnar, the median, or the radial nerve, compression of either of these nerves by a tumor of whatever kind, may each give rise to this symptom. Dr. Poore mentions a case of subclavio-axillary aneurism in which the first thing noticed by the patient was that he could write only with great effort, and then illegibly.

As a rule, a person affected with writers' cramp goes on without medical advice for a long time, struggling against what he deems a foolish incapacity to carry on his duties. But when it does happen that one is consulted at a very early period, there may be great difficulty in determining whether the disease is really present or not. This is particularly the case if the patient is a nervous professional man, whose mind has for some time been dwelling upon the subject; or if he is a bank clerk, who has heard all about it from his fellow officers, and who has perhaps been reading a number of medical books. Such persons come to one complaining that the act of writing causes a number of unpleasant sensations; and one may be in great doubt whether to laugh at their complaints or to take them seriously. It seems to me that the best indications are afforded by the effects of rest. If the supposed symptoms show themselves only at the end of a hard day's work, and are entirely gone the next morning or after the interval of a Sunday, they are not likely to be of serious consequence. Thus Dr. Poore speaks of an eminent pianist who, after practicing for a considerable time, suffered from an inability to adjust the movements of his fingers, so that he could not always strike the right note, but who, a few hours later, could play as well as ever. In such a case it is evident that there is nothing seriously wrong. But I believe that I have seen one instance of true writers' cramp at an early stage, in which the patient said that his hand felt perfectly well every morning when he first got up.

Prognosis and Treatment.—In most cases a right diagnosis is of the utmost importance, for on it may depend the whole future of the patient. At this period the discontinuance of all writing—or of whatever other action may be the exciting cause of the complaint—may completely cure it in the course of a month or two. Even then, indeed, there is always a risk that the disease may return if the muscles should be again overworked. But, most commonly, the prognosis that must be given is very unfavorable. At an advanced stage of the disease there is scarcely any chance of recovery, unless the patient can altogether give up his occupation for six months, or even for a whole year. And in many cases treatment fails entirely, the disease advancing steadily, in spite of all that can be done. The only remedy appears to be galvanism. Of this Erb speaks highly, recommending the employment of ascending currents along the cervical spine, as well as peripheral galvanization of the nerves and muscles specially concerned. But I am not aware that any results have been obtained by these methods which can be compared with those which Dr. Poore got by a method of his own; this consists in making the patient perform rhythmical movements with the affected muscles, while a continuous current of moderate intensity is passed through them. His first case was that of a man, aged thirty-two, who had suffered

from writers' cramp for more than nine years, and in whom it presented its severest form. Dr. Poore exercised separately the deltoid, the biceps, the triceps, and the other muscles, as well as those of the hands and fingers. When the treatment was commenced, the patient could not finish writing the word George; at the end of five months he was able to write a whole epistle, forming his letters well and firmly. He had been unable to feed himself; he could now do all that he wanted, and had embarked in a small business. Another of Dr. Poore's cases is little less remarkable. A man, aged forty, had for four years suffered from the disease, and had for six months been unable to write, the attempt to do so giving him great pain. After the first application of the current this pain disappeared, within a week he could sign his name with tolerable ease, and at the end of ten months his handwriting looked as good as it had ever been, although he was not at that time able to hold his pen quite properly and tightly between the thumb and fingers. Unfortunately, however, Dr. Poore records no similar successes in his later papers on the subject.

Bromide of potassium is said to be sometimes beneficial to patients affected with writers' cramp, by removing the depression and irritability from which they suffer. Strychnia is often injurious. Stimulating liniments and douches appear to be almost, if not quite, useless. I have already mentioned that using a quill instead of a steel pen is frequently found to be of great service. Erb further says that many patients are relieved by applying a narrow bandage or strip of plaster round the wrists. This cannot, however, be expected to do any good where the only muscles affected are the little muscles of the thumb and fingers.

Paralysis Agitans.—Another disease whose precise seat in the nervous centres is uncertain is that known as "paralysis agitans," or "shaking palsy."* It consists in a more or less violent oscillatory movement, affecting especially the limbs, but sometimes the neck and the tongue also. It almost always begins in one of the upper limbs; the movement may at first be very slight, in fact, scarcely more than a tremor. Charcot says it is sometimes for a time limited to one thumb. In the beginning the patient commonly attaches but little importance to it, but gradually the space occupied by the oscillations becomes greater and greater and the whole limb is involved in them, so that he is unable to go on with his work and has to seek medical advice. Sometimes, however, the agitation of the muscles is severe from the very first. Charcot speaks of this as the result of a sudden shock or terror, but I have notes of two cases in each of which the patient went to bed as usual and woke in the morning with the disease fully developed, the right arm being in one instance the part affected, in the other both the left arm and the left leg. In two other cases that have recently occurred at Guy's one side of the neck has been the earliest seat of the movements. After a variable interval—generally after some months—the oscillations appear somewhere else, most frequently in the leg of the same side as the affected arm, but sometimes in the other arm. Presently the remaining limbs suffer in their turn. The head also begins to shake, and the tongue becomes very tremulous when it is protruded, or even when it is lying within the mouth, but nystagmus, or oscillation of the eyeballs, seems never to occur, nor do the jaws ever take part in the movements.

During the early part of its course, paralysis agitans is almost always paroxysmal. Each attack generally lasts some hours, and it is followed by a feeling of great fatigue, which gradually passes off in the interval of rest before the next one begins. At a later period the movements become continuous.

* [Abroad it is commonly known as "Parkinson's disease."—Ed.]

The oscillations bear to voluntary motions of the affected parts a relation on which great stress has been laid by successive writers, but which is hardly so simple as it has been represented to be in their definitions. Mr. Parkinson, who in 1817 first described "the shaking palsy," makes it a point that the movements occur "in parts not in action, and even when supported." He remarks that this distinguishes the disease from the mere tremor of old age, as well as from other complaints, among which is that caused by the vapor of mercury; and it is the fact that during the early period of the disease the hand may become perfectly steady when it takes up a tool or a pen, and that the patient is able to control the agitated limb for some minutes by an effort of his will. Moreover the oscillations often go on without intermission when the elbow rests upon the knee. But, on the other hand, I believe that in most cases at this stage the affection is dormant so long as the patient lies quietly on his back in bed, and it is certain that no movements occur during sleep except in the most advanced period of the disease, when (according to Parkinson) the rest may be entirely broken by them. This writer also mentions another curious fact, namely, that one of his patients had an intercurrent attack of right hemiplegia, in which the face was drawn to the left side, and this lasted a fortnight; during that time neither the arm nor the leg on the paralyzed side was in the least affected with the tremulous agitation, but it returned as the limbs regained their power. In the later stage of shaking palsy, indeed, the attempt to perform any voluntary movement brings on the oscillations with greatly increased violence. They are also much augmented by emotion or excitement. Even the presence of a looker-on often affects such patients to a degree that seems extraordinary; a man who can write quite well when alone may be unable to form a letter while he is being watched by a clinical clerk. A patient of mine found much relief from keeping his arm in a sling, which greatly diminished the oscillations. In him, as in some others, I observed that grasping the agitated limb with my hand, so as to check its movements, led to a marked increase in them and to the development of similar movements in the opposite arm, which was less severely affected with the disease.

According to most writers there is in paralysis agitans, besides the oscillatory movements, a certain degree of impairment in the muscular power of the affected parts. Eulenberg, indeed, has found that even in severe cases already of long standing the reaction to induced or galvanic currents is perfect, and both Trousseau and Charcot have taught that the muscles really retain their full force. The former writer relates a case in which the patient was able to exert much more power with the affected hand (which, however, was the right one) than with the other which was healthy—considerably more power than Trousseau himself could exert. But this must not be taken as showing that the disease had actually augmented the force of the limb, since it was not known what the man's strength had previously been. Another point observed in that case was that the contractility of the muscles was very quickly exhausted. When told to open and shut his hand in quick succession the patient at first moved rapidly, but after scarcely fifteen seconds more slowly, and soon not at all. This point also is adopted by Charcot, and he further remarks that there is often a retardation of the influence of the will, there being, for example, an unduly long interval between a thought and its expression in words. Recently, however, M. Bourneville, the editor of "Charcot's Lectures," has made on six patients observations with the dynamometer which appear to show that their strength was really diminished to a considerable extent.

One thing which impedes voluntary movements in the more advanced

stages of paralysis agitans is the occurrence of rigidity in the muscles of the affected parts. Charcot has pointed out that the patient commonly maintains an attitude which is characteristic of the disease. The head is bent so that the chin approaches the sternum, and he can with difficulty raise it or turn it to the left or right. The body is almost always bowed forward when he is standing. The elbows are generally drawn slightly away from the chest, the forearms are partially flexed, the hands, which rest together upon the waist, present a deformity somewhat like that which occurs in osteo-arthritis, the three inner fingers being inclined toward the ulnar side of the hand, and their joints being alternately extended and flexed, while the thumb and forefinger are stretched out and brought close together, as in holding a pen. In the lower limbs the rigidity is sometimes very marked; they are semi-flexed, the knees are brought together by a movement of adduction, the feet are curved inward, as in talipes equino-varus, the toes are arched like the three fingers. Lastly, according to Charcot, the features present a peculiar immobility and want of expression. There is no actual impairment of articulation, but the speech is slow and jerking, as though a considerable effort of the will were needed for the pronunciation of each word. Charcot remarks that it is like that of a person who, unaccustomed to ride, is on the back of a trotting horse.

The way in which the head and body are bent forward in the more advanced stages of paralysis agitans accounts for a symptom which Parkinson noticed, namely, that the patient tends to fall upon his face when he attempts to walk, and that his steps are consequently hurried, so that he runs instead of keeping to his ordinary pace. This had, indeed, been previously described by Sauvages, but as a separate complaint, under the name of *Scelotyrbe festinans*.* The man has been said to be constantly trying to overtake his own centre of gravity. Parkinson mentions a case in which an attendant was obliged to walk backward in front of such a patient, with one hand on each of his shoulders, to prevent him from falling. Moreover, when he first gets up from his seat, which he does very slowly, he makes a few hesitating and ineffectual steps before he seems to be able to start off, and in walking he treads only upon his toes, being unable to bring his heels to the ground. But the most remarkable circumstance of all is that some persons affected with paralysis agitans walk backward without intending it, and when they mean to go forward. Some years ago a striking instance of this occurred in my out-patient practice at Guy's Hospital. The patient, after a few ineffectual efforts to rise from his chair, would stand up, pause, give two or three abortive attempts at starting, and then succeed in making a few steps toward the door, when suddenly he found himself hurried against his will backward into the umbrella stand in the corner of the room. 'I was reminded of nothing so much as the way in which the engines of a steamboat can be instantly reversed. Charcot met with a case in which he could at any time induce such retrograde movements by unexpectedly giving a gentle pull at the patient's dress when she was standing up.

In the more marked cases of paralysis agitans the movements of the hands are not merely oscillatory, but resemble, to some extent, such as might be purposive. Charcot points out that the thumb may be carried over the fingers, as in rolling up a piece of paper, and that the fingers may be brought together, just as though a morsel of bread were being broken into crumbs.

Profuse sweating is an almost constant symptom when the oscillatory movements are at all severe. The patient may become so bathed in perspiration as to have to change his clothes many times a day. Charcot

* [*Σκελοτύρβη* (staggering) occurs in Strabo, Pliny and Galen. It has been also applied to chorea.—Ed.]

also mentions that he often suffers greatly from a feeling of heat, especially about the epigastrium and in the back, so that he insists on being kept lightly covered. But with the thermometer the temperature is always found to be normal.

In most cases, limbs affected with paralysis agitans have their cutaneous sensibility unimpaired. Charcot, however, says that a feeling of pins and needles in the hands and feet is sometimes complained of; and I find this mentioned in one case of which I have notes, the patient also saying that he was unable to feel the ground. Headache and vertigo are not uncommonly present; and at an advanced period of the disease there may be loss of memory, and even delirium.

Paralysis agitans is a disease which runs a very slow course. Charcot speaks of it as sometimes lasting thirty years. Toward the end, the patient falls into a very sad condition. The movements, which are now incessant, at least while he is awake, may be so violent as to shake the bed, and even the room in which he lies. He is unable to get up, or to dress or feed himself, without assistance. His speech may become unintelligible. His mouth remains open, and the saliva runs from it. His fæces and urine are passed involuntarily. Bed sores may form, and he may die of sheer exhaustion, or he may be carried off at an earlier period by pneumonia or some other intercurrent disease. Charcot remarks that a few hours before death the movements sometimes cease entirely.

Pathology.—As I have already observed, it is still uncertain whether paralysis agitans should be regarded as an affection of the brain or of the spinal cord. Parkinson supposed its seat to be in the cervical region of the cord, extending up to the medulla oblongata. But, perhaps, indications adverse to such a view may be found in the fact that after one upper limb the corresponding leg is generally next affected, rather than the opposite arm; and still more in the cessation of the oscillations during sleep, and throughout the continuance of a hemiplegic attack, so far as the limbs paralyzed are concerned. Hitherto anatomical investigations have thrown no light on the question. I have made one autopsy, that of a woman, aged forty, who died of phthisis after having suffered from paralysis agitans for eight years; for a year her speech had been impaired, and at the last it was unintelligible. The only unusual appearance that I could fancy I detected was that the substance of the pons toward the floor of the fourth ventricle looked unduly gray. Charcot has been able to examine the nervous centres in six cases; in three they were perfectly healthy; in three they presented slight microscopical changes which were believed to be merely senile. In some cases recorded by other observers there has been an obvious atrophy of the brain, but only such as is commonly found in persons of the same age as that to which the individuals in question had attained.

Age.—Paralysis agitans is by no means a common complaint. During the ten years from 1866 to 1875, I believe that only about fourteen cases were admitted into the wards of Guy's hospital. It is not often seen in persons who are not already advanced in years. Parkinson speaks of it as seldom occurring in persons below the age of fifty; but I find that among the fourteen cases at Guy's, there were seven in whom it began at an earlier age, and in three of them before forty years had been reached, one being thirty-six, another thirty-two, and the third but twenty-one years old. This last instance is not without precedent; for Charcot mentions a case of Duchenne's in which the patient was only twenty, and one of Fioupe's which occurred in a girl of fifteen or sixteen who had been terrified by a bombshell during the siege of Paris. On the other hand, in six of my fourteen cases, the disease began between the fiftieth year and the fifty-ninth, so that there was only one in which the patient was still older when

first attacked, namely, at the age of sixty-four. It is, therefore, a great mistake to suppose that shaking palsy is essentially a senile disease. But, on the other hand, I am not sure that a strict line of separation can be drawn between it and the tremor which is so common in old people, and which (as Dr. Maclachlan found among the inmates of Chelsea Hospital) has little or no tendency to shorten their lives. He even mentions one case in a pensioner, aged 107, who had been affected with it ever since he was sixty. But it may be that the same affection, which in young subjects is progressive, is in older persons comparatively stationary, or runs so slow a course that death overtakes them before it has had time to develop itself fully. Dr. Handfield Jones has, indeed, expressed an exactly opposite view of the matter, namely, that paralysis agitans in young subjects is a less serious form of the disease, and curable; whereas, in older patients, it is incurable. But I cannot find any evidence in support of such an opinion; at any rate, no satisfactory results have been obtained from treatment in the cases, occurring at a comparatively early period of life, which have been observed at Guy's Hospital, or which are recorded by writers on this subject.

Charcot says that paralysis agitans is equally common in women and in men; but this is a mistake. In all Parkinson's cases, and in twelve out of my fourteen cases, the patients were of the male sex.

Cause.—The disease seems frequently to arise without any definite exciting cause; but sometimes it follows close upon some violent shock of terror or other emotion. Charcot says that in many of his female patients it developed itself during the political commotions which have agitated France. The prolonged action of cold and moisture has been mentioned as occasionally giving rise to paralysis agitans, but apparently on no very good grounds. It has sometimes followed a local injury, as in a case of Charcot's, that of a lady who severely bruised her left thigh in falling from a carriage, and in whom, shortly afterward, that leg began to shake, and at a later period all the other limbs.

The *diagnosis* of paralysis agitans is seldom difficult. A disease which was until recently confounded with it is the insular or multiple sclerosis, described above (p. 483); the points of distinction between them were there fully stated. One must not forget that local organic disease of the brain—a tumor, for example—may give rise to paroxysmal attacks of spasm in one arm, or in one arm and leg, that in themselves are not unlike those which occur at the commencement of paralysis agitans; but the history of the case and the other symptoms will generally prevent one from making a mistake in this direction. Hysteria may simulate it. I have notes of a girl, aged eighteen, in whom the right arm began to shake three weeks after a fright. When she was admitted into the hospital it was in a state of continuous agitation, and if it was held the other limbs began to move in a similar way. However, she had had a screaming fit the very day after her complaint began, and she had globus and headache. She was treated with frictional electricity, sparks being taken from her body, and she quickly recovered.

As regards the *treatment* of paralysis agitans, it appears to me that at an early stage, when one limb only is affected, it would be well worth while to try the effect of keeping it at absolute rest for a period of several weeks; if necessary, by the aid of a mechanical appliance. I do not find that this plan has hitherto been tried.

Even at an advanced period of the disease recovery sometimes takes place, but whether in consequence of medical treatment or spontaneously it is hard to determine. Dr. Elliotson supposed that he cured one case with the subcarbonate of iron; Brown-Séquard, that he cured another with the chloride of barium. I know of two cases in which the last-named

salt was given, in doses of gr. ¼ to gr. j. In one instance it seemed useless; in the other marked improvement took place, the patient (who was under Dr. Moxon's care) being able within six weeks to walk twice the length of the ward merely holding a nurse's hand, whereas he had been so helpless as to be unable to get in or out of bed, and for five years he had had to be dressed by others. A man to whom I gave two grains of valerianate of zinc three times a day improved very considerably, but I did not know whether the result was due to the medicine or to the quiet and freedom from excitement which his residence in the ward secured for him. He went out of the hospital for a time, and the noise of a passing wagon, as he was walking home to Bermondsey, brought back the jerking movements, which had ceased entirely for some days. Dr. Ramskill (Syd. Soc.'s Translation of "Trousseau," vol. i, p. 449) had a well-marked case in which recovery occurred, after the failure of other treatment, under the administration of four-minim doses of the phosphorized oil of the Prussian pharmacopœia, with one drachm of cod-liver oil three times a day.

A plan recommended by Eulenberg consists in the subcutaneous injection of the liquor potassæ arsenitis diluted with two parts of water. The quantities which he employed would correspond with from 1½ to 2½ minims of Fowler's solution. He says that in one case four injections produced a decided diminution in the movements, and that in another case a similar result was produced by fifteen injections, and was maintained for at least two months. But Charcot has made trial of this treatment, and found it useless.

Eulenberg says that he has employed galvanism without any benefit—the constant current passed through the head or along the sympathetic nerves. We have at Guy's had one or two cases in which the application of this form of electricity down the spine caused at least a temporary improvement. The most striking was in a patient of Dr. Habershon's. It was first found that galvanization for ten minutes was attended with marked benefit for the time. The poles from a Cruikshank's battery were, therefore, fixed upon the neck for three hours without intermission. After this treatment had been continued for some time the limbs became much steadier, but he was not cured.

5. Chorea.—Like some other diseases, this is now universally known by a name which, etymologically, is by no means appropriate, and which was at first applied to quite a different complaint. The *chorea Sancti Viti* was originally the dancing mania which prevailed in certain parts of Germany in the fourteenth and fifteenth centuries. It is said that the designation is first met with in the account of an epidemic at Strasburg, in 1418, when those who were attacked were sent by the authorities to the chapel of St. Vitus, at Zabern, to be calmed by religious processions and masses. To such cases I shall hereafter refer when speaking of hysteria. They are altogether unconnected with the disease which I have now to describe, and to which the name of chorea was first applied by Sydenham in his posthumous work, the "Processus Integri." The use of so old an appellation in a new sense could not but tend to confusion, and for a long time afterward Sydenham's chorea was distinguished from the other as "chorea minor," or by the Germans as "chorea Anglorum." But, as I have already stated, the other meaning has altogether died out, and not the slightest ambiguity can now arise from our employing the name without any qualifying epithet. The corresponding appellation in English is "St. Vitus's dance."

Symptoms.—These are chiefly two: an inability to keep at rest while awake, and an incapacity for performing voluntary movements with precision. If the patient means to sit or to stand still, she (for chorea

is more common in females than in males) begins after a few seconds to fidget. She scrapes or shuffles her feet over the floor, or she throws one foot over the other, or twists it first outward and then inward. Or, perhaps, she lays her hand palm upward upon her lap and then suddenly reverses it. Or she may shrug up one shoulder, or throw it forward, or open and close her mouth or her eyelids without purpose. Von Ziemssen remarks in his "Handbuch" that such involuntary motions are often quickly followed by similar voluntary ones which the patient makes in the hope of concealing her complaint. But she probably is herself unaware of some of the movements, as, for instance, when a squint develops itself, or when her eyeballs are jerked from side to side. If one asks her to show her tongue she often seems at first unable to put it out, but afterward she suddenly thrusts it forward and then as suddenly withdraws it, her jaws snapping together in front of it. If she wishes to carry a cup to her mouth she cannot help throwing her arm in various directions, and accomplishes her object only after several unsuccessful attempts, and then she, perhaps, seizes the edge of the vessel with her teeth, and is obliged to gulp down all its contents at once, for fear of spilling them. When she tries to walk she moves by fits and starts, and jerks her body and limbs first to one side and then to another. Rosenthal and Benedikt have tested the reaction of the muscles to faradic and galvanic currents, and have found their excitability greater than under normal conditions.

The respiration is disorderly, the rate at which the inspiratory efforts succeed one another being very irregular. The articulation is apt to be hurried. The patient, if told to count, may give out several numbers, one after another, with explosive violence, and then pause to take a deep breath; or she may utter only one sound with each expiration, drawing in air hastily before she goes on to the next. Von Ziemssen says that he has seen with the laryngoscope unsteady and quivering movements of the muscles of the glottis, and that an imperfect degree of tension of the vocal cords is shown by the low pitch and monotonous character of the voice, and by the short space of time during which a note can be kept up in singing. Romberg relates a case in which there was an annoying hiccough, and two other instances in which inspiration was attended with a whistling or snapping noise; in one of them the chest was observed to be suddenly drawn inward by spasm of the intercostal muscles. According to Trousseau, deglutition is sometimes impaired.

During sleep the choreic movements cease. Jaccoud also remarks that in slight cases they sometimes remain absent for a little while after the patient awakes. Marshall Hall, however, is said to have observed that if she dreams they may for the time return.

When chorea is slight or of moderate severity, the spasms are often confined to one side, so far as the limbs are concerned. The disease is then sometimes called "hemi-chorea," but a special name is hardly needed. Dr. Hughlings Jackson has pointed out that the muscles of the trunk and face are always affected bilaterally, a fact which I shall presently show to be of considerable theoretical interest. Different writers have made different statements with regard to the relative frequency with which the right and the left limbs are affected. According to Dr. Jackson, the former more commonly exhibit the movements; according to Jaccoud, the latter. Dr. Austin also remarks that one may often at the first glance recognize a child affected with slight chorea from her sitting with her right hand grasping her left wrist, to keep it still. And among thirty-three cases in which the affection was unilateral, Dr. Pye-Smith found that there were eighteen on the left to fifteen on the right side.

The sensibility of the skin is generally said to be unaffected, but Trousseau declares that it is almost constantly disordered, the patient experiencing

formication and tingling, especially on the affected side, or even anæsthesia. I find that numbness is not uncommonly mentioned in the reports of cases admitted into Guy's Hospital. Choreic patients often complain of headache. Sir Thomas Watson remarks that he has sometimes known the pain to be limited to the side of the head opposite to the limbs which presented the movements. The pupils are generally torpid and rather widely dilated. In one case Von Ziemssen observed that the pupil was much larger in the eye corresponding with the affected arm and leg. In one patient Rosenthal noted the fact that the pupils returned to their normal condition when the chorea subsided, a proof that the dilatation was really an effect of the disease.

The pulse is commonly quickened, especially when the movements are severe. Some writers have asserted that the heart's action may be irregular or intermittent, but this is altogether exceptional. On auscultation over the cardiac region a blowing *systolic murmur* is often audible. With regard to the significance of this sign widely different opinions have been held. Many observers have maintained that the bruit is functional; some that it is anæmic, others that it is due to choreic spasms of the musculi papillares interfering with the due closure of the mitral valve. But, as we shall presently see, when the disease happens to terminate fatally, that valve is almost invariably found to have vegetations upon it which are identical with those that are present in acute rheumatism. And since the murmur, also, is the same in the two diseases, one cannot but attribute it to the endocarditis in the one as much as in the other. In some cases no bruit is audible. Its absence, however, is not to be taken as a proof that the valve is not inflamed. Dr. Kirkes and Dr. Wilks have both recorded instances in which vegetations were found after death, but in which the heart-sounds during life had been natural. Again, in some cases in which a murmur is present it passes off as the patient recovers from the chorea. This fact has been regarded as a proof that at least in these cases the sound is due to functional disturbance and not to valvular inflammation; but such a conclusion is not warranted, for a precisely similar disappearance of the murmur is often observed in cases of acute rheumatism.

The bodily temperature is almost always normal. Dr. Woodman, however (Wunderlich, *Syd. Soc's. Trans.*), says that in weakly children he has found it as low as 97° or 96°, and, on the other hand, he quotes a case of Dr. Finlayson's in which the average evening reading of the thermometer was 103.2°. The urine, if it deviates from the normal state, is commonly scanty and high colored, in marked contrast to the pale fluid which is passed so abundantly by females who are hysterical. According to Dr. Bence Jones there is an excessive secretion of urea, and others have said that the amount of urates is increased. Analyses made in two cases by Dr. Handfield Jones ("*Clin. Trans.*," iv) yielded contradictory results.

In some cases of chorea, however, the symptoms are but little marked at its commencement; and even throughout its whole course the patient may exhibit comparatively slight movements. A much more prominent feature may then be a want of power in one or more of the limbs. Thus, she may complain that her arm feels heavy, or she may drag her arm slightly in walking. Such cases are often brought to one as paralytic. In other instances she merely has a trick of bringing a particular set of muscles unexpectedly into action, so that she makes a grimace, or throws her hand or arm into some extraordinary position. Or she may let a jug fall from her hands and get broken, and this may happen two or three times in succession in the course of a week or ten days. Or, if she is a child, it may be noticed that she does not write her exercises so well as she used, or that she no longer plays steadily on the piano. All these things are apt to be set down to carelessness, and her parents and teachers are the more likely to inflict punish-

ments for them because she commonly exhibits a change in her disposition, becoming inattentive and forgetful, apathetic, peevish and ill humored, or extravagantly gay and excited in her manner.

On the other hand, it not infrequently happens that the disease reaches a far higher pitch of severity than has hitherto been described. The patient becomes unable to stand; even when she is recumbent her limbs are tossed about in all directions, so that boards well padded have to be fixed on each side of her bed to prevent her throwing herself on to the floor. In spite of all the precautions that can be taken she is very apt to bruise herself and to rub off the skin from her elbows and knees and other bony projections, which accordingly become covered with crusts and sores. She ceases altogether to sleep, and the violent movements go on day and night without intermission. In such cases emaciation takes place with wonderful rapidity and to a remarkable extent. Thus, Dr. Tuckwell has related the case of a boy whose flesh was wasted in the highest degree at the time of his death, but who six days previously had borne the appearance of vigorous health, with remarkably well-developed muscles; and, conversely, von Ziemssen mentions one in which a girl, eleven years old, gained during convalescence 5½ lbs. in ten days, her weight rising from 55½ to 61 lbs. in that time. One cause of the extreme wasting which accompanies severe chorea is doubtless the difficulty with which food is administered. The patient often seizes a spoon, or the spout of a feeding bottle, as if she would bite it in two, and so as actually to break her own teeth, or perhaps she wounds the fingers of her attendants. Indeed, such injuries are not always inflicted accidentally, for the mental state of a patient affected with severe chorea is often one of mania. Thus she may be violently delirious, shouting, singing, and talking incoherently. Or she may unexpectedly perform some strange action; for instance, a girl who was in hospital some years ago, suddenly got out of her own bed, and turned a somersault across that of another patient. There is no necessary relation between the intensity of the choreic movements and that of the psychical disturbance. Dr. Wilks says that the mind remained perfectly clear in one of his patients, who had the worst attack of chorea which he ever saw terminate favorably. On the other hand there occurred some years ago in Guy's Hospital a fatal case in which there were such marked mania and so complete a loss of consciousness, while at the same time the movements were so jactitating and like those seen in epilepsy, that the diagnosis remained doubtful until the autopsy was made, when recent vegetations were found upon the mitral valve. Dr. Hills, of the Norfolk County Asylum, recently stated that more than one patient suffering from chorea had been sent to that institution as insane, the real nature of the disease having been overlooked.

The *course* of chorea may be said to be generally chronic, but its duration is very variable. I believe that almost all the statistics which have been published concur in stating a period of from two to three months as the average. Thus Wicke found it eighty-nine days in a series of 125 cases, and Sée sixty-nine days in one of 117 cases; while from much smaller data, Gray, Tuckwell, and Hillier in this country each made it about ten weeks. Individual cases, however, range widely on either side of these limits; and, as might be expected, far more widely *above* the average duration than *below* it. A necessary consequence is that the introduction of one or two very prolonged cases may greatly disturb the statistical result; even in this series of Drs. Gray and Tuckwell ("*Lancet*," 1871), in which the longest case did not extend beyond twenty weeks, there were only six cases above the average to ten below. But we shall see, hereafter, that patients sometimes apply for treatment in whom the disease has already lasted for many months or even for some years. A single instance of this kind would inevitably

spoil, for the purposes of comparison, any series which did not embrace a very large number of cases. A point of considerable importance is that, as a rule, chorea is more likely to last for a great length of time when its symptoms are comparatively mild; patients who are obliged to have boards placed along the sides of their beds often get well before those who are able to be up and about the ward throughout their stay in the hospital.

Fatal Event.—Children, who are the chief sufferers from chorea, very rarely die of it. At Guy's Hospital I believe that between the years 1848 and 1875 there had been in all twenty cases of the disease which terminated fatally. But five of these have to be left out of consideration, death having arisen either from mere accidental complication (such as dysentery or diphtheria) or from concurrent rheumatic pericarditis, cardiac dropsy, or the like. Now, of the remainder there are only four in which the patients were below the age of fourteen (two were seven years old, one twelve, one thirteen). The remaining eleven patients were more than fourteen years old (nine of them between the ages of fifteen and eighteen, one forty years old and one fifty years old). Few of the girls or women among them seem to have been pregnant; but it is an ascertained fact that the mortality is very great where *pregnancy* is present, abortion, or premature delivery being also of frequent occurrence. It seldom happens, even in the most severe forms of chorea, that the patient dies within three or four weeks from the commencement of the disease. The most rapid case that I have read of is the one referred to at p. 651, in which there was at first a doubt whether the neurosis was not mania rather than chorea; the whole duration of the patient's illness was said to be only two or three days. In 1853, a girl, aged sixteen, who was already in the hospital for syphilis, was attacked with maniacal chorea, and died in six days, and about two months previously another case, a boy of the same age, had terminated fatally in nine days. The immediate cause of death seems to be almost always the intensity of the nervous symptoms themselves, but the movements often subside, and may even cease entirely during the last few hours, the patient lying comatose and passing urine and fæces involuntarily. The temperature of the body may rise during this period of the disease. I made an autopsy in one case in which it was 104.7° before the patient died, and Dr. Frederick Taylor had in our clinical ward a case in which the thermometer registered a temperature of 108° immediately after life was extinct. It does not appear that endocarditis is ever concerned in bringing about the fatal issue, or, indeed, that its development affects in any way the progress of the disease. The presence of bronchitis has been noted in several of the reports of autopsies at Guy's; in one instance it, perhaps, accelerated the patient's death. It has been supposed to have been caused by exposure to cold, owing to the bed clothes having been thrown off in the violent movements. In 1873, Dr. Habershon had a fatal case in a boy, aged twelve, who had been three weeks in the hospital when his breathing became obstructed by swelling of the tongue. Tracheotomy was performed, but without saving the patient's life. Probably the glossitis was the result of injuries inflicted by the teeth, for in another instance, in which the tongue had been severely bitten in two places, there was discovered after death a foul ulcer which exposed the sublingual gland and extended for the depth of an inch into the muscular and other tissues.

According to Dr. Radcliffe and von Ziemssen meningitis occurs in some cases of chorea, and in others myelitis; and they accordingly mention those affections as sometimes bringing chorea to a fatal termination; but I must confess that I should feel very skeptical about the accuracy of the diagnosis of any case in which well-marked inflammatory changes were not discovered either in the membranes of the brain or in the spinal cord.

Recovery.—This is generally gradual. Sometimes, however, the breaking out of an exanthem, or of some other febrile disease, is followed by the sudden subsidence of the movements. This is in accordance with a maxim of Hippocrates: "Spasmos febris accedens solvit." Dr. Radcliffe states that he has met with seven cases in which it occurred; and some striking examples of a similar kind are recorded by Rilliet and Barthez. According to the observations of Sée, however, the neurosis is not likely to be cut short unless it has reached its acme of development before the commencement of the fever. In many instances the first effect of the latter is to aggravate the movements, which yet may in those very cases cease a short time afterward, when the crisis of the acute disease has passed.

It sometimes happens that the subsidence of the spasms in severe cases of chorea is not followed by any evident amelioration in the patient's general condition. She may become perfectly quiet, and yet may remain for two or three weeks unable to stand, and possessing very little power in her arms. Or there may be a paralysis limited to a single limb, or to the two limbs on one side, the "choreic hemiplegia" of Dr. Todd. In association with such symptoms, or independently of them, the intelligence may seem to be very defective; or a condition of mania or melancholia may develop itself. Dr. Barnes met with a case in a pregnant woman, who as she regained her strength became insane and had to be removed to Bethlem. Other patients, when the choreic movements pass off, remain absolutely silent for days together, making no attempt to reply to the anxious solicitations of relatives and friends. The observations of Dr. Hughlings Jackson have associated such defects of speech with a preponderance of spasm in the right limbs, or limitation of it to them.

Alarming as these various symptoms are, I believe that they almost always pass off in their turn, and that the patient ultimately regains a state of perfect health, at least so far as the muscular and nervous systems are concerned. Trousseau, indeed, speaks of children who have never again shown the same degree of intelligence as before, and von Ziemssen says that slight defects in the co-ordination of the movements, a precipitancy in the performance of certain manual actions, or a tendency to facial grimaces, may persist for years or even be permanent. Dr. Radcliffe, on the other hand, is inclined to think that the disease is apt to be followed by other neuroses, particularly epilepsy, at a later period of life. The strong tendency of chorea itself to relapse again and again will be mentioned further on. Dr. Bristowe mentions a case in which there was a degree of rigid flexion of the hip and knee joints, with overlapping of the knees from preponderant action of the adductors of the thighs, and with a tendency to talipes equino-varus—conditions which seemed to him to indicate that degenerative changes had supervened in the lateral columns of the spinal cord.

The *pathology* and the *ætiology* of chorea are so intimately connected together that it is impossible for me to discuss the one apart from the other. They involve a series of problems which are of great interest, and which have important bearings upon the theory of the causation of the neuroses in general.

In the first place, there are still differences of opinion as to whether the disease has its seat in the spinal cord or in the encephalon. When the functions of nervous centres in the cord were first discovered, and when it was found that frogs and other animals could perform co-ordinated movements after excision of the cerebrum, it was natural that attempts should be made to refer to disorder of those centres all affections of which the main symptoms are irregular motions of the body and limbs. Thus Romberg placed chorea, without limitation, among the "spinal spasms;" and so recently as 1873 Jaccoud has defended a similar doctrine in an elaborate argument.

In England, however, the opinion has for some years prevailed that the disease is seated in the sensori-motor ganglia at the base of the brain, and especially in the corpora striata.

In favor of the spinal theory of chorea, certain vivisections have been adduced. It appears that dogs are liable to a similar complaint. Now, Chauveau, of Lyons, conceived the idea of cutting through the cord close to the skull in such animals, and he found that after this had been done the movements went on in exactly the same way as before, the spasmodic contractions of the diaphragm being in one instance sufficient to keep the dog alive for three hours. In two other experiments, that muscle happened to be paralyzed, so that artificial respiration had to be maintained; but the result was the same as in the first observation, as far as concerned the choreiform spasms. And one further point was made out, namely, that a second division of the cord, at the bottom of the dorsal region, put an end to the motions in the tail and in the other parts beyond the section. Chauveau's experiments have been repeated by Legros and Onimus; and they have ascertained, in addition, that by irritation of the posterior columns of the divided cord the spasms were increased, while they were arrested by complete section of the same structures.

It may, indeed, be a question whether the chorea of dogs is identical with the disease to which man is liable, and, again, whether in the human subject the higher nervous centres do not assume functions which in brutes are performed by lower ones. At any rate, I think that English pathologists have been able to make out a strong case in favor of their own opinions. Some of the points were stated by Dr. Russell Reynolds as far back as 1855. As he remarked, the spasms produced by persistent irritation of the cord are tonic rather than clonic; and it is further inconsistent with a spinal origin that the choreic movements should be in any degree capable of being controlled by the will, that they should be increased by emotions or by voluntary efforts, that they should cease during sleep, and that they should be diminished by direction of the patient's attention to other objects. In addition, Dr. Broadbent ("*Brit. Med. Journ.*," 1869) has insisted on the fact (which Romberg had previously observed) that tickling the palm of the hand or the sole of the foot of a child affected with chorea leads to no increased spasms; on the contrary, it is borne without difficulty, and the tendency to the excitation of reflex actions sometimes seems to be less than in health. Another point on which he lays still more stress is that the spasms are so often unilateral. An affection of one-half of the spinal cord throughout its whole length, from the crus cerebri downward, without implication of the other half, is, he declares, scarcely conceivable. And the improbability of such a localization of the disease is indefinitely increased by the fact that the muscles of the face and trunk are bilaterally affected, these very muscles being liable to be set in action on both sides by stimuli derived from a single corpus striatum, whereas there is no reason to suppose that disturbance of one-half of the cord could affect them in a similar manner. Lastly, the fact that the mental faculties are so frequently impaired in severe cases of chorea would naturally incline one to localize the disease in the closest possible proximity to the hemispheres, since it must be due to an extension of the morbid process to the highest cephalic centres.

Dr. Dickinson made ("*Med.-Chir. Trans.*," 1876) a series of anatomical investigations in fatal cases of chorea, from which it would appear that certain morbid changes, consisting of congestion of the vessels, of periarterial degenerations, and of minute spots of scleroses, are discoverable in the upper regions of the spinal cord, as well as in the lower parts of the brain. These observations of Dr. Dickinson's have not yet been con-

firmed by other pathologists; but even if the constant occurrence of the appearances which he has described should be fully established, it would still be a question whether they are not merely secondary effects of the disease. Whatever view one may hold as to the starting point of the choreic spasms, one cannot doubt that all the nervous structures which lie between it and the muscles must have their functional power strained to the utmost during the continuance of such violent movements; and therefore we ought to expect that they should all exhibit any degenerative changes which may after a time develop themselves. It is important to note that Dr. Dickinson's cases had all reached an advanced stage. Moreover, it is clear that the persistence of the morbid changes which he describes is compatible with the subsidence of the spasmodic movements and the restoration of health. For in one patient who had twice before had chorea (the last time having been a year previously), the duration of the fatal attack was only thirteen days. And yet changes of old date—peri-arterial degeneration and scattered spots of sclerosis—were found, besides recent congestion of the spinal cord and of the sensori-motor ganglia.

On the whole, then, it seems to me most probable that the corpora striata are the essential seat of chorea; one of these ganglia being affected when the disease is unilateral; both of them when all the four limbs take part in the movements.

The next question is as to the nature of the change in these. Now, Dr. Broadbent maintains that the spasmodic movements which characterize the disease are not significant of any one anatomical condition, but indicate that the functions of a particular part of the brain are deranged. To adopt his language, chorea is "a *symptom* not a *disease*. It has been called an *insanity of the muscles;* it would be better designated a *delirium of the sensori-motor ganglia*, since it bears the same relation to those parts that the delirium which may occur in a variety of maladies bears to the cerebral hemispheres."

Now, it possibly may be true that choreiform spasms may accompany different morbid states of the corpora striata. But it is no less true, and I think it far more significant, that the disease which I have been describing as chorea—instead of being an accidental complication of other maladies—occurs chiefly in a particular class of patients, and under circumstances peculiar to itself. And I must maintain, in opposition to Dr. Broadbent, that there is not a member of the whole nosology which better deserves to be called "a disease," according to the principles laid down at p. 19. Thus it is greatly more common in females than in males; and (like hysteria) it shows its predilection for the former sex even in the case of children before the age of puberty, in whom, however (being so far unlike hysteria), it is much more apt to occur than in adults. Tabular statements in regard to these points have been published by Dr. Pye-Smith ("*Guy's Hosp. Reports*," 1873). He found that among a number of patients at Guy's Hospital forty-two were males and one hundred and six females. The ages at which first attacks began were in five cases between two and five years, in sixty-two cases between six and ten years, in forty-four cases between ten and fifteen years, in nineteen cases between sixteen and twenty years, in five cases between twenty-one and twenty-six years, and in only one case thirty-eight years. Several instances have, indeed, been recorded, of the occurrence of chorea at a still more advanced age. Dr. Graves mentions the case of an apothecary in Dublin who was attacked when seventy years old; and Trousseau relates in detail an instance which came under the observation of Dr. Henri Roger, in a lady, aged eighty-three, who recovered from the disease in five weeks. On the other hand, examples are not altogether wanting of the presence of chorea in new-born infants. Thus Richter is quoted by

von Ziemssen as having recorded two cases in each of which a female infant was affected at birth, the mother having received a fright while advanced in pregnancy; the children suffered while awake, from clonic spasms, which were absent during sleep, and afterward ceased almost entirely. A somewhat similar instance is related by Dr. Long Fox as having occurred congenitally in an infant born six weeks before the proper time.

Still the fact remains, that in the great majority of cases chorea is a disease of childhood, between the ages of six and fifteen years, or between the period of the commencement of the second dentition and that of puberty. The preponderance of females among those who are attacked by it is doubtless due to the peculiar sensitiveness and mobility of their cerebral organization. It accords with this supposition that the children most liable to suffer are those who are delicate and excitable. As to the question whether this disease is especially apt to occur in those who inherit a tendency to the neuroses in general, there is some difference of opinion. Trousseau and Anstie answered this question affirmatively. Sée gave a contrary opinion.

Among persons more than seventeen years old the preponderance of females is infinitely greater than it is in children. In Dr. Pye-Smith's series of cases there were ten women above the age of seventeen to only two men.

Another important point in the ætiology of chorea is its liability to recur again and again in patients who have once suffered from it. A large proportion of the patients admitted into any hospital have had the disease once or oftener before. Dr. Pye-Smith records the case of one young man who had an attack every autumn from the age of fourteen to that of twenty-two, and of a girl who was seized each May, from her eighth year to her fourteenth. The duration of relapses is generally less than that of the first illness, but Trousseau mentions some exceptions to this rule.

The immediate exciting cause of chorea is often a severe nervous shock, such as a fright. Many striking instances of this have been placed on record. Thus Romberg relates the case of a girl, aged ten, who was one morning violently alarmed by a dog which jumped at her and barked, and who was seized with chorea the same evening. Again, von Ziemssen speaks of a boy, aged ten, who was terrified by a shot falling unexpectedly close to him on a field, and in whom the disease reached a great pitch of severity within a few hours. Trousseau gave the case of a girl, aged sixteen, who had been caught hold of by a man as she was going down stairs one evening without a light, and who was so frightened that she had a nervous fit, and from that moment became affected with St. Vitus's dance. Bright described the case of a boy who had already recovered from an attack of chorea, and who was sleeping with his father when the latter was seized with a fit of apoplexy; the boy was so alarmed that his disease returned. And, to give an instance that has recently occurred at Guy's Hospital, I find notes of a child admitted under Dr. Pavy with chorea, who was frightened by seeing her brother in flames, ran out of the house screaming, jumping over three walls, took refuge in a neighbor's house, and was brought home and put to bed, her mother at the time noticing a peculiar twitching movement about her face and irregular motions of her limbs; next morning she was unable to stand, and the disease quickly developed itself. There is no doubt whatever that much exaggeration has prevailed in regard to the association of chorea with mental impressions, and that parents and relations often jump too hastily at conclusions in regard to it, particularly when the fright occurred some time before the disease begins to appear. But the cases just quoted seem to be beyond cavil or dispute, and I think that they afford good grounds for supposing that the same

cause has really been in operation in other instances where a longer interval has elapsed. Again, Dr. Hughes ("*Guy's Hosp. Rep.*," 1855) related a fatal case in which the symptoms were slight when the patient was admitted, but became suddenly aggravated in consequence of a fright caused by another woman in the same ward. In other instances chorea is apparently the result of *imitation*.

It has long been taught at Guy's Hospital, both by Dr. Addison and by the physicians who have succeeded him, that not more than one or two patients affected with this disease should be placed in the same ward with other children, lest they should in their turn be attacked by it. I cannot, indeed, learn that any of the sisters or nurses remember an instance in which this has occurred, and I believe that at the Children's Hospital such precautions are believed to be altogether unnecessary. But some examples of the occurrence of chorea in an almost epidemic form have been placed on record, of which the most striking appears to be that related by Bricheteau. At the Necker Hospital, one afternoon, a young girl was admitted, suffering severely from the disease; in the evening a patient already in the ward, who had previously had chorea and was suffering from hysteria, began to exhibit movements, and in twelve hours had to be tied down; next day two other cases occurred, and within the four following days five more, making eight in all; the disease might probably have spread still further had not the patients been from that time isolated.

Another condition which plays an important part in the causation of chorea in girls after the age of puberty and in young women is *pregnancy*. Dr. Barnes has collected ("*Obstet. Trans.*," vol. x) a series of fifty-eight cases of this kind, and Wurzel has added eight others, making a total of sixty-six. The period of gestation at which the spasmodic movements are most apt to begin is from the first to the third month, but sometimes it is much later; and two instances have been recorded in which they followed parturition. First pregnancies are much more often accompanied by chorea than subsequent ones. This, of itself, suggests that one element in the production of the disease may be the emotional excitement which necessarily arises in a woman who finds herself for the first time *enceinte*, and the suspicion is confirmed by Dr. Wilks's observation that a large proportion of those who are attacked are unmarried girls, to whom their condition is one of shame and disgrace. Thus a connecting link is established between the chorea of pregnancy and that which occurs under other circumstances, and a still stronger one is the fact that in a considerable number of cases the patient has already had the disease on one or more occasions at an earlier period of life. Among the sixty-six cases already referred to, there were fourteen in which previous attacks had occurred.

So far the causal relations of chorea differ little, if at all, from those of the neuroses in general; and it may be added that, according to Trousseau, chlorosis and anæmia are among the conditions which dispose to its development. But I have now to pass to an entirely different set of agencies which play a most important part in the ætiology of this, but of no other nervous, disease; I refer to *rheumatism* and its unknown causes. There is, indeed, still much difference of opinion with regard to the nature of the connection in question. We have it, on the authority of Dr. Bright, that as far back as 1802 rheumatism was stated to be one of the causes of chorea, in the Syllabus of Lectures on Medicine delivered at Guy's Hospital. That great physician was himself convinced that an intervening link between the two disorders was to be found in the presence of inflammation of the pericardium, from which he imagined that irritation was transmitted to the nervous centres, just as in other cases it might be communicated from the intestines or the uterus. And in support of such a view there certainly is a case recorded

by Dr. Wilks, in which pericarditis caused by renal disease was accompanied by choreic movements. That case, however, is at present unique; and inflammation of the pericardium is, after all, present in very few cases of chorea. But, on the other hand, it is certain that the association between the neurosis and rheumatism is far wider and closer than would appear from Bright's theory. Beside acute rheumatism, chorea is the chief and almost the only cause of a simple inflammation of the valves of the heart, and this lesion probably occurs in almost every case of chorea. At least, out of eighteen fatal cases, which have occurred in Guy's Hospital between 1848 and 1876, and in which autopsies were made, in only one was endocarditis altogether absent; and that the frequency of its occurrence was not dependent merely upon the severity of the chorea in those particular instances is evident from the fact that in five of them the patient's death was accidental and due to some complication or intercurrent disease. The circumstance that the neurosis and acute rheumatism are almost alone in giving rise to such an affection of the cardiac valves would of itself suggest a closer relation between the two; and this is strongly confirmed by the frequency with which they occur in the same individual; a child who has had St. Vitus's dance falls ill with rheumatic fever a few years afterward, or *vice versâ;* or, again, slight choreic movements appear in the midst of a rheumatic attack, or some rheumatic affection of one or more joints in the course of chorea.

Of late years attempts have been made to explain the connection between the two diseases upon another theory, which, although it commands the support of some distinguished observers, I cannot believe to be well founded. Its author was the late Dr. Kirkes. Starting from the frequent association of endocarditis with chorea, he suggested that the cause of the spasmodic movements might be the introduction of inflammatory products and fibrinous particles from the diseased valves into the blood, and the consequent disturbance of the functions of the parts in which it circulated. More recently, Dr. Hughlings Jackson has expressed a similar view in a more precise and definite form, maintaining that the cause of chorea is *embolism* of minute arteries in the region of one or both of the corpora striata.

A few observations have since been made by different pathologists which have been thought to corroborate Dr. Jackson's theory. In a very severe case, attended with maniacal symptoms, Dr. Tuckwell ("*Med.-Chir. Rev.*," 1867) found at the under and outer aspect of the right hemisphere a large red patch of softening, affecting to some extent the white as well as the gray matter; and a branch of artery which ran straight into it contained a small, white, tough, fibrinous concretion, tightly wedged into an angle of bifurcation, and connected on all sides with long, black coagula, extending into the trunk of the vessel and its branches. On the outer aspect of the same hemisphere was a circular patch, but smaller, and limited to the superficial cineritious substance of the convolutions; in this nothing like an embolus was discovered, but the parts had been cut through in various directions before the dissection of the vessels was commenced. In another case of very acute chorea, which terminated fatally by hemorrhage into the brain, Dr. E. L. Fox ("*Med. Times and Gaz.*," 1870) believed that he detected microscopic emboli in the vessels of the corpus striatum.

I cannot say that these observations strike me as very convincing, even from a merely anatomical point of view. It must surely be very difficult to determine whether clots in minute cerebral arteries are of ante-mortem formation, still more whether they entered the vessels from below, instead of being formed *in situ*. Moreover, the minute, firmly adherent vegetations which are found on the valves in chorea, do not seem at all likely to be detached and carried away by the blood stream. Indeed, if they were so

liable to be washed off. I do not see what could prevent some of them from being carried into the spleen and kidneys, and producing infarctions there; but such appearances have never been discovered in any one of the fatal cases of chorea at Guy's Hospital. Again, as Dr. Bristowe remarks, it is difficult to understand the limitation of chorea to the limbs of one side on the embolic theory, since a shower of minute emboli must be supposed to enter the arterioles of one corpus striatum, leaving those of the opposite side entirely free. On the other hand, we are familiar with a form of endocarditis in which vegetations are frequently carried away from the diseased valves, giving rise to embolism in the brain and in other parts; and in this disease I believe I may say that chorea never develops itself.

But the strongest argument of all—and one that seems to me to settle the question—is the relation of chorea to *mental shocks*. Some writers have supposed that the cases in which the disease follows a fright are distinct from those in which it is associated with endocarditis; but this is certainly not the fact. On the contrary, as I have already stated, vegetations are found on the cardiac valves in all fatal cases, almost without exception; and I may quote as a special instance, that of a child under Dr. Wilks's care who was attacked by chorea after being terrified by the gunpowder explosion at Erith, and in whom the mitral valve was found inflamed. Now, it is obvious that the disease cannot possibly have two different exciting causes in a single case. It cannot be at one and the same time the result of a mental shock and of embolism of several minute cerebral arteries. In such cases we cannot but admit that the endocarditis is an effect of the chorea, and when once we have made this admission it is difficult for us to dispute the justice of the conclusion that the same thing is true of all other cases likewise.

Still, it is not to be denied that this view of chorea presents some remarkable features. It is very strange that a disease so closely related to acute rheumatism should be called into exertion by a nervous shock; it is still more strange that, when so produced, it should possess as great a power as rheumatism itself of setting up inflammation of the cardiac valves—an affection which we are apt to regard as a proof that the blood contains some noxious ingredient. No more striking instance could be found of the interdependence and combined working of different predisposing and exciting causes, which is so constantly met with throughout the ætiology of the neuroses in general.

Treatment.—This is a very difficult question; for chorea offers peculiar obstacles to the satisfactory investigation of the action of remedies. Many of the severer cases tend naturally to a more than usually rapid recovery; and in other instances (as Dr. Wilks has proved) the being admitted into the ward of a hospital, and kept in bed, is of itself sufficient to bring the complaint quickly to a termination. Under either alternative, the medicine which may have been prescribed is apt to get undue credit. Lastly, in the great majority of cases, chorea subsides at the end of from eight to twelve weeks, under whatever treatment. Now, the method adopted by most medical men is to give one drug for three or four weeks; if that fails, to change it for another, which they continue for about an equal period of time; and, if there is still no result, to begin the administration of a third. The result necessarily is that even if all these medicines are all really equally inert they gain very different degrees of credit. The uselessness of the one which is first used is sure to be apparent; but the second runs a chance of being supposed to be successful; while the last one (if a third is needed) is almost certain to acquire the reputation of having cured a case in which its competitors had altogether failed. Yet that very drug, if placed first on the list for a succeeding case, of course, shows itself as

impotent as they were. Probably, a very large part of the prevailing uncertainty as to the influence of remedies upon chorea is explicable in this way.

It would seem that there are only two methods of avoiding these difficulties. One would be to treat a considerable number of cases with some one medicine throughout the whole course of the disease, and then to compare them with the series of cases related by Drs. Gray and Tuckwell, which were allowed to terminate without interference. Accordingly these observers administered arsenic to fifteen patients; and they state that the average duration of these cases was almost absolutely the same as if no medicine had been given. And the very volume of the "*Lancet*" in which their investigations are recorded contains reports of twelve other cases by Mr. Butlin, treated by Drs. West and Dickinson with sulphate of zinc. But it is impossible to compare their results with those of the Oxford physicians. Two of the patients had had the disease for so great length of time before they came under observation (thirteen months and four years respectively) that their introduction would necessarily swamp the whole series. And after all they only present in an extreme form difficulties which belong in a less degree to all the other cases, every one of which had lasted for several days, and many of them for some weeks, before the administration of the medicine was commenced. It is not easy to see how one could avoid this source of fallacy, except by confining one's observations to the children in one particular school or public institution, where a uniform treatment could be employed from the very beginning of the disease. So that it is almost impossible to obtain statistical proof of the value of medicines in the treatment of chorea.

But there is very strong evidence, of another kind, in favor of at least one remedy. Very protracted cases, which had resisted all other methods of treatment, have sometimes been found to yield in a very short space of time to arsenic. Some striking examples of this are recorded by Romberg.

One is that of a girl, aged eleven, who had for eight years suffered from intense chorea, affecting especially the right half of her body. All drugs had been found useless until she began to take Fowler's solution; in about two months there was a marked improvement, and at the end of two months later still she had entirely recovered. Another patient, a girl, aged ten, had had the disease two years; arsenic was prescribed, and within three weeks the symptoms presented a marked abatement; and ten or eleven weeks afterward she was discharged cured. A third instance is that of a girl, eight years old, who for six months had been the victim of chorea to such an extent that she could not walk, nor stand, nor speak articulately. The remedies which had been tried had failed; Fowler's solution, in doses of four drops three times a day, established a cure in eight weeks. So far as I can see, the only objection that can be offered to these cases is that it is not distinctly stated whether or not the patients, while the medicine was being given, remained under absolutely the same conditions as before, in all other respects.

But if arsenic be capable of curing certain cases of chorea, there is surely a presumption that it may hasten the recovery of the patient in other cases, the circumstances of which are such as to prevent one from drawing any positive conclusion from the results of its administration. And most observers are of opinion that others of the so-called nervine tonics are also useful. Thus, the sulphate of zinc has for many years been largely used at Guy's Hospital; one-grain doses of it used to be given at first, which were gradually increased until the patient took a scruple or more. It seems to me advisable to begin with a larger dose, as, for instance, with five grains. I

seldom find that this causes nausea or sickness more than once or twice. Another drug which has had a great reputation is the carbonate of iron.

In the milder cases of chorea, again, there is reason to believe that the perversion of the voluntary movements may to some extent be checked by gymnastic exercises, by military drill, or by the use of a skipping rope. The French physicians have laid stress upon this method of treatment, and I have seen instances in which it has proved successful.

On the other hand, one sometimes has to deal with cases which are so severe that the administration of nervine tonics is obviously inapplicable, because the patient is in imminent danger of dying within two or three days, whereas these remedies require time for their operation. The alternatives, then, are either to abstain altogether from medicinal treatment, or to give drugs of which the action is more rapid. It must be admitted that there is the greatest difficulty in determining the value of remedies in cases of this kind. Probably death is sometimes inevitable. But even then the inhalation of chloroform gives great relief to the patient's sufferings. And if there be a chance of recovery it may do something toward economizing his strength, and it also saves him from the eschars which would form over the bony prominences if the movements were uncontrolled. In cases which are a little less severe, chloral appears to be the best medicine. Several writers have related cases in which it seemed to be effectual; not the least striking is, perhaps, one of Dr. Gairdner's, of a girl who took a drachm of it by mistake, and was poisoned, but who on her recovery was found to be cured. Again, the depressants of muscular activity—conium and the Calabar bean—have sometimes been employed; at one time I prescribed the succus conii in considerable doses for several cases. Some of the patients who took the drug recovered more quickly than I had expected, but I do not know that there is really any evidence that it possesses the power of controlling the disease.

A point of great importance in very severe chorea is that the patient should be kept well supplied with food; nutrient enemata ought to be administered frequently; and probably it is right to give full doses of alcohol.

Tetany.—In the paroxysmal affections hitherto described convulsive movements, if present at all, have generally been of a clonic kind. But there is one member of the group in which tonic spasm is the essential symptom. This is the disease to which Lucien Corvisart, in 1852, gave the name of tetany; previously it had been described by Dance, in 1831, under the title of "intermittent tetanus;" and other observers had designated it "idiopathic contraction of the extremities" or "rheumatic contraction of the extremities." I believe that Trousseau's lectures, one of which is upon "tetanilla," or tetany, first drew attention to it in this country; the earliest case that I know to have been recognized clinically is one recorded by Dr. Moxon in a paper in the "*Guy's Hospital Reports*" for 1870.

As may be supposed from some of the other names that have been given to it, tetany is generally confined to the limbs, and affects chiefly the distal ends of them. Sometimes it is limited to the forearms and hands; much more rarely to the legs and feet. Commonly it involves all four extremities at the same time or alternately.

The patient first has a sensation of tingling in the parts which are to be affected, and then begins to find that their movements are no longer free. Soon the thumbs become forcibly adducted; the fingers of each hand are closely pressed together, and are half flexed on the thumbs; the palms are hollowed, by the approximation of their inner and outer surfaces. The nails may be driven into the skin so violently as to produce marks, or even (it is said) to give rise to sloughs. The wrists are half flexed, and the forearms

strongly pronated. In the lower limbs the toes are bent down and adducted; the soles of the feet are hollowed, and the backs arched; the heels are pulled up by spasm of the muscles of the calves.

The contracted muscles feel hard; their resistance may be overcome by the employment of some force, which generally gives pain, but sometimes relief, to the patient. Thus the fingers and thumb may be straightened; but as soon as they are released they again become flexed as before. Another way of arresting the spasms is by means of cold. Trousseau mentions that persons in whom the lower limbs are affected can often regain the free use of them, for the time, by merely standing with naked feet on a stone floor; and in the hands and forearms the same result can be brought about by immersing them in cold water.

During the paroxysms the affected parts have their movements much impaired. If, as is sometimes the case, the patient is a woman nursing a child, she cannot hold it in her arms. There is partial anæsthesia, the power of determining the size and hardness of objects being lost. In walking, the sensation experienced is the same as if the feet were treading upon a carpet. Pain is sometimes altogether wanting, but it is usually present in more or less severity, and is compared to that which accompanies ordinary "cramps" of the legs.

Another common symptom is that the backs of the hands and feet become slightly tumid, and their veins a little prominent.

In from five to fifteen minutes the spasm generally passes off, but sometimes it lasts without intermission for an hour, or two or three hours, or even longer still. As it subsides sensations of formication are again experienced. After a variable interval another attack commences, and this goes on for several days, and even for two or three months. Even when a long period had elapsed the disease may return. A child, whose case is recorded by Dr. Moxon, was first attacked by tetany when five months old, and had it on five distinct occasions, at intervals of from five to twelve months before it reached the age of three years and a half. One of Trousseau's patients, a young man of twenty-one, had the disease every winter for four years, the contractions coming on every day during a period of two months.

A curious circumstance, which Trousseau has pointed out, is that in a patient affected with tetany one can at any time bring on the spasms by compressing the principal veins and blood vessels in the upper part of one of the limbs.

The electrical relations of the motor nerves and muscles in this disease have been investigated (Ziemssen's "Cyclopædia," xii, p. 335) by Erb and by some other German observers, with the result that the excitability both to faradic and to galvanic currents has been found greatly increased in all parts, with the exception only of those to which the facial nerve is distributed.

Cases presenting the characters that I have been describing are decidedly of rare occurrence; I am not aware that more than two or three have presented themselves at Guy's Hospital since the one which came under the care of Dr. Moxon in 1870. But it appears to me that a very common affection is really a minor variety of tetany. I refer to the "carpo-pedal contractions," or "turning in" of the thumbs and great toes, which one looks for almost as a matter of course in rachitic children, whose nervous centres are in a state of irritation. Dr. Hughlings Jackson, indeed, regards these as rudimentary forms of ordinary epileptiform convulsions; but surely they bear a much more obvious relation to tetany. And I cannot help thinking that careful observation in out-patient practice would bring to light intermediate conditions which have as yet escaped notice.

But it is by no means necessarily the case that in tetany the spasms are

limited to the extremities. Trousseau describes instances in which the face and trunk were also affected. In these cases the face became distorted ; the eyes acquired a squint ; the sterno-cleido-mastoidei and the pectorales were rigid ; the recti abdominis stood up like tense cords. The jaws were firmly clinched, and the speech even was impaired, in consequence of the tongue having become involved. Laryngeal spasm sometimes occurred, causing lividity and apparent danger of suffocation. Febrile disturbance was also present. Yet, even in such severe cases, the patient would often get up ; and if an adult, she would attend on other patients, although suffering from pains in the loins, and feeling bruised and exhausted. Sooner or later recovery almost invariably took place. Trousseau, indeed, mentions one instance in which death occurred from phthisis during a relapse of tetany, and another, which he supposed to have terminated fatally within a few hours from its commencement. Dr. Moxon has suggested that this was really a case of tetanus ; but the hands and feet were characteristically affected ; and these are the very parts which in that disease constantly escape the spasms.

If I am right in regarding tonic spasm of the fingers and toes as distinctive of tetany, I think that we must also include under that head another affection of early life, namely *Trismus neonatorum*. This occurs in infants within a week after birth, and sometimes even in the first twelve hours. From Dr. West's description of its symptoms it appears that, besides trismus and opisthotonos, there are powerful clinching of the hands, flexion of the feet upon the ankles, and bending of the toes ; and he goes on to say that "when the fit subsides the child still lies with its hands clinched, and its thumbs drawn into the palm, the legs being generally crossed, and the great toe separated widely from the others." The head is thrown back ; and the opisthotonos continues, although in a diminished degree. Before long the little patient becomes unable to swallow, and perhaps comatose ; and death quickly ensues.

It may be thought that the presence of opisthotonos disproves the view that I am disposed to take concerning the "trismus neonatorum." And it is true that Trousseau nowhere mentions by name that symptom as occurring in tetany. But he does speak of one patient as having all his muscles rigid, and being as stiff as a poker. And I may remark that in children tonic spasm of the muscles of the back of the neck commonly enough occurs, in association with the so-called "carpo-pedal contractions."

The relation of these various affections to tetany is, I think, confirmed by their ætiology. The trismus of newly-born children was at one time supposed to be a traumatic tetanus, excited by irritation which started from the umbilical cord. But that notion was refuted by the subsidence of the disease in the Dublin Lying-in Hospital, in consequence of the introduction of an effective system of ventilation. Previously, one in every six of the infants born there had died when less than a fortnight old ; and nineteen deaths out of twenty were due to trismus. Afterward the mortality was only one in fifty-eight and a half, and but a ninth part of it was from the disease in question. I believe that in the West Indian islands it is still common, and also in St. Kilda ; probably wherever it occurs it is due to a vitiated state of the air in the lying-in chamber. In London it must be of very rare occurrence, for Dr. West has only seen one case in this city.

I have already remarked that "carpo-pedal contractions" occur chiefly in children affected with *rickets ;* and the same morbid condition has been present in every case of tetany that I have seen. There could be no clearer indication that the hygienic influences have been bad. It is curious that Trousseau speaks of tetany as occurring most frequently in women between the ages of seventeen and thirty, particularly in those who are suckling their

children. One is tempted to think that there may be something in the *régime* adopted during the puerperal period by French physicians which lowers the health, and so brings on a disposition to the disease. Trousseau, however, speaks of it as being the direct result of exposure to cold. The woman whose case he records as having terminated fatally, had shortly before been delivered, and had suffered from obstinate diarrhœa. On several occasions she got out of bed in the night, and went to fetch water from a fountain in the yard of the hospital; indeed, she did this on the very night before she died. Trousseau described diarrhœa as a frequent predisposing cause of tetany; he had also seen it after enteric fever and after cholera. He had met with instances of it in women over forty years of age, and even in adult males.

The *diagnosis* of tetany is not difficult, spinal meningitis being the only affection with which it is likely to be confounded. Dr. Moxon has pointed out that the disease which has been described as spasmodic ergotism, and which was prevalent in certain parts of Germany in the earlier part of the present century, was very similar in its symptoms. I think that a perusal of Mr. Wright's account of it ("*Ed. Med. Journ.*," 1839) is sufficient to show that it really was identical with tetany, and that it was not caused by a specific poison, but was merely due to a general deterioration of the health from a defective supply of nutriment. He points out that in the eighteenth century a similar disease occurred in Sweden, which was caused by the admixture of the seeds of the Raphanus with the barley on which the people lived.

The *treatment* which Dr. Moxon adopted in his case of tetany consisted in the administration of bromide of potassium in five-grain doses; in four days the spasms ceased to return, and the child afterward took cod-liver oil and steel wine. I think there can be no question that such remedies are to be preferred to bleeding and cupping as suggested by Trousseau. Indeed, that writer himself makes an exception for weakly and debilitated subjects, for whom he recommends quinine with small doses of opium or belladona. In very severe cases he says that the inhalation of chloroform has sometimes given marked relief. He also mentions a case which Aran cured by applying to the affected parts pieces of linen soaked in chloroform, and kept in position by a few turns of a bandage.

TETANUS.—We now come to a disease the history of which dates back to Hippocrates, who knew it by its present name (τέτανος, from the Greek verb τείνω, I stretch). Indeed, so completely has the conception of *tonic* spasm become identified with this affection that physiologists commonly speak of a rigid state of the muscles as "tetanic," or even as a "tetanus," whatever may be its cause. Physicians, on the other hand, now recognize as entirely distinct several neuroses which are all attended with this symptom. Thus we have hysterical contraction of the limbs, certain reflex disorders (especially a form of trismus and tonic wry neck), and, lastly, the complaint just described under the allied name of tetany, and the scope of which I have ventured to enlarge by assigning to it a group of cases hitherto described under the title of *trismus neonatorum*. Evidently, therefore, something more than the mere presence of a particular kind of spasm is at the present time required to characterize tetanus. And the further elements of its definition are found in the seat of the contractions and in the certain more or less regular order in which they develop themselves.

Symptoms.—In the immense majority of cases tetanus begins as a stiffness or rigidity of certain muscles of the face or neck. Very commonly those first affected are the masseters and the other muscles of mastication; the

consequent inability to separate the teeth freely is known as *trismus*, and has given to the disease the common English name of "lockjaw." Sometimes, however, the earliest symptom is a "stiff neck," which the patient for a time regards as quite unimportant. Or, as in a little girl, who some years ago died in Guy's Hospital, it may be a peculiar grinning expression of the face; her mother found fault with her for it until she explained that she could not help it; soon afterward she became nearly choked in attempting to swallow some food. The disease is often first discovered when the patient wakes up in the morning. As a rule, it is preceded by a wound, or by an injury of some sort, which may or may not have been sufficiently severe to require surgical treatment. Sometimes, before the tetanus sets in, darting pains are complained of in the injured part, which may shoot up the limb; there are four instances of this among seventy-two cases which were collected from our books for the "*Guy's Hospital Reports*," in 1857, by the late Mr. Poland. Sometimes, again, it is said to be ushered in by rigors, but in general there are no *prodromata* whatever.

Now and then it happens that the trismus and other early symptoms, after lasting a few days subside; the disease may then be said to abort. Much more frequently the tonic spasm increases and spreads to the muscles of the trunk, and to at least the upper parts of the limbs. The patient's aspect is then very remarkable. The face may be described as having an unnaturally aged appearance, the forehead being wrinkled and the features contracted and drawn. The angles of the mouth are wide apart, and the lips are stretched over the closed teeth, so as to produce a fixed smile, which is known as *risus sardonicus*, and is at once seen not to be indicative of any pleasurable feeling. The alæ nasi are thrown outward, and the naso-labial furrows are exaggerated. The eyelids are half closed, but the eyes are said to have a staring expression, although their muscles are seldom, if ever, affected by the cramp. The jaws may be so firmly clinched that not even a paper knife can be wedged in between the teeth, or, perhaps, they can still be separated a little way from one another. The substance of the tongue seems not to be often involved in the spasm. The body is rigid, and it is almost always curved, so that the back forms a deep hollow. Thus the occiput is buried in the pillow, and the throat is stretched upward. If the patient were to attempt to lie straight, his frame would be found arched and supported upon the head and the heels. This condition is called *opisthotonos*. At the same time the chest is, of course, thrown forward, and it is more or less fixed in a state of expiration, while the abdomen is flat or sunken. The tension of the affected muscles is obvious to the touch and sight; this is particularly the case with the recti abdominis, which are often described as feeling "as hard as boards," and which, in persons who are not too fat, stand out in knotty masses through the integuments. Sometimes their fibres give way, and blood is extravasated so as to form a rounded swelling on one side of the linea alba. The limbs are commonly extended, and there may be a marked stiffness of the shoulders and hips, and even of the elbows and knees. But beyond a little undue resistance to passive flexion of the wrists, one does not generally notice any impairment of the movements of the hands or fingers. I have notes of but one case in which I find it recorded that the soles of the feet were arched and the extensor tendons on their dorsal surfaces rigid.

From an early period of the disease there is pain resembling that which is experienced in a limb affected with cramp, and one of the first symptoms is often an acute pain at the lower part of the sternum, piercing through to the back; this is supposed to be due to spasm of the diaphragm. There may also be a sense of oppression, of distress, from embarrassment of the breathing, and the same cause may render the voice feeble, or even reduce

it to a whisper. The patient is frequently unable to micturate, in consequence of the rigidity of his abdominal muscles.

According to Rose, of Zürich, many cases of tetanus end fatally without being attended by any symptoms beyond those which have been already described, but in a much larger number the spasms undergo aggravation from time to time, at longer or shorter intervals; during these paroxysms, which last from a few seconds to three or four minutes, the aspect of the patient may be frightful. The contraction of the features and the opisthotonos are greatly augmented. The tongue is often caught between the teeth and is severely bitten. The face and even the hands become livid from interference with the respiration. It is said that clonic contractions of the muscles may in some cases be observed. The paroxysms sometimes seem to arise spontaneously; sometimes they are obviously reflex, being brought on by a touch from another person, a draught of cold air, a sudden noise, or even some voluntary effort, such as the attempt to turn round, to speak, or to swallow. Bauer remarks that they cannot always be excited by the application of a stimulus for the purpose, particularly if the patient expects it, so that, for example, the prick of a needle may be ineffectual to elicit them. They are generally attended with a great increase in the pain, which may amount to the most extreme anguish. Sir Gilbert Blane, however, met with a case in which, although it terminated fatally, there was merely a sort of tingling sensation of rather a pleasurable kind.

Sleep is generally absent from an early period of tetanus, but a case related by Sir Thomas Watson shows that when the patient does fall asleep the tonic spasm ceases for the time, even the abdominal muscles becoming perfectly soft and yielding, but instantly resuming their contracted state as soon as he is awakened. The mind is perfectly clear and unclouded; only when the fatal termination is near at hand is there sometimes a little delirium.

The pulse is at first natural, but toward the last it becomes very rapid, perhaps 160 or 200 in the minute.

It was long a disputed question whether the disease is attended with fever. Observations made during the last few years have shown that the temperature may be normal throughout the whole course of even the most acute and severe cases. On the other hand, in those which are comparatively slow in their progress, the thermometer sometimes indicates 102° or 103° without there being any discoverable cause for it in an inflamed state of a wound or in any other complication. And, before death, hyperpyrexia sometimes rapidly develops itself, temperatures of 110° or 112° being registered. The suggestion has been made that the heat is then evolved by the muscular contractions themselves, especially since in tonic spasms no "external work" is performed. But in reality only a very small part of the rise could be accounted for in this way, and its great height must be ascribed to disturbance of a central regulating machinery, exactly as when the same thing occurs in a case of fractured spine, or of cerebral hemorrhage, or of the *status epilepticus*. A further point is that tetanus is among the few diseases in which the temperature has been observed to rise one or two degrees after death; the heat has then been supposed to be derived from the solidification of the muscles in *rigor mortis*. This explanation, however, does not seem to be quite satisfactory, for, although the post-mortem rigidity is said to begin early and also to last longer than usual, there appears to be an interval of relaxation from the vital spasm before it sets in.

In severe cases the skin is generally bathed in sweat; an eruption of sudamina is not infrequently present. Dr. Wilks has recorded in the "*Guy's Hospital Reports*" for 1872 an instance in which the perspiration from the forehead gave to white linen clothes a reddish stain, which looked as if it

were due to the presence of blood, but which was found by Dr. Stevenson to have different chemical properties.

Varieties.—But the symptoms of tetanus sometimes deviate from the ordinary type. Until recently, and from the most ancient times, several distinct forms of it have been described. Writers have said that the body may be arched forward instead of backward; the head and the knees meeting in front of the chest. Aretæus knew this by the name of *emprosthotonos*. And in another variety, for which the name *pleurothotonus* has been invented, the curve has been described as directed toward one side. But it has long been known that in comparison with opisthotonos they are exceedingly infrequent; and it is very inconvenient to make of them so many separate kinds of tetanus. Indeed, Rose has lately maintained that they are in reality never seen in this disease, although he admits that they may occur in hysteria. The French military surgeon, Larrey, has been the great authority for emprosthotonos; but Rose shows by detailed criticism that none of his cases belonged to any but a very mild form of tetanus, so that it certainly seems possible for mistakes to have been made as to the significance of a position of the body which is, after all, generally adopted by patients suffering under irritation of the nervous centres; and the more so since Larrey entertained the notion, which is certainly without foundation, that the distribution of the spasms varied according as the wound which caused the tetanus was in front or behind.

In 1870, however, a woman, aged forty, died in Guy's Hospital, of this disease, in whom it is said that "the anterior muscles were mainly affected, there being a condition of emprosthotonos."

There is one aberrant variety of tetanus, in which the earliest symptom of all is a spasmodic affection of the muscles of the part originally injured, and in which the paroxysms, when they set in, affect those muscles far more than any others. Such a case was recorded by the late Mr. Key in the 3d volume of the "*Guy's Hospital Reports.*" I once saw a well-marked instance of the same kind.

An occasional complication of tetanus, to which Rose has drawn attention, is facial paralysis. In 1871 a woman died in Guy's, under Mr. Poland, in whom this symptom was present on the left side, and whose ocular muscles were also affected in a strange way, the left eye being immovable, turned upward and outward, while the right one was as rigidly set straight forward. Neither meningitis nor any lesion of the brain itself was discovered at the autopsy. The original accident was a fracture of the orbital plate of the frontal bone, caused by the point of an umbrella. This corresponds with a statement of Rose's, that the starting point of the tetanus in such cases is always within the distribution of the facial nerve; he supposes that the trunk of the nerve becomes swollen, and is compressed within the bony canal through which it has to pass. If this view is correct, the symptom in question affords a proof of the occurrence of an "ascending neuritis" in tetanus, and is thus of very great theoretical interest. Indeed, in some few cases, nerve trunks are said to have been found reddened and swollen in parts of their course from the seat of injury up to the end; but as such changes are often not to be discovered, it has been considered doubtful whether much importance is to be attached to them.

Ætiology.—In reference to the *causes* of the disease I do not propose to enter upon various questions which are fully discussed in surgical works, as to how far the liability to it is influenced by the seat or the extent of an injury, by the healthy or unhealthy state of the lacerated structures, or by the introduction of foreign bodies. But it is important for the physician to be alive to the fact that it often follows very slight cuts and trifling abrasions, which are apt to be altogether forgotten by the patient and overlooked. Rose

says that nearly half of his cases were sent into the hospital as cases of rheumatism or of some internal disease. Instances have been recorded in which it has apparently been set up by the extraction of a tooth, by venesection, by the application of a blister or a cupping glass or a seton, by the sting of a bee, and by a cut from a whip. It has now and then been observed after simple fractures of the limbs, or after blows or falls upon the back of the neck, without any breach of surface. In 1860, a girl, aged five, died in Guy's Hospital, who on the day before she was attacked by the disease had had a fall, in which she was said to have slightly strained her back and grazed her elbow. Three other patients showed, one a slight scar over the knee, another a small sore on the elbow, and the third a little scab with pus beneath it at the elbow, in addition to a cicatrix half an inch long on the forearm, due to a cut received about six weeks previously. In 1873 a boy was admitted under my care in whom stiffness of the neck and jaws had come on four or five days after the healing up of a small "gathering" on one big toe, due to irritation from a small nail in his boot. The patient recovered. Again, tetanus has sometimes occurred soon after parturition or abortion. In one case which was observed at Guy's, in 1870, no cause for it could be discovered except a prolapsed and excoriated condition of the cervix uteri. Whether it is ever due to any lesion of an internal viscus, or to the presence of worms in the intestines, appears to be exceedingly doubtful.

It is remarkable that in only two of our cases, in which the disease has appeared to be referable to a trifling injury, has there been reason to suppose that cold played any part in its ætiology. One was in a patient of mine who, besides having a festering sore on the elbow, had got wet through in a shower of rain three days before the tetanus set in; this, perhaps, affected him the more because his work was behind the ovens at a biscuit manufactory. The other was in a man under Mr. Bryant, who had had a slight scratch on the little finger, which soon healed up; he also was exposed to wet two days before being attacked with stiffness between his shoulders. Nor do I know that any observations have ever been made at Guy's tending to show that tetanus is more apt to occur in those surgical patients who lie near an open window. Yet it seems to be an established fact that in some cases in which the affection is clearly traceable to a severe wound it is, nevertheless, also due, in part, to changes of temperature. Army surgeons have often noted that after a battle the wounded are especially apt to be attacked by it when they lie in tents on a damp surface, or when cold nights contrast with hot days. And it would seem that the liability to exposure to draughts in tropical climates is the best reason that can be assigned for the frequency with which, as is well known, the disease follows all sorts of injuries among the colored populations of the East and West Indies.

So, also, there appears to be no doubt that cold sometimes gives rise to tetanus directly in persons who have received no injury whatever. On July 17th, 1863, a man sat in a draught, and afterward felt a stiffness in his limbs; this continued during the 18th and the 19th; on the 20th more marked tetanic symptoms set in, and he came to Guy's Hospital and was admitted; at 7 A. M., on the 21st, he had a severe paroxysm affecting his chest, in which he died. Another patient, in 1869, had got his feet wet the day before the disease began. Sir Thomas Watson cites a case of Dr. Gregory's, that of a man who, "having fallen asleep in moist grass, awoke with a stiff neck, which afterward went on into regular tetanus."

But it now and then happens that not even this cause can with any plausibility be assigned to it, and that its occurrence remains altogether inexplicable in the present state of our knowledge. I find notes of three such cases which have ended fatally in our wards between 1863 and 1875.

Whenever the disease arises independently of an injury—whether it is traceable to cold or wet—it is commonly called "idiopathic," to distinguish it from the ordinary "traumatic" tetanus.

On the whole, tetanus is more common in men than in women; in young persons than in those who have passed the middle period of life; in the robust and the healthy than in those who are weakly or diseased.

Pathology.—Until within the last few years pathologists have been generally agreed as to the absence of any considerable changes in the nervous centres in tetanus. But in 1865 Mr. Lockhart Clarke recorded in the "*Med.-Chir. Transactions*," some cases in which he believed that he had discovered "areas of fluid, or of granular disintegration," both in the gray matter and in the white columns of the cord. Similar observations were afterward made by Dr. Dickinson, who, however, considered that the morbid material which he found in the same structures, and which closely surrounded the blood vessels, was an exudation. Still more recently Dr. Joseph Coats, of Glasgow, has demonstrated like appearances in the bulb and the pons. All these pathologists also lay stress on the congested state of the blood vessels, but that had long ago been noticed, and had been shown to be of no real significance, being attributable either to the mode of death or to cadaveric hypostasis. The question whether any greater importance is to be attached to the supposed lesions above referred to is one which has still to be further discussed, and in relation to several other diseases as well as tetanus. But whatever conclusion may be arrived at, there are obvious difficulties in bringing such morbid changes to bear in any way upon the pathology of the latter affection. The theory suggested by Mr. Clarke himself was rather complex. He supposed that the primary affection of the cord was a "hyperæmic and morbid state" of the blood vessels, due either to direct extension along the injured nerves (a *neuritis migrans*) or to reflex action excited upon the spinal vessels from the periphery. The hyperæmia, he thought, caused the lesions which he discovered, and the two together induced an abnormal excitability of the gray matter which, in its turn, originated the tetanic spasms under persistent irritation from the peripheral nerves. It is evident that on this view of the matter the lesions, even if they are present, form a comparatively unimportant link in the chain of causation. For my own part, I doubt whether we can, at the present time, form any clearer conception of the action of tetanus than of any other member of the group of spasmodic neuroses to which it seems to belong.

Diagnosis.—This is seldom difficult. It is only at the very commencement of a trismus from dental irritation that one could mistake it for a dangerous "lockjaw." Rose says that even at the earliest period of tetanus he has always been able to discover a certain degree of stiffness of the back of the neck, the patient being unable to bring the chin freely down to touch the chest. Another practical suggestion of his is that by introducing one's finger into the patient's buccal cavity on each side one can feel the hard edge of a rigid masseter much more distinctly than from outside the cheek. An inability to open the jaws, dependent upon a chronic affection of the temporo-maxillary joints, is in this way easily distinguished from all forms of trismus.

A fully-developed tetanus is sometimes simulated by hysteria. Sir Thomas Watson mentions an instance of this kind in a girl, who "would all at once be drawn into a position such that the top of her head and her feet alone supported her, while her body was bent backward like a bow; then, after a time, with equal suddenness, the opposite position was assumed, her forehead and her knees being brought together." His statement, I believe, suggests the criterion which is applicable to all cases of this kind; namely, the irregular and inconsistent nature of the symptoms.

Another affection which has perhaps to be considered is spinal meningitis. A case which is given in Reynolds' "System" as a typical example of the latter disease would show that they may resemble one another very closely. If we could accept the interpretation of it without question; but I am disposed to agree with Dr. Moxon that, after all, it may have really been an instance of tetanus.

When I describe hydrophobia, I shall have to mention that it is sometimes complicated with spasms like those of tetanus.

The most important point of all is that one should not set down to this disease the effects of the poisons strychnia and brucia, or of the substances from which those alkaloids are derived. A boy, aged twelve, was brought into Guy's under my care at 9 A.M. one morning, suffering from opisthotonos, and from spasms of the respiratory muscles, so severe, that he almost ceased to breathe and had a sense of immediately impending death. He was employed in a druggist's shop, and he confessed that between 7.40 A.M. and 8.30 he picked up some black stuff (afterward ascertained to be extract of nux vomica) and put it into his mouth for liquorice, until he found it bitter, when he spat out as much of it as he could. Ice applied to his spine gave him relief. From some urine passed at 1.20 P.M., Dr. Stevenson succeeded in obtaining the color reactions of strychnia and brucia. By that time his symptoms had passed off, and he was discharged from the hospital a few days later. The characteristic features of such cases are the suddenness with which opisthotonos and the most violent general spasms set in, the absence of persistent rigidity of the affected muscles during the intervals (so that the mouth can be freely opened), the fact that the hands are involved, and the rapid death or recovery which ensues. Even if the poison should be administered in small doses repeated at frequent intervals, there is no reason to suppose that the progressive development of an ordinary tetanus would be simulated.

Fatal Event.—As a rule, tetanus ends fatally between the third and the seventh days. The occurrence of death within twenty-four hours from the commencement of the disease is very infrequent. Among Mr. Poland's seventy-two cases there were only two instances of it; one patient, a young man who was attacked six days after admission with a compound fracture of the leg, died in nineteen hours; the other, a girl who had been burned, is said to have lived only four or five hours. The most quickly fatal case on record would be one of a negro servant who lacerated his thumb in breaking a china dish, and who was almost instantly seized with convulsions and died in a quarter of an hour, but Sir Thomas Watson, although he cites it, expresses a doubt as to whether this can be relied on as a genuine example of tetanus. In most instances death occurs during a paroxysm which surpasses all previous ones in severity. Its cause is generally supposed to be spasm of the diaphragm or of other respiratory muscles, or perhaps of those which close the glottis. Mr. Poland mentions that in one of his cases the heart's action continued for a short time after the breathing had ceased. In 1875 I had a patient under my care who on several occasions turned perfectly livid, so as to excite the strongest apprehensions that he was at the point of death, while a clinched state of his hands was the only outward indication of an increase of spasm. At last, after four days, one of these seizures did in fact prove fatal. Bauer, however, expresses the opinion that the danger from failure of the respiration in tetanus has been generally over estimated; carbonic-acid poisoning, he says, relaxes the muscles before the mischief is done, and there is no doubt that death sometimes begins from the heart. Rose relates an instance (which, however, was attended with hyperpyrexia) in which he had his finger on the pulse when it suddenly stopped forever. Some observers have sup-

posed that in cases of this kind the ventricles are seized with spasms. Stress has been laid on the fact that on post-mortem examination the heart is often found closely contracted, but it has been forgotten that this is in all probability merely the result of rigor mortis. Rose speaks strongly in favor of the view that the real cause of death is cardiac paralysis; he thinks that the muscular contractions oppose a resistance to the circulation through the arteries which the central organ is unable to overcome.

Lastly, there are cases in which life is prolonged for three or four weeks, but which yet ultimately prove fatal. The cause of death is then generally exhaustion. Sometimes the spasms have altogether subsided, so that the patient is thought to be in a fair way toward recovery, although he is excessively weak and emaciated and prostrate, with sunken features and a scarcely perceptible pulse. Larrey is said to have referred such cases to starvation, in consequence of the difficulty of getting food into the stomach during the acute stage of the disease. Rose thinks that the patient often really succumbs to a slight return of spasm which is so little marked as to escape notice. Among the cases collected by Mr. Poland there are two which proved fatal during a paroxysm as late as the twenty-first or the twenty-second day, and two others in which death was referred to exhaustion on the sixteenth and the thirty-second days respectively.

Convalescence.—When recovery from tetanus takes place, a certain degree of stiffness of the muscles often remains for a considerable time, and very slowly passes off. Mr. Poland mentions a case in which there was still some stiffness of the jaws after a year had elapsed. In 1871 Mr. Golding-Bird had a boy under his care in Guy's, in whom the spasms lasted for fifty-one days, and even after they had subsided his limbs still remained rigidly flexed, and he became exceedingly emaciated and had bed sores, so that, although he was floated on a bath of water, he ultimately died on the one hundred and seventh day.

Course.—Hitherto I have avoided describing separately an acute and a chronic form of tetanus, and, although almost all writers recognize this distinction, they generally admit that no actual line of demarcation can be drawn. It is true that when a series of cases are arranged in groups according to the length of time which happened to elapse between the injury and the onset of trismus or of stiffness of neck, a rapidly fatal termination is found to occur more frequently in those in which the trismus is early than in those in which it is late. A similar difference in intensity, although not so marked, seems to be observable in cases in which the primary lesion is extensive and severe, as compared with those in which it is slight. It is therefore not surprising that instances of "idiopathic" tetanus should, as a rule, be less urgent in their symptoms than those which are of "traumatic" origin, and since the subsidence of the disease, in those cases which recover, is never otherwise than gradual, one can understand how it has come to pass that the idea of chronic tetanus has become associated not only with a hopeful prognosis, but also with the absence of a severe injury or of any injury at all; or, again, with the occurrence of a long interval since the date of an injury, rendering the connection between them doubtful. But although this is tolerably correct, so far as groups of cases are concerned, it is very apt to mislead when applied to an individual patient. In some instances of idiopathic tetanus death ensues as rapidly as in most of those which follow quickly upon a severe compound fracture or a burn. So impressed is Rose with the vagueness of the current distinction between the acute and the chronic form of the disease, that he limits his "acute tetanus" to those cases in which there is nothing but a continuous tonic spasm. The supervention of paroxysms is described by him as belonging to a so-called "third stage" which the cases in question do not reach, although he himself points out

that the most rapidly fatal cases of all are seen in those in which paroxysms are present from the very first. Need I say anything further in order to prove that the division of tetanus into acute and chronic forms had better be given up altogether?

The only ground on which a *prognosis* of the disease can with any safety be based is that afforded by the rate at which the symptoms of the disease are developing themselves in the particular case under observation. And I think it should be added that rapid progress is much more surely indicative of a fatal ending, than a slow course of a good prospect of recovery, or even certainty that death may not be very near at hand. Spasm of the respiratory muscles sometimes destroys life suddenly and almost without warning. The average mortality of the disease among the seventy-two cases collected by Mr. Poland in 1857 was 86 per cent.

Some years ago Dr. Wilks and I saw with Dr. Anderson, of New Cross, a gentleman whose symptoms seemed to deviate from the ordinary features of tetanus, so that we none of us felt able to speak positively as to the probable issue of the case, nor even as to its diagnosis. It seems to me now that it might fairly have been regarded as one of those instances in which the paroxysms constitute the most marked element of the disease, but in which, although they recur but seldom, they destroy life early. The patient's main complaint was of a spasmodic pain in the right hypochondrium, which he attributed to wind. When it came on, he used to get up and walk about, declaring that he could not lie down; he would stand leaning against the bed post, and would call to his wife to rub his back. About a fortnight previously he had run a garden fork through his great toe; four days before I saw him he began to complain of stiffness of his jaws, and from that time he was unable to open his mouth fully. These facts, of course, led to a strong suspicion of the real nature of the abdominal pain. He died in three days from my first visit, and about twenty-four hours previously Dr. Wilks found him sitting up in bed and making his will. However, he then had frequent spasmodic catchings of the respiratory muscles.

Treatment.—The difficulty of prognosis in this disease necessarily brings with it a corresponding difficulty in estimating the results of treatment. There are, indeed, certain measures, the propriety of which is obvious. The patient should be placed in a quiet, dark room, and should be withdrawn from all noise and bustle, and from the visits of too many sympathizing friends. He should speak as little as possible. The food which is given to him should be nutritious but in a fluid form. He should probably have wine or brandy in rather full quantities. If he is unable to swallow, enemata of beef tea and brandy may be administered at regular intervals, unless even this brings on paroxysms of spasm, as is too often the case. Rose throws out the suggestion, which appears to me very valuable, that anæsthesia should be induced regularly once or twice a day by chloroform, for the purpose of enabling food to be injected into the stomach through a tube. Purgatives should not be given, except at the commencement, nor unless there is clear evidence of the necessity for them; nor should they ever in severe cases be repeated so long as the disease is at its height.

I leave to surgical writers the question whether an injured limb should be amputated on account of the supervention of tetanus, and whether the excision of nerve trunks is, or is not, advisable. But there can be no doubt that any small festering cut or sore that may be present should be poulticed and soothed; and another point of importance is that in some cases a splinter of wood or some other foreign body has been unexpectedly found beneath a cicatrix, and sometimes even embedded in a nerve trunk.

As regards the treatment of tetanus by drugs, the first thing to be said

is that at present we know of no medicine sufficiently potent to arrest the disease in those cases in which its symptoms advance uniformly and quickly toward a fatal termination. Rose, indeed, mentions an instance in which trismus set in and was followed in a few hours by a violent paroxysm of opisthotonos, but in which no further development of tetanus occurred after the administration of one-sixth of a grain of acetate of morphia, and the liberation of a quantity of pent-up pus and putrid matters by the knife. But, so far as I know, this case is unique. When the disease runs a rapid course, one can often do nothing better than keeping the patient continuously under chloroform, so as at least to secure euthanasia.

On the other hand, when the progress of tetanus is slow, it often seems that the death of the patient is little more than an accident, from the supervention of a paroxysm which happens to exceed a certain limit of severity. In such cases it is reasonable to suppose that narcotic medicines, or those which depress the activity of the muscles, may directly save life. As might be expected, opium has been largely employed. Persons suffering from tetanus have been found to be highly tolerant of it. Sir Thomas Watson mentions the case of a lady who took more than four ounces of laudanum a day during twenty days, and who recovered. And he refers to another case in which an ounce of solid opium was swallowed in divided doses every day for three weeks. Of late years, the hydrate of chloral has sometimes been used in cases of the same kind with apparent success. In 1870, for instance, Mr. Birkett had under his care at Guy's a man, aged twenty, who, on June 24th, had received a kick over an old ulcer. Next day he felt rigidity of the muscles of the face and was unable to swallow solids. He was admitted on the 28th. At first, thirty grains of chloral were given every four hours, but on the 30th, thirteen doses having been taken, a grain of opium was ordered to be taken every three hours instead. However, the spasms became more severe; and on July 4th a drachm of chloral was prescribed at one dose, and half a drachm on the following evening. After this fifteen grains were given every other hour until the 11th, when the quantity was increased to twenty-five grains every other hour. Two days later it was reduced again, and the disease now gradually subsided in the course of the next three or four weeks. A full report of this case will be found in a paper in the "*Guy's Hospital Reports*" for 1878 by Dr. Frederick Taylor, who remarks that in some other instances chloral has been found to give rise to great drowsiness, without very much affecting the spasms.

Among the depressants of muscular action the Calabar bean and curare are those which seem most likely to be useful. The first of these medicines has been employed at Guy's, in one case which terminated favorably. The patient was a man, aged twenty-one, who, having got drunk on Nov. 30th, 1874, began to suffer from stiffness in the back on Dec. 1st. He was admitted, under Dr. Wilks, with fully-developed tetanus on Dec. 5th, spasms recurring every three or four minutes. The extract of Calabar bean was given at first in small doses, but afterward one grain of it every two hours; and on the 10th, after a very severe seizure, seven doses of a grain each were administered at intervals of fifteen minutes in succession. He began to improve about Dec. 17th, but the stiffness of the joints did not finally disappear until after the middle of January, 1875. Against this case, however, must be set another one which came under my care in the summer of 1875, and in which death occurred on the sixteenth day, in spite of two and a half grain doses of extract of physostigma, repeated at one time at intervals of only an hour. Subcutaneous injections of curare were administered to a man who was under the care of Mr. Durham in 1876; but it seems doubtful whether the doses of $\frac{1}{400}$ to $\frac{3}{200}$ of a grain which

were employed were sufficiently large to be really efficacious, although the report says that they were followed by sleep and relief of pain.

Another medicine which has now and then been used in cases that have ended in recovery is quinine. An instance of its administration with apparent success was recorded by Dr. Bright, in 1836, in the first volume of the "*Guy's Hospital Reports.*"

It must, of course, be admitted that the occasional subsidence of tetanus under different remedies is not, in itself, a proof that they are really of value. But, on the other hand, I think it is equally true that the occurrence of a rapidly fatal termination in a far larger number of cases, in spite of the same remedies, does not show that they are useless. I am therefore not greatly discouraged by the collection of cases published by Dr. Taylor in the "*Guy's Hospital Reports*" for 1878, where there are recorded nine cases in which death occurred under chloral (six by the fourth day, and the other three on the eighth, tenth, and twelfth days), and seven cases which ended fatally under Calabar bean (six by the fourth day, the seventh on the eighth day). What is really wanted is a sufficiently extensive experience to enable one to say whether the number of deaths at advanced periods of the disease can be diminished by the administration of any drug.

Hydrophobia.—From the time of Aristotle it has been known that dogs are liable to a fatal disease which they transmit by their bite; and by Celsus this disease, when occurring in man, was called "Hydrophobia," from the dread of water which is one of its chief symptoms. But it so happens that in all other animals, except man, that very symptom is absent; and some writers consequently prefer to use the name of Rabies, or that of Lyssa, as being more generally applicable. I think, however, that there are advantages in the usual English practice of indicating by the special title of hydrophobia the fact that the human form of the malady is clinically distinct.

Incubation.—Judging from what we know of the inoculation of poisons in general, we should have expected that the introduction of the virus of rabies through a bite would have produced its effect after a definite interval, and without much delay. The contrary, however, is the case. After the healing of the wound, which takes place naturally and quickly, there occurs a "period of incubation," which is often prolonged beyond that of all other infective diseases, and which is of the most uncertain duration, both in the lower animals and in man. Different writers state its average length somewhat differently. Bollinger, in Ziemssen's "Handbuch," says that in 60 per cent. of all cases in the human subject it is between eighteen and sixty days, in 6 per cent. between three and eighteen days, and in 34 per cent. longer than sixty days. How protracted it may be we cannot yet positively tell. Instances have been recorded in which three years, five years, even twelve years, were supposed to have elapsed. In 1854, there occurred at Guy's Hospital, in the practice of Dr. Hughes, a case in which it was said that five or seven years had passed since the bite. Some writers, indeed, endeavor to discredit such statements, suggesting that a subsequent infection took place without the patient's knowing it. And that this is possible must, I think, be admitted; for, on the one hand, the origin of hydrophobia is sometimes altogether inexplicable, whereas it is most unlikely that it ever arises *de novo;* and, on the other hand, Mr. Youatt succeeded in tracing certain cases to sources that might very easily have been overlooked; one, in a man, to his having tried to untie with his teeth a knot in a cord by which a rabid dog had been confined; and another, in a woman, to her having used her teeth to press down the seam in mending a tear in her dress caused by the bite of an animal suffering under the disease. It has been suggested that fleas and other creatures which live upon the blood of

the higher animals may, perhaps, sometimes take up the virus and inoculate it. But, for my own part, I fail to understand why, seeing that we must admit that the incubation of hydrophobia may vary from a few days to several months, we should find very great difficulty in allowing the possibility of its being prolonged almost indefinitely.

Prodromata.—In some cases the earliest indication of the onset of hydrophobia is afforded by an uneasy sensation in the seat of the bite, which becomes painful, or tingles, or itches, or feels cold; sometimes the cicatrix itself may be seen to be reddened, livid, or swollen; perhaps it may even open afresh, or there may arise about it a papular eruption. The pain or other morbid sensation extends upward along the nerves; from the hand, for example, into the arm and up to the shoulder, as in a coachman whose case is recorded by Sir Thomas Watson. In a gentleman who came under the care of my colleague, Mr. Cooper Forster, in 1866, the pain was of extraordinary severity; it came on in paroxysms, obliging him to stop suddenly in the street, and to cry out; it was not referred to the course of any particular nerve. There was, in that instance, no redness or tenderness of the cicatrix. The arm, however, felt much colder than the other one. He had been bitten eleven months previously; in the intervening period of time he had three or four times complained of pain up the arm and twitchings in the hand. It seems very doubtful whether there is any tendency to lymphatic inflammation, beyond what might arise after any other injury of an equal degree of severity. Watson cites two instances of such an occurrence; but in one of them, at any rate, the red lines which extended up the patient's arm were probably due rather to a caustic, which had been liberally applied, so as to produce a terrible wound. In one case, however, Klebs is said to have observed after death a reddened and swollen state of lymphatic glands in various parts of the body, and also of the tonsils and of Peyer's patches in the intestine; while with the microscope he discovered in all these structures, as well as in the submaxillary glands, accumulations of certain highly refractive granular brownish bodies, which he supposed might perhaps contain the specific virus of the disease. These statements require confirmation.

Other early symptoms are a peculiar restlessness, irritability, and depression of spirits. The patient's appetite fails him; he may vomit; he complains of headache, is sleepless, and has a distressing sense of apprehension. It is curious that he often says nothing about having been bitten, and will even vehemently deny that this has been the case. And yet he may make other remarks which show that his mind is dwelling on the subject, and that he is trying to persuade himself that he need not be afraid. What is really characteristic is a repugnance to fluid, which may show itself in various ways. Sometimes there is already a little difficulty in swallowing, from a feeling of tightness about the throat. Sir Thomas Watson's patient, the coachman, refrained, on account of a similar sensation, from sponging himself as usual with cold water, though he remarked that he "could not think how he could be so silly." Very often the breathing is interrupted by frequent sighs, which may even stop the man in the middle of a sentence, while he is speaking.

Symptoms when Developed.—The above prodromal or early stage generally lasts twenty-four, forty-eight, or seventy-two hours, but sometimes it is altogether absent. In either case, the full development of the disease is ushered in by the sudden occurrence of violent convulsive paroxysms, affecting the muscles of deglutition and those of respiration, and repeated at more or less frequent intervals. They are brought on by a variety of causes. The attempt to drink almost always instantly precipitates one; the sight of fluid in a basin often has a similar effect. Sir Thomas Watson mentions a case in which the

patient was thrown into a violent state of agitation by hearing a dresser micturate, who was sitting up with him. The least draught of air, the waving of a mirror before the eyes, the opening or shutting of a door, the slightest touch upon the skin, the attempt to comb the hair, may each excite an attack. Yet there is an apparent caprice about them. Sometimes the patient succeeds in gulping down a little fluid by carrying it to his mouth with the eyes shut; or he may insist on the withdrawal of the bystanders as enabling him to swallow better. Sir Thomas Watson saw one man who so dreaded any one's breath on his face that he would not converse with the apothecary of the hospital except in such a position that the chin of each of them rested on the other's shoulder. The seizures may simply consist of a series of shuddering or sobbing movements, more or less like those which occur when one steps into a cold bath. Sometimes, however, they are attended with tonic spasms of the cervical muscles, the sterno-mastoidei (for example) starting forward strongly. The fullest description that I have met with of the more violent paroxysms is one which was drawn up some years ago by Dr. Bushell, now Physician to the British Legation at Pekin, from a case which occurred while he was dresser to Mr. Cooper Forster. "At the onset of a severe spasm the patient springs up in bed, and puts his hands furiously to his throat, as if to tear something away; the head is thrown violently back, the mouth is opened, and the eyeballs are protruded; then he makes several expiratory efforts, sometimes with a shrill, screaming cry; the head is thrown violently from side to side; the hands are tossed wildly about, beating his chest, and striking anything that is near." Sometimes the jaws are sharply brought together, so that one can hardly tell whether the patient is not trying to bite those about him. The production of a barking noise, like that of a dog, appears to be fabulous. Bollinger denies that trismus ever occurs, or a general tonic spasm like that which characterizes tetanus. Nor have I met with any recorded case in which complete opisthotonos was present, although Dr. John Ogle, some years ago, described one in which there was said to be emprosthotonos ("*Med.-Chir. Rev.*," 1868). The duration of the paroxysms is variable. According to Bollinger, they may even last from thirty to forty-five minutes. When they are at all protracted, the patient's face and limbs become pale and livid and covered with sweat. The countenance assumes an aspect of terror; the pupils are widely dilated. In women and young children the course of hydrophobia is said to be comparatively mild, although it is no less quickly fatal; this statement accords with Sir Thomas Watson's description of two of his cases, one in a lady aged thirty-two, the other in a little girl only five years old. In some very rare instances the paroxysms are said to be altogether absent, the patient being able to drink, although with difficulty, all through the disease. But, as a rule, there is an almost absolute inability to swallow, even during the intervals. The patient, who is much distressed by thirst, may resolutely carry the cup, or the glass, to his lips, but as soon as a little fluid has entered his mouth, it is forcibly ejected, and all the rest is spilt over his clothes. The tongue is said to be generally clean and moist; but the fauces and the palate may be seen to be injected, and their glands are swollen and prominent. A viscid saliva which collects in the mouth is a source of great annoyance; it is hawked up with noisy effort, and spat out upon the floor in all directions, or even upon the faces and clothes of the nurses and attendants.

Another symptom, which is present in certain cases, is priapism, with frequent involuntary emissions. Trousseau speaks of the hyperæsthesia of the genital organs as very painful.

The mental state, even between the convulsive attacks, is almost always one of great agitation. As the disease advances, the patient often raves at

those around him, accusing them of being the cause of his illness, and taunting them with gazing upon his misery. Even if what he says is not nonsense, he generally talks hastily and excitedly. Sometimes he is wildly maniacal, so that he has to be confined in a straight jacket, or to be held down by several powerful men, with whom he struggles violently.

Not infrequently, death occurs by suffocation in the middle of a paroxysm, and more or less suddenly. The whole duration of the disease is seldom more than from two to four days, and sometimes it terminates within from twelve to forty-eight hours from its commencement. Cases which last six days or longer are very exceptional; the coachman who came under the observation of Sir Thomas Watson did not die until the middle of the seventh day. In that instance the end was very gradual; the pulse grew rapid and weak, the mental powers failed, he lay moaning and tossing from side to side, frothy saliva ran from his mouth, he lost the power of moving his left arm, fluid stools were passed involuntarily, the lower extremities became cold and the coldness extended up to the chest. Some patients have toward the last become paraplegic. In the more protracted cases the spasms sometimes cease for some hours before death, the patient being quite calm and able to talk, or to drink, or to wash his hands without discomfort, but nevertheless showing, by the coldness of his surface, and by the absence of pulse at the wrist, that there is no real improvement in his state.

Origin.—Hydrophobia is doubtless caused in all cases by the transference to the patient of the specific virus of rabies; but as to the conditions under which this may occur many interesting questions arise. Thus it has not yet been finally determined whether the disease is communicable from one human being to another. At least, I believe that there are no modern or trustworthy cases on record in which it has had such an origin. Experiments made by Magendie and others seem to have shown that dogs can be infected by inoculating them with the saliva of a person suffering from hydrophobia; but if so, it is certainly strange that medical men and nurses should always escape, seeing that the poisonous secretion is so often ejected upon their clothes and hands and faces. In former times the dread of the disease was so great that the patient was often smothered between feather beds by his relations, or was allowed to bleed to death through an opened vein. Bollinger says that on the military frontier of Austria persons laboring under hydrophobia, or suspected of it, are even now liable to be shot by their neighbors, and that those who have been bitten by rabid dogs sometimes commit suicide at once.

Rabies itself may occur in many kinds of animals. It is common in wolves, and jackals, and foxes. Cats are sometimes affected by it, but surely far less frequently than dogs. A scratch from a cat is believed to have conveyed it to a human being, but in all probability the claws had been previously impregnated with poisonous saliva by being introduced into the mouth. Among herbivora, horses, oxen, goats, sheep, pigs, rabbits, and guinea pigs are capable of being infected experimentally by inoculation, or if they are bitten by dogs suffering from it. Mr. Youatt recorded a case in which a groom took hydrophobia through a scratch which he received from the tooth of a rabid horse. Even ducks and hens are said to be susceptible of it.

At one time rabies was supposed to occur chiefly in temperate climates, but the more recent observations have rendered this opinion very doubtful. Like other specific diseases, it is often absent from a place for several years together, until some accident introduces it. Thus it has recently been much more common in London than for many years before. Between 1837 and 1865 only a single case of hydrophobia seems to have occurred in the wards

of Guy's Hospital. Such facts are strongly corroborative of the view that the disease is always due to contagion. Another circumstance pointing in the same direction is that rivers sometimes seem to limit its diffusion in a remarkable manner. Schrader has stated that in 1852 and 1853, when it was raging in Hamburg, and in the neighborhood on both sides of the Elbe, no cases occurred in the islands in that stream. Contrary to the popular belief, the disease is scarcely more frequent in the hot seasons of the year than during the winter and the spring.

Of those persons who are bitten by rabid animals some only are attacked by hydrophobia; several writers agree in fixing the proportion as about 50 per cent. Those who escape, perhaps, sometimes owe their immunity to an idiosyncrasy rendering them insusceptible of the virus; at least, this appears to be the case in the dog. Sir Thomas Watson mentions that a dog was caused to be bitten on thirty different occasions, but without any result. Another circumstance affecting the transmission of the disease may be the presence of clothes or other coverings, by which the teeth are wiped clean of the virus before they penetrate the skin. Wolves are said to be especially dangerous because they fly at naked parts, such as the throat. Writers are agreed that a breach of the cutaneous surface is necessary to allow of the entrance of the poison; in Sir Thomas Watson's oft-quoted case, however, both the coachman and his fellow servants declared that the terrier dog by which he was inoculated had drawn no blood, but merely indented the skin of the hand. Mucous membranes are supposed to be capable of being infected without any interruption of their continuity. A case is recorded in which a rabid dog licked the face of a sleeping man near his mouth and communicated the disease, although not the slightest scratch or abrasion could be found after a very strict search.

A question of some consequence to pathologists is whether the virus retains its powers after the death of a rabid animal. Mr. Youatt thought that this was not the case, but Bollinger cites the case of a student at the Veterinary College at Copenhagan, who opened the body of a dog that had died of the disease the night before; his finger was slightly fissured at the time, and about six weeks afterward he died of hydrophobia.

But the most important question of all is as to the indications that should lead one to fear the subsequent development of hydrophobia when a person is bitten by a dog which may or may not be rabid. Some writers have supposed that a healthy animal may convey the disease if it should happen to be fierce and angry, but such an idea is opposed to all our knowledge. Mr. Youatt met with cases in which a dog exhibited no symptom of rabies when it inflicted the fatal bite, though it was soon afterward attacked. Thamhaya is said to have collected no fewer than eighteen or nineteen instances of this kind. The simplest way of accounting for them is to suppose that the disease is infective even during its period of incubation, which in those animals is believed to be generally of from three to five weeks' duration, but occasionally to be prolonged over two, four, or even eight months. It is evident, therefore, that when a dog is killed soon after having bitten a human being, one can never be quite sure that hydrophobia may not supervene. The proper course is to keep the animal securely confined for some weeks. I cannot in this work detail the symptoms which indicate that an animal is actually affected with rabies. Bollinger describes two varieties of the disease, one characterized by maniacal excitement, the other by paralysis of the jaw, so that it hangs down and allows a frothy saliva to run out of the mouth. In each form the bark is altered into what is described as a "hoarse, inward sound, dissimilar from its usual tone, and generally terminating with a peculiar howl." Toward the last the hind legs and the loins become paralyzed, so that the dog

staggers about and falls. One of the earlier symptoms is an extraordinary perversion of the appetite, the animal eating hair, straw, sticks, bits of leather, earth, stones, and a number of other substances, which remain undigested and may be discovered in the stomach after death, mixed with a dirty-brown, pulpy fluid. Their presence often affords a valuable indirect proof that a dog which has been killed under the suspicion of rabies was really affected with that disease. It has been laid down as a rule by some German writers, that a verdict of acquittal may be given in a case of this kind whenever the digested remains of the animal's natural food are found in its stomach and chyme in its small intestine; but, of course, the disease might even then have been incubative.

I have already remarked that a dread of water is not a symptom of rabies in any animal except man. "Mad dogs," as they are commonly called, plunge their muzzles into water and lap it up eagerly, being very thirsty, although they may not be able to swallow.

Seat.—Until lately it has been generally admitted that hydrophobia, like the neuroses in general, has no morbid anatomy, at least so far as the nervous centres are concerned. But Dr. Gowers and Dr. Joseph Coats have recently stated that the vessels in the bulb toward the floor of the fourth ventricle are surrounded by masses of leucocytes within their sheaths, and even that there are collections of them (miliary abscesses) among the nervous elements. Similar changes, but less marked, are also found in the spinal cord. I think, however, that they cannot occur in all cases, for I have seen some very successful preparations made by my colleague, Dr. Frederick Taylor, in which no deviations from the normal appearances are to be recognized.

The real *pathology* of the disease is one of the most difficult problems that we have to encounter in the science of medicine. What becomes of the virus during the prolonged and indefinite stage of incubation? What change ushers in the development of the disease itself, which is now to prove fatal within a very few days? Some writers suppose that a "recrudescence" takes place, the poison having hitherto been imprisoned in the wound, but being at this time absorbed into the blood. A German observer, named Eichhorn, is said to have described in an inaugural dissertation a case in which he found several small branches of the ulnar nerve thickened in the immediate neighborhood of the cicatrix. But others have found the nerves healthy, and even if this were not the case, one cannot imagine that rabies or hydrophobia is due merely to a transference of morbid action along them (such as, perhaps, occurs in tetanus), for fresh poison is generated, the saliva and even the blood becoming infective. Indeed, if it is the fact that a dog can communicate the disease during the period of incubation, the whole theory of recrudescence must be given up. And how it comes to pass that the symptoms are so long in making their appearance is a mystery.

Diagnosis.—Hydrophobia is not generally difficult to recognize. I do not think that there has been the least doubt as to the nature of any of the cases, all of them fatal, that have been observed at Guy's within the last few years. It is, indeed, necessary that I should allude to one of the oddest of all vagaries of medical opinion, the notion that there is really no such disease, and that all the persons who are supposed to have died of it have really succumbed either to a traumatic tetanus or to fright. This idea seems to have been started early in the century by Bosquillon, and it was upheld by Sir Isaac Pennington, Regius Professor of Physic at Cambridge, and more recently by Prof. Maschka, of Prague. But, as Sir Thomas Watson long ago remarked, young children and idiots, who could never have understood anything about the disease, have fallen victims to it, and many of the adults who have been attacked have been men of strong minds, who have refused to believe that they were seriously ill. And as for tetanus, the symptoms

in most cases are altogether different. I cannot, for my own part, agree with Rose in thinking that it is necessary to recognize under the name of "tetanus hydrophobicus" a variety of that disease which is said to be especially apt to be mistaken for hydrophobia.

It must, however, be admitted that medical literature contains a considerable number of well-recorded cases, concerning which it is perhaps impossible to say positively whether they were instances of hydrophobia or not. I am not now referring to cases which have proved fatal, for one can easily suppose that they may really have been examples of this disease, in which the symptoms were masked or otherwise irregular, as undoubtedly sometimes happens. The difficulty is with regard to those cases in which recovery is supposed to have taken place. For all experience tends to show that whenever the clinical characters of hydrophobia are present in a typical form the patient almost invariably dies, or, in other words, nearly every case in which the fatal issue has been arrested seems to have presented some aberrant feature or other which casts a doubt upon its genuineness. And yet one cannot deny that, taken as a whole, the symptoms have often resembled those of hydrophobia more closely than those of any other known disease. Let us take, for example, the case recorded by Dr. Nicholls, of Chelmsford, in the "*Lancet*" for 1878. The patient, a carter, aged twenty-five, was bitten by a stray dog in a neighborhood where there had recently been a case of hydrophobia, and where several dogs known to have been bitten were still at large. Some weeks afterward, having in the meantime thought little about the matter, he became weary, his legs ached, and he had loss of appetite. Two days later he refused a glass of ale at a customer's house, saying he could not drink it. On the fourth day he drank a quantity of coffee, but said that he "gulped it down," and complained at intervals of his throat. He was also much annoyed by trifling noises, and particularly by a toy windmill outside his house. On the fifth day, having delivered a load of coals in the country, he called at a public house, and after three efforts got down a pint of ale. He then muttered that he was as thirsty as ever, wherefore another cup was passed to him, but on attempting to sip from it he failed. After this he drove into the town, dashed through the streets at a gallop, and became maniacal and unconscious. Dr. Nicholls was called to him, and found him with a rope tied around his legs, struggling furiously, and beating his arms and head. He was uttering a peculiar noise, between a howl and a scream. His face was livid and covered with a cold sweat; his jaws were clinched; he was foaming at the mouth. Convulsions like those of tetanus then came on, and continued at intervals for about twenty-four hours, being, however, controlled to some extent by inhalations of chloroform. The opisthotonos was extreme, the body resting on the head and heels for a minute at a time. On the evening of the sixth day he partially regained consciousness, and asked for drink, which he gulped down in small quantities, the greater portion being expelled from the mouth. Two days later he displayed a remarkable horror of anything white, such as a bandage, a basin, a white glove, etc., turning aside and becoming convulsed as soon as he saw them. By about a week from this time he was well. I have already remarked that I know of no other case of hydrophobia in which spasms so like those of tetanus were present. And yet, if the disease was not hydrophobia, what was it?

In some cases of epilepsy, hysteria, or mania, there has been a spasmodic difficulty of swallowing liquids which has been more or less like that which occurs in hydrophobia. But I do not find any recorded instance in which a real difficulty of diagnosis existed, and which was ultimately proved to be an example of any one of these diseases.

Treatment.—I have already implied that the treatment of hydrophobia is almost, if not quite, hopeless. In the "*Medical Record*" for 1878, I find a

supposed case in which the symptoms were arrested on two successive days by inhalations of oxygen, and which went on to recovery under the monobromide of camphor. Another German case is said to have been cured by the subcutaneous injection of curare in doses of one-third of a grain every fifteen minutes. The only other medicine that I should be disposed to make trial of is the extract of Calabar bean in full doses. But morphia, or some other narcotic, may always be injected beneath the skin so as to quiet the patient. And he should, of course, be kept free from all excitement and disturbance, in a darkened room, and with as few persons about him as possible.

On the other hand, it does appear probable that the disease can be prevented if the poison can be in some way removed from the wound immediately after the infliction of a bite by a rabid or suspected dog. The complete excision of the injured part seems to be the best procedure to adopt; and, if that is impracticable, most authorities think that it is right even to amputate a limb, where this would be effectual. During the interval which must almost always elapse before medical advice can be sought, the wound should be washed out thoroughly with warm water, or soap and water, or a solution of any disinfectant wash or carbolic acid; in an emergency a man might even use his own urine for this purpose. Instead of practicing excision, however, Mr. Youatt trusted entirely to cauterization with nitrate of silver; and he himself was bitten seven times, and operated on 400 persons besides, among whom he had only one death, which he ascribed to fright. It is reasonable to suppose that even greater reliance might be placed on a deliquescent substance, like the *potassa fusa*. Probably it is advisable to apply some caustic thoroughly to the surface exposed by excision of the original wound. It is much to be desired that evidence should be carefully collected as to the efficacy of these various measures when applied to a cicatrix during the period of incubation, or even at the commencement of the prodromal stage of the disease. It is at least possible that they might sometimes be useful; and, of course, no chance of saving a person from so terrible a death should be thrown away.

THE PAROXYSMAL NEUROSES.

(*NERVE STORMS.*)

INTRODUCTORY REMARKS—RELATION TO OTHER NEUROSES AND MUTUAL RESEMBLANCE.

Migraine—NOMENCLATURE—SYMPTOMS—PATHOLOGY AND CAUSATION—TREATMENT.

Epilepsy—DEFINITION—E. MINOR—E. MAJOR: AURA: FIT: SEQUELÆ—PATHOLOGY, SEAT AND CAUSATION—DIAGNOSIS—TREATMENT OF THE FIT AND OF THE INTERVALS.

Paroxysmal Vertigo—SYMPTOMS—AUDITORY VERTIGO—MENIÈRE'S DISEASE.

Paroxysmal Insanity—PECULIARITIES, AND DANGER OF VIOLENCE OR SUICIDE—RELATION TO EPILEPSY—SOMNAMBULISM—NIGHT TERRORS.

We have now to enter upon a remarkable series of affections, which, although they differ widely in their symptoms, nevertheless have close mutual relations, and present many points of resemblance in their causes and in the circumstances under which they manifest themselves.

They all occur paroxysmally, at periods which are (at least in some cases) more or less regular. Many of the persons who are subject to them are in perfect health during the intervals between the seizures. We may distinguish these affections as "paroxysmal neuroses;" or, adopting the expression of Dr. Edward Liveing, as "nerve storms."

The chief among them are Migraine, Paroxysmal Vertigo, Epilepsy (including both the *petit mal* and the *haut mal*), Catalepsy, Somnambulism, and Paroxysmal Insanity. But with these several others should probably be included. I may mention Tic douloureux, Angina pectoris, Gastralgia, spasmodic Asthma, spasmodic Croup, and Laryngismus stridulus.

No modern writer has so clearly pointed out the relations of these various diseases to one another as Dr. Liveing, who, in his treatise on "Megrim or Sick Headache," insists on the fact that in the same patient they are often transformed, one into another, in the course of time. Most other writers have dealt with them from too narrow a standpoint; they have aimed at including every variety under some one single affection, such as epilepsy, rather than at giving a comprehensive view of the whole group.

An important character of these diseases, at least of most of them, is that they are essentially innate and hereditary; but I forbear to dwell on this, because it is common to many other neuroses as well; and for the same reason I pass over the circumstance that in different members of the same family the inherited tendency may show itself in different ways—one child being epileptic, another asthmatic, a third subject to migraine, etc. Indeed, I by no means wish to represent the paroxysmal neuroses as a perfectly isolated group of affections, bearing no relation whatever to other nervous diseases. On the contrary, there are unmistakable evidences of such a relation; as, for instance, between Epilepsy on the one hand, and Chorea and Hysteria on the other. But I am strongly of opinion that by studying Migraine, Vertigo, and Epilepsy together, one is led to more just conclusions about them than by viewing them either as altogether independent affections, or, on the other hand, as unconnected members of the large class of neuroses in general.

In more than one of the affections now to be described, each attack is made up of a regular *succession* of phenomena. In migraine it often

begins with a peculiar affection of sight; afterward follow in turn numbness of the fingers, vertigo, headache, vomiting, and sleep. In epilepsy there is, perhaps, first a sensation passing up from one of the limbs to the head, and then, successively, complete insensibility, a general tonic spasm, a series of clonic convulsions, and a prolonged stupor. We can hardly fail to regard such seizures as dependent upon the gradual extension of a morbid change from one part to another of the nervous centres; and it seems highly improbable that any part of them should essentially depend upon a mere alteration in the cerebral blood supply, as supposed by many writers.

Another point in which most of these affections agree is that the attacks gradually *culminate* in a certain pitch of intensity and then subside; and yet another is that there is a kind of compensation between the frequency and severity of the paroxysms, a slight one being followed by another at an unusually short interval, and *vice versâ*.

The seizures themselves are often directly traceable to *causes* which are very similar for different members of this group. In describing the different varieties of "nerve storm," one has again and again to mention gastric or hepatic disorder, irritation of the brain from dentition or disease of the teeth, exhaustion from deficient food or excessive bodily exercise, affections of the sight, smell, or hearing, as giving rise to a first attack, or sometimes to the succeeding ones also. Many of these neuroses bear a relation to puberty and the other great epochs of life, as well as in women to the occurrence of the catamenia and to pregnancy and the puerperal state. Most of them are apt to break out for the first time at some particular age; migraine and epilepsy at or about puberty, laryngismus stridulus and spasmodic croup in early childhood, and tetany in childhood—except when it occurs in puerperal women. Some of them tend to disappear spontaneously when a certain period of life is reached; thus, migraine often ceases to recur after about the age of fifty.

As I have already remarked, a feature which is common to the paroxysmal neuroses is their tendency in the same patient to undergo *metamorphosis* in course of time. This is particularly the case with epilepsy; its attacks are liable to be replaced by vertigo, catalepsy, mania, or other forms of nervous disturbance. Indeed, if we had convenient English names for the *petit mal* and the *haut mal*, it would be well that they should be described separately, for their relation to one another is not very much more close than to some of the other affections which I regard as distinct. The connection between migraine and epilepsy is certainly much less intimate, but I shall hereafter mention some cases which have been regarded as proofs that they are to some extent related to each other.

Migraine.—One very important paroxysmal neurosis is *migraine* or *sick headache*. An attack of this disease in its most typical form begins with a peculiar dimness of sight; after a little while the fingers of one hand may become numb and tingle, or the patient may experience some difficulty of speech; presently the power of vision is restored, but a more or less severe pain in the head comes on, which may last for some hours; before it ceases, vomiting often takes place. The same succession of phenomena recurs again and again at more or less regular intervals.

Great confusion prevails with regard to the nomenclature of this complaint. Many names for it are in use, but each of them is properly applicable to only one of various forms that it may assume, and several of them would naturally be taken to include other and distinct affections.

The pain is often limited to one side of the head. To such cases the name of Hemicrania (*ἡμικρανία*) has been applied since the days of the old Greek writers; and that word has undergone corruption into the French

migraine and the English *megrim*. In strictness, therefore, all these terms are inapplicable to any case in which both temples ache at the same time, but we shall presently see that such cases constitute the majority, and certainly cannot be separated from the others. Still less can those instances be isolated in which the pain is intense and limited to a very small point, and which previously received the special designation of *clavus hystericus*, based upon a comparison with the effect that might be produced by a nail driven into the skull.

Again, the defect of vision has by some writers been styled Hemiopia, or Hemiopsia, from its affecting only one-half of the visual field. Aiming at still greater accuracy of expression, Dr. Hubert Airy has recently proposed to call it *teichopsia* (τεῖχος, a city wall, ὄψις, vision) from a remarkable feature of it which I shall presently describe. But as this visual affection, even when it is present, generally constitutes only a small part of the complaint, it would be far better to let all three names fall into disuse. Lastly, we have the English terms *bilious headache*, *sick headache*, and *sick giddiness*. But many cases present no indication of gastric or hepatic disorder; and, on the other hand, there is a different form of headache in which such disorder plays a far more important part. Evidently one name should be given to all the varieties which the same disease may assume. And, if we can forget its etymology, the word migraine appears to be by far the most suitable for the purpose. Dr. Liveing, indeed, has already extended its signification in this way.

Symptoms.—I have already remarked that one symptom of migraine invariably precedes all the others, if it occurs at all; this is the *affection of sight*. The patient first notices that he cannot see distinctly some part of what he is looking at. The portion of the visual field which is thus blotted out is originally very small. It may be at the exact centre of the field, but more generally it is a little to one side of it, although so near that in reading from a printed page he has to glance slightly away from the word he wishes to see. The dim spot is not black, but seems like a faint cloud, of the same color as the rest of the surface upon which the eyes are directed. It quickly begins to enlarge, and gradually overspreads more or less of one lateral half of the field. It often acquires a peculiar zigzagged outline, with angles like those of a fortification; this is why Dr. Airy proposed the name of "teichopsia." The surface within seems to have a peculiar undulatory motion which has been compared to that of a boiling liquid; and the angles themselves appear to flicker or to revolve, The form of the cloud is originally oval, but as it grows bigger a gap forms in that side of it which is toward the centre of the field, so that it becomes horseshoe shaped. One area of the curve then seems to touch the point of sight; and in this the angles are much smaller and closer together than in the other area, which spreads away into the outer part of the field of vision. Sometimes the cloud is uncolored; sometimes it presents brilliant gleams of red, blue, and other colors. If the eyes be closed, or if the person should go into a dark room with his eyes open, the whole figure appears to be faintly luminous. As it increases in size, the middle of it clears up and accurate vision then is regained. On a printed page, for instance, a few letters can now be plainly recognized in the midst of the glimmering horseshoe-like curve. Before long, the latter likewise disappears, and the person can see as well as ever. The whole process occupies from ten to twenty minutes, or at most half an hour.

While the oval cloudy patch and its zigzagged border are visible, they are seen in their minutest details by both eyes alike. To this rule a single exception has been recorded by Sir John Herschel, who once satisfied himself that his left eye was alone affected. But I cannot help thinking that he

may have been mistaken. As Dr. Airy points out, every one is at first inclined to suppose that one eye only is concerned, namely, that of the side on which part of the visual field is blotted out. The fact that the impairment of vision is thus almost constantly referred to both retinæ, of course, proves that the seat of the affection is above the optic chiasma. And limitation to one-half of the field is just what we should expect from our knowledge of the anatomy of the nervous centres, the halves of which are in great measure isolated from one another, each communicating with corresponding halves of the two eyes. Sir John Herschel, indeed, has stated that in his own person he once observed "the shadowy pattern of a fortification which passed completely across the field of vision from left to right." If this was so, we must suppose that on that occasion the disturbance (of whatever nature) which is the cause of the affection extended over to the opposite side of the brain at an early period of the attack. We shall hereafter see that at a later period this commonly occurs.

In some cases, as soon as natural vision is restored, the attack is at an end. But in the great majority, a more or less severe headache comes on at this period. It is generally said that a precisely similar headache may also arise without having been preceded by any affection of sight. I think, however, that it is admissible to express a doubt whether this really occurs, or at least whether it is not far more rare than has hitherto been supposed. A very curious circumstance in regard to the visual affection is that some of the best and most careful descriptions of it have been written, not by medical men, but by astronomers and philosophers. Wollaston, Arago, Sir David Brewster, Sir John Herschel, Sir Charles Wheatstone, Sir George Airy, and Professor Dufour, of Lausanne, may be mentioned as having been liable to this paroxysmal defect of sight, and as having carefully noted its phenomena; and I do not know any other malady which, within the present century, has been the subject of two papers admitted into the "*Philosophical Transactions*," as well as of communications to the "*Philosophical Magazine*" and other scientific publications in this country and abroad. It may be a question whether persons who are not accustomed to employ the eyes for minute observation would notice the dimness of sight, or regard it as of sufficient importance to be mentioned to a medical man. Indeed, when it commences at some distance from the centre of vision, I believe it is sure to be overlooked, unless the patient's attention is specially directed to its occurrence. And this may, perhaps, be the reason why Professor du Bois Reymond does not mention it in describing this form of headache as occurring to himself.

The *pain* of migraine varies greatly in severity, both in different cases, and in the same case at different times. It commonly begins at some one spot in the brow or temple and gradually spreads all over these regions. Sometimes it remains confined to a single point, which is generally over the frontal or parietal bone on one side. As I have already remarked, the special name of *clavus* was formerly used when such was the case, A strict limitation, however, is altogether exceptional. According to Dr. Liveing, it is not even the rule that the headache should keep to one lateral half of the head. He finds that in the majority of cases it affects the whole forehead and both temples, although with more severity on one side than on the opposite. It often extends to the orbit, and is referred with special intensity to the back of the eye. More rarely it passes behind the ear to the occipital region. Some writers describe it as of a stabbing, cutting, or boring character; others as throbbing, and especially as undergoing augmentation with each beat of the heart. It is generally much increased by every bodily movement that the patient makes, or by exposure of his eyes to light, or by every noise that he hears. He therefore lies down, and keeps

the room as dark and quiet as possible. But Dr. Wilks tells me that he knows of one patient in whom the recumbent posture aggravates the pain, and who will sit up all night rather than recline before the attack has passed off. And Dr. Liveing speaks of cases in which the pain was so intolerable that the sufferer cannot lie in one position for any length of time, and is obliged to get up and move about. It has been said that the patient can sometimes make the visual affection more marked, if not increase the headache, by lying on one side rather than on the other; namely, the side opposite to that on which the dimness of sight is observed. But this appears to be seldom the case.

The headache scarcely ever remains long at the same pitch of intensity. Generally it goes on gradually augmenting in severity until it reaches a culminating point, after which it begins to decline. Its increase is usually steady, but sometimes this takes place by fits and starts, with more or less distinct remissions between them. When the pain becomes very intense the patient often begins to feel *nausea;* and presently he retches and is sick. Anything that the stomach may contain is rejected, including sometimes a considerable quantity of undigested food. But frequently it is empty; and then the retching is ineffectual, or some mucus at first is brought up, and afterward a bilious fluid. After the occurrence of free vomiting the pain often quickly passes off, and thus many persons regard it as curative, saying that they get relief as soon as they can be sick.

But in some individuals, however severe the headache may be, sickness seldom or never occurs. In them the pain gradually passes off of its own accord. Very frequently it lasts for the remainder of the day; the patient at length becomes worn out and drops off to sleep; and when he wakes on the following morning he finds that his suffering is over, all that remains of it being a slight soreness of the forehead or temple. Some persons lose the pain if they can sleep for a short time, even during the early part of an attack. Dr. Liveing mentions a gardener who, if he could at the commencement leave his work and lie down under the shade of a tree, would wake at the end of half an hour as well as ever. Lastly, in some rare cases, the complaint terminates by epistaxis, by a copious secretion of tears, by profuse perspiration, or by the flow of a large quantity of pale urine.

Less Common Symptoms.—An attack of migraine may be attended with other symptoms besides those which have been hitherto described. In certain cases, which are generally of considerable severity, common *sensation* is impaired in one hand, especially toward the ends of the fingers; and sensations of tingling, thrilling, or formication may be also experienced. Sometimes the surface of all one arm and of the corresponding leg seems to have gone to sleep, or numbness may extend to the neighborhood of the mouth, the lips, tongue, and throat. Dr. Liveing says that all these parts are affected bilaterally. Dr. Anstie noticed in his own person that even in the intervals between attacks of pain the power of distinguishing impressions was permanently less in the skin round the inner angle of the right eye than on the opposite side; during and after the paroxysms the impairment of sensation was always more marked, and also affected a more extensive area.

Muscular *power* sometimes seems to be more or less distinctly impaired. Ptosis and strabismus from paralysis of one of the recti muscles have each been present in cases which have been regarded by good observers as of this nature. Some patients have been known to drop things which they were carrying in the hand; but Dr. Liveing suggests that this may be due to a loss of the sensations which should guide the muscles, rather than to a true muscular palsy. Another symptom which is sometimes, but not often, present is *giddiness* or vertigo.

Again, in some cases the faculty of *speech* is disordered. The patient

has a difficulty in finding the expression which he wishes to use, or in constructing a coherent sentence. He may even substitute one name for another; and an instance is recorded by Dr. Liveing in which not a single word could be uttered. This may occur without any confusion of thought, or the patient may be painfully conscious that his memory is failing him and that his intellect is embarrassed. Actual hallucinations are very rare. But there are often much general mental depression, and a vague and unaccountable sense of anxiety and dread.

Even in those who are subject to ordinary migraine, such apparently serious symptoms are comparatively of very rare occurrence. Hence the patient may fail to perceive that they have any connection with the habitual complaint. The period at which they commence is generally before the headache, and after the affection of sight has continued for some time. An oppressive drowsiness is also sometimes present, and the patient may even lie in a half-unconscious state, not heeding when he is spoken to; but this symptom rather accompanies than precedes the pain in the head.

In some instances there are also symptoms which indicate an implication of the vaso-motor system of nerves. The temporal artery becomes enlarged, and its tortuosities are much more plainly visible on the affected side of the head; it feels hard and like a cord to the touch. The conjunctivæ may be much reddened. The *pupil* is sometimes altered in size, but observers are not agreed as to the character of the change. Du Bois Reymond says that in himself it is always dilated; Piorry and Latham describe it as being contracted. The eyeball is said to appear retracted in some cases. In one instance Möllendorff found with the ophthalmoscope that the background of the affected eye was of a bright scarlet red color, the optic papillæ red and œdematous, and the central artery and vein enlarged and tortuous. We shall presently see that stress has been laid on these various facts, as indicating that the sympathetic (vasomotor) nerves play a very important part in the production of migraine.

Again, after the subsidence of these paroxysms, certain very curious changes are sometimes observed in the tissues of the affected parts, which can only be ascribed to an interference with their nutrition consequent on the nervous disturbance. One such change is a localized *grayness of the hair.* Anstie relates that when he himself had a severe attack the eyebrow would show a distinct patch of gray opposite the supra-orbital notch, but that subsequently the individual hairs regained their natural color. He found that as many as eleven out of twenty-seven patients showed more or less grayness of the hair of the forehead and temple on the side on which they suffered most pain. In other cases the hairs become brittle or fall out. Some years ago I was consulted by one of our students, who wished me to examine the hairs from his eyebrows with a microscope to see if I could detect any fungus. More than half of each eyebrow, at its outer part, had become denuded of hair; and this condition was more marked on one side than on the opposite. I at once inquired whether he was liable to migraine, and found that this was the case, and, also, that it was especially severe on that side on which the eyebrow was the more deficient. In a few weeks, under treatment for the neurosis, the hairs began to grow again.

According to Anstie, more or less thickening of the solid tissues occurs in many cases as the result of repeated attacks of migraine; he even observed periosteal swelling, which had a close resemblance to syphilitic nodes, but which he believed to have no such origin. He also speaks of iritis, glaucoma, opacity and ulceration of cornea as resulting from neuralgia of the fifth nerve; but I am not sure whether such effects ever occur in cases of true recurrent migraine. An erysipelatoid eruption ought perhaps, however, to be mentioned; for Anstie relates more than one

instance in which a patient suffered from two or three successive attacks of this kind, in connection with neuralgic pain. I have myself seen the upper eyelid greatly swollen.

Another affection which is frequently consecutive to migraine is *xanthelasma* of the eyelids. Except in cases of jaundice, I believe that xanthelasma invariably affects the eyelids, and occurs nowhere else.* It always begins near the internal canthus, and generally first in the upper lid; and a curious circumstance is that (according to Mr. Hutchinson) it constantly appears on the left side earlier than on the right. This observer found that most of the patients in whom he noticed xanthelasma of the eyelids had suffered from frequent sick headaches, and some of them very severely. The cutaneous affection seldom appears in those who have not reached the age of thirty-five or forty years. I have seen it develop itself in three successive generations; and Dr. Church has placed on record a family tree in which it recurs again and again, but unattended with any liability to migraine.

The general *circulation* is interfered with in severe attacks of migraine. Möllendorff has found the beats of the heart reduced to fifty-two or even forty-eight per minute. The pulse at the wrist becomes small and contracted. The hands and feet are cold. The face is pale and haggard, and dark borders appear round the margins of the orbits.

It seldom if ever happens that migraine, in all its attacks in the same patient, occurs in the same side of the head. But, as a rule, there is one side rather than the other which is especially apt to be affected. Tissot, however, mentions the case of a lady who had it alternately on each side with great regularity. Wollaston and Sir George Airy may also be mentioned as having been equally liable to the affection on either the right or the left side. With regard to the mutual relations of the various phenomena which make up an attack, there are some differences of opinion. Dr. Latham describes the headache as beginning, and as more intense, on the side *opposite* to that on which the dimness of sight is noticed, but Dr. Liveing deduces from the observations which he has collected that they often both occur on the same side. Each writer says that when the affection of sight and the numbness in the fingers are present together, it is on the same side of the body. Lastly, Dr. Liveing has pointed out the fact—interesting in connection with what has been said as regards aphasia from organic changes—that when the speech is interfered with in migraine there is very generally numbness in the fingers, and that this always affects the right hand, either alone or in association with the left. He has not met with a single instance in which sensation was impaired in the *left* hand only, and in which there was any affection of the speech.

Pathology.—Migraine is undoubtedly one of the paroxysmal neuroses. As regards its anatomical seat, I have already remarked that the disturbance which causes the affection of sight must occur somewhere above the optic chiasma. Indeed, this was long ago pointed out by Wollaston, who observed that the hemiopia which he described would probably be found to arise in the thalamus of one side. And, as Dr. Liveing says, the only correction that this statement seems to require at the present time consists in the inclusion, within the area of disturbance, of the ganglia of the sensory nerves, down to the nucleus of the vagus. The order in which the symptoms follow one another in the several attacks renders it likely that the affection generally starts in the thalamus, and passes downward and backward along the sensory tract. The numbness and tingling in

* [This rule is not absolutely true. See cases reported in the "*Pathological Transactions*" for the year 1882 (vol. xxxiii, p. 372, and third table, p. 383). I have seen one or two myself, in addition.—Ed.]

the fingers or other parts are, perhaps, due merely to disturbance in the thalamus itself. But when disorder of speech and impairment of memory are present, one can hardly doubt that the change must have spread upward toward the convolutions. That it may also extend to the opposite side of the brain, along some of the commissural fibres, appears to follow from the facts that the numbness in the tongue and throat is sometimes bilateral, and that the headache very frequently is so.

As to the exact nature of the change in the sensory tract which gives rise to migraine, I do not think that anything definite can at present be stated. The most recent hypotheses refer it to vaso-motor disturbance. Thus the theory of Dr. Latham (1872) is that in the early stage the affected side of the brain is anæmic; that the contraction of the blood vessels of this hemisphere is itself due to a morbid activity of the sympathetic nerve; and that this in its turn results from a defective control or inhibition on the part of the cerebro-spinal system, which he supposes to be enfeebled. In the stage of headache he supposes that there is a secondary hyperæmia, consequent upon exhaustion of the vaso-motor apparatus. Du Bois Reymond had previously (1860) maintained that, at least in his own case, migrainous headache was due to a tetanus of the muscular coats of the vessels of the affected side, in the territory of the cervical portion of the sympathetic. On the other hand, Möllendorff (1867) and Wilks (1869) endeavored to prove that the complaint is caused by a paralysis of the very same nerves, with dilatation of the vessels and consequent hyperæmia. The former writer lays great stress on the fact (which had been pointed out nearly a century ago by Dr. Parry, of Bath) that compression of the carotid on the affected side of the head often removes the headache as if by magic, but only temporarily.

But I think it is clear that diminishing the blood supply to one side of the brain may very well suspend for a time the disturbance in it which is felt as pain, and yet the disturbance need by no means have been caused by an overflow of blood. Moreover, as Dr. Liveing points out, the statements of different observers with regard to the condition of the pupil are so diametrically opposed that no other inference seems possible, but that it must really differ in different cases; while as for the dilatation of the temporal artery, the flushing of the face, the redness of the conjunctiva, the injection of the fundus of the eye, there is direct evidence that each of them is only occasionally, not constantly, present; so that the only possible conclusion seems to be that all these vaso-motor phenomena are accidental rather than essential characters. As Dr. Liveing remarks, there is a clear analogy between the paroxysmal neuroses and certain minor consensual and automatic movements (such as those of sneezing, coughing, and gaping), and some of those which serve for the gratification of the natural appetites; yet no physiologist thinks of referring any of these to vaso-motor disorder.

Thus it would seem that at present we can form no clearer conception of an attack of migraine than that it is a "nerve storm," the result of an irregular accumulation and explosive discharge of nervous influence.

Ætiology.—The *hereditary* character of migraine is well marked. Dr. Liveing found that in twenty-six cases out of fifty-three it was said to be a "family complaint;" and the twenty-six patients in question had among them forty near relations who were liable to it. In many cases it is transmitted without the slightest change of type, and sometimes it passes from a parent to those children only who in other respects resemble him.

Women appear to be slightly more prone to this complaint than men.

The *age* at which it commences is generally about the seventh or eighth year, at the beginning of the second dentition, but sometimes it is the period of puberty, and sometimes that of early adult life. It rarely occurs for the first time in a person over thirty. The more marked the hereditary tendency

the greater the probability of its beginning in childhood. At about the age of thirty, persons often suffer from it much more severely than they had done previously; and as Anstie remarks, at this period the attacks cease in many instances to be accompanied by vomiting, so that the complaint is no longer regarded as mere "sick headache," and the patient consequently seeks medical advice, perhaps for the first time. When fifty years of age are reached, or even rather earlier, it commonly happens that the liability to migraine ceases; old people comparatively seldom suffer from it.

In some persons the attacks of migraine recur with great regularity. The period is sometimes a fortnight, sometimes a month, sometimes longer still. There are, however, cases in which it is much shorter. We shall presently see that the immediate exciting cause of the paroxysm is very often excessive fatigue from brain work. The constant repetition of this may render the attacks correspondingly frequent. I remember the case of a bank clerk, who for a considerable time had an attack regularly every week day, but was free on Sundays. And some years ago a governess was under my care who had a headache every night. In cases of this kind some of the more characteristic features of the complaint are very apt to be missing; but I believe that their relation to true migraine can often be established by the account which the patient gives of his previous state of health. Further observation may, perhaps, show that a headache which is persistent, and lasts for a great length of time, may grow out of the paroxysmal affection. Such a case would be strictly parallel to one of epilepsy, in which the so-called status epilepticus is developed.

In the cases just referred to the complaint may return every day, or every other day, as regularly as the paroxysms of an intermittent fever; and this fact, together with the striking therapeutical influence of quinine, often makes it difficult to exclude the possibility of miasmatic poisoning. But, on the other hand, these circumstances, taken by themselves, are far from justifying the conclusion that a migraine is really due to such a cause, and deserves the name of "brow ague." I do not myself believe that in persons living in London this origin of the complaint can ever be established satisfactorily; and it is probable that even in districts where marsh miasm prevails, cases of simple migraine and of other forms of neuralgia are often wrongly ascribed to it, just as I believe the same thing to have happened in the case of intermittent hæmatinuria. But it appears to be certain that it is sometimes really the cause; and in some parts of Spain a miasmatic migraine is said to be endemic. Again, Dr. Macculloch has stated that this kind of headache may occur as a substitute for ague during the whole of one relapse of the disease, and that he has seen a "double tertian" ague, in which the headache and the ague fit occurred regularly on alternate days.

In England, however, it is universally believed that migraine, instead of being essentially a nervous malady, is the result of "bilious" disorder. And, when the attack is accompanied by vomiting, this is supposed to expel a materies morbi, in the shape of vitiated bile. Until one has happened to discuss the matter with some non-medical friend or patient of average intelligence and education, one can hardly conceive how firmly fixed the belief in question is in the mind of every one who has not been taught the contrary. A notion of this kind could only be derived from the medical science of a former age; but one might well wonder how the teachings of a previous generation of physicians should have left behind them so absolute a conviction. The truth, however, is that it is a relic of one of the most ancient doctrines in the history of the healing art—that of the four Cardinal Humors, one of which was "yellow" and another "black" bile. There is not, therefore, any ground for surprise at the difficulty with which it is eradicated from the popular mind.

But although it is certain that migraine is never solely due to disorder of the chylopoietic viscera, there is yet no question that some error of diet is often the direct exciting cause of an attack in a person who is liable to it. I am not now referring to the diffused headache and giddiness which are apt to be more or less constantly present in persons who suffer from dyspepsia or from the so-called congestion of the liver; this has doubtless been often confounded with true migraine. But what is conclusive as to the reality of the influence of improper food is the fact that some persons at least can always bring on an attack by eating particular articles of diet toward the end of the interval between one headache and another; whereas, for a few days after a paroxysm, they might partake freely of the very same things without suffering in any way. Dr. Fothergill, nearly a century ago, stated that he had found nothing more apt to cause "sick headache" than "melted butter, fat meats, spices, meat pies, hot buttered toast, and malt liquors when strong and hoppy." A medical man who had suffered all his life from the complaint told Dr. Liveing that he could never take the smallest quantity of wine nor eat the smallest fragment of burnt pastry without bringing on a headache. Many persons speak of butter and pork as particularly frequent exciting causes of migraine; and, making every allowance for the influence of preconceived opinions, I do not think that the validity of such statements can be entirely impugned.

In women, again, the recurrence of the catamenia is often an exciting cause of attacks of migraine, which, perhaps, generally precede the flux, but sometimes accompany or even follow it. Not unfrequently each monthly period brings with it a series of more or less distinct paroxysms of headache. A striking illustration of this connection is afforded by a case related by Dr. Liveing, of a woman who was very liable to the complaint when menstruating, but who throughout repeated pregnancies was always entirely free from it. This writer also mentions an instance in which the headache and the catamenial discharge recurred simultaneously at fortnightly intervals.

But fatigue is a far more important exciting cause of migraine than either of those which I have mentioned. In some persons a straining effort, such as lifting a heavy weight, will bring it on, and in others the exertion of running is apt to have the same effect. Many patients are exceedingly liable to be attacked by it after protracted labor, such as a hard day's washing; or after prolonged exercise, particularly if the stomach be not duly supplied with food. Another frequent cause is severe mental work, but, above all, anxiety and worry. A long railway journey is apt to be followed by a paroxysm in some individuals, and in others merely driving in the streets of London has the same effect. Many persons always have a sick headache after a day's sight seeing, or after passing an evening in a crowded concert room or ball room; and in some susceptible individuals an attack may be brought on by glaring lights, loud noises, or the strong odors of certain flowers. Dr. Airy mentions the case of a person in whom the peculiar affection of sight was occasionally caused by looking at a striped wall paper or a striped dress; and Sir John Herschel states that he had it as the result of allowing his mind to dwell upon a description of the affection.

In several of the conditions already alluded to as exciting causes of migraine, one element is exhaustion of the visual apparatus. This is true, for instance, not only of over-study, but also of railway traveling and the like. And, many years ago, Piorry propounded the theory that the complaint, or at least one variety of it, was the result of irritation of the optic nerves, from straining efforts to see very small objects, or from want of care in regulating the amount of light. Now, this view is altogether untenable, if applied to all cases of migraine, or even if limited to that form of it in

which symptoms referable to the visual apparatus preponderate, the headache being slight. But it is perfectly true that when the eyes are structurally imperfect, the forced use of them may be the immediate cause of attacks of migraine. The defects which lead to this result are chiefly those of the transparent or refracting media of the eyes; their direct effect is the production of spasm of the ciliary muscles, and with this is associated an irritation of nervous filaments, which may diffuse itself over a wide area within the distribution of the fifth nerve. Every practitioner now knows that hypermetropia is a frequent cause of attacks of dimness of sight, headache, and giddiness, which recur when the eyes are used for near work for any length of time, particularly under artificial light. A student lately consulted me for similar symptoms, and it was not for some little time that I discovered that they were the result of the employment of too powerful concave glasses, which he had chosen without proper advice, in order to correct a moderate degree of myopia, and which he wore even when reading or writing. In this connection, too, astigmatism must not be overlooked. Another cause of such symptoms is weakness of the internal recti muscles. Three years ago I had a bank clerk sent to me who had previously more than once had to give up work for a period of two or three months, on account of cerebral symptoms. These had been thought to be of a serious character, but I had him examined by my colleague, Mr. Higgens, and he discovered that the internal recti muscles failed to make the eyes converge properly upon near objects; when suitable glasses were supplied to him, he soon lost all his complaints. In practice, therefore, one should make it a rule never to prescribe for any kind of frontal headache without eliminating the possibility of its being caused by imperfection of the organs of sight.

Lastly, affections of the teeth must not be overlooked as causes of migraine, at least, if clavus be included as a form of it. I have already referred to Mr. Salter's statement with regard to this at p. 376. One of the cases recorded by him is that of a young lady who for eight years was subject to attacks of headache, confined to a space of about the size of a crown piece, rather to the left of the vertex. They sometimes recurred three or four times a week, beginning after breakfast and lasting all day; they were attended with great prostration. The affected spot became hot, and pressure with the hand gave relief to the pain. At length the patient conceived the idea that the left upper canine tooth, which was known to be impacted in the palate, was in some way connected with her sufferings. It was removed, and she never afterward was attacked by the headache.

Diagnosis.—In its typical form migraine presents little or no difficulty of diagnosis. I have seen one or two cases in which a syphilitic periostitis of the margin of the orbit has produced recurrent pains of somewhat similar character; but this could not escape the notice of any but a very careless observer.

When the phenomena of the attacks are ill developed, however, one may not find it easy to determine whether they belong to the disease now under consideration, or to some other neurosis. I have more than once been disposed to think that migraine and the *petit mal* of epilepsy are really linked together by transitional forms. Another form of migraine about which one may fall into error is that in which an affection of speech is a main symptom. Some months before his death, the late Dr. Phillips had a severe attack of headache, attended with marked aphasia. When he had recovered, I one day happened to discuss with him the question whether it could have been of the nature of migraine; but his fatal attack of apoplexy, which doubtless was the result of embolism, afterward began in precisely the same way, and presented the very same symptoms. It seems to me very doubtful whether Dr. Liveing is right in regarding as a mere paroxysmal neurosis a case in

which attacks of loss of speech and right hemiplegia recurred, persisting for a week or more at a time.

Relation to Other Neuroses.—Migraine is so common a complaint that one can hardly attach very much importance to the mere fact that some other neurosis occasionally develops itself in those who are subject to it, as indicating that there is any real relation between them. Dr. Liveing, however, believes that a transformation sometimes occurs between migraine and epilepsy; and he relates cases in which persons who had suffered from the former afterward became affected with the latter disease; but it is to be noted that some of them had relations who had been epileptics. This writer also refers to an instance in which migraine became replaced after a certain period by asthma; and to another in which a constantly recurring gastralgia disappeared and was followed by a typical migraine, while this in its turn was succeeded by a kind of spasmodic croup. He also relates a case in which attacks of sick headache were followed after a time by angina pectoris; and another in which insanity developed itself.

I do not think that any medical man can have suffered again and again from migraine without having the thought forced upon him that such attacks must indicate a defect of cerebral organization which might subsequently result in serious mischief. And Dr. Liveing quotes Calmeil as having remarked that both the intellectual faculties and the moral disposition of the patient are sometimes impaired by the repeated occurrence of migraine. He also refers to the cases of Parry and Wollaston, both of whom, after having long been subject to this complaint, died of organic cerebral disease. But such results are in the highest degree rare and exceptional.

Treatment.—This involves two distinct questions: (1) the management of the patient during the intervals between the attacks of migraine, with a view to prevent their occurrence or diminish their severity; and (2) the treatment of the attacks themselves.

(1) Under the first head hygienic measures are of primary importance. One must insist upon the importance of daily exercise, short of fatigue, in the open air; one must prohibit an excess of brain work, and take care that the patient is as far as possible shielded from domestic worry and anxiety. The state of the digestive organs must be carefully inquired into; and due weight must be allowed to any indications of dyspepsia or lithæmia, or to evidence that the attacks are brought on by errors of diet. But in many cases it is a mistake to restrict the patient too closely to what is termed plain food. Rather he should be advised to take a freer quantity of fat, in the form of butter or cream. Alcoholic beverages, however, should generally be avoided, or sparingly indulged in. In severe cases a change of climate is often advisable, and particularly a sea voyage.

One of the good results which may fairly be anticipated from the discussions which have taken place within the last few years as to the nature of migraine is that those who have the care of young people should look out for the early manifestations of the complaint during childhood or after puberty, and insist upon the avoidance of over-study and of undue excitement in those who seem likely to suffer severely from it. Until recently, migraine was almost universally looked upon as being at once incurable and almost unworthy of notice on the part of a medical adviser. But the truth is that if systematically taken in hand it is very amenable to treatment; and those who suffer from it know best how serious a matter it is to them, interrupting, as it frequently does, all the engagements and pleasures and duties of life.

Among medicinal agents Dr. Liveing says that he has sometimes found the regular administration of belladonna and hyoscyamus of great service.

Cannabis indica is also recommended; and I have prescribed it more than once with decided advantage. Bromide of potassium is, perhaps, more generally useful.

In many cases the preparations of iron (especially the sesquioxide and the saccharated carbonate) are very serviceable, and this even though there may be no anæmia. Strychnia in small doses (one-twentieth of a grain) is said to be another valuable remedy. I have myself prescribed arsenic with marked success; it was long ago recommended by Dr. Bright and Sir Thomas Watson. Quinine is said to be less serviceable than it is in many forms of neuralgia, except in cases of malarious origin.

The remedies hitherto mentioned possess physiological properties which explain in some measure their beneficial action in the prevention of attacks of migraine. But there are other medicines of which the good effects are less readily intelligible. One such is iodide of potassium. Dr. Todd is said to have found this more successful than anything else; and Dr. Liveing mentions a case in which the attacks were so frequent and severe as to render the patient's life a burden to him, and in which five grains of the iodide three times a day set him almost free from them. In other cases chloride of ammonium seems to answer better than anything else. Valerian and valerianate of zinc are said by Dr. Liveing to be sometimes of great value.

(2) The treatment of the paroxysms of migraine must necessarily depend on their severity; it is only in very violent attacks that the patient is likely to seek for medical advice. Of his own accord he will return to a darkened room and maintain absolute quiet. If the feet be cold, they should be wrapped in blankets, and a hot bottle should be placed in contact with them; or they may be suspended in hot water to which some mustard has been added. Hydrate of chloral may then be administered in a dose of from twenty to thirty grains. Dr. Anstie speaks of it as being of the greatest possible value in quickly bringing sleep to the patient, who, when he wakes up, may be free from pain. Bromide of potassium also is very useful; a scruple, or half a drachm, taken when the sight begins to be affected, sometimes cuts short the attack. At this period a cup of strong tea or coffee is in some persons capable of producing the same effect; even sipping hot water gives relief in some cases. But the best remedy of all appears to be guarana. This drug—a compound prepared in Brazil from species of *Paullinia*—has been principally recommended in England by Dr. Wilks; half a drachm of it may be taken, mixed with water; or about twenty or thirty minims of the liquid extract. Even this, however, is sometimes altogether useless. Dr. Anstie mentions the extract of cannabis indica, in a dose of a quarter to half a grain, as being very serviceable in the migraine of the young; he says the dose should be repeated in two hours if sleep be not obtained. In some cases a full dose of brandy or a glass of sherry or champagne is very effectual; but, as Dr. Anstie points out, there is great danger in allowing a patient to fly to such remedies for the purpose of allaying pain.

Locally, some measure of relief may be afforded by the pressure of a handkerchief tied tightly round the brows. Dr. Liveing mentions a case in which plunging the head into cold water was often effectual. In some cases it has been found advantageous to apply to the seat of pain a little piece of cotton wool, on which a few drops of ether have been poured, and which is covered with a watch glass; and the bisulphide of carbon has been used in a similar manner. Trousseau speaks highly of the application of extract of belladonna to the painful temple; and Dr. Anstie says that a diluted ointment of veratria is often very serviceable.

Another measure which appears sometimes to be highly successful is the application of the constant galvanic current. Dr. Anstie recommends

that it should be passed from one mastoid process to the other. Only three or four cells should at first be employed, and never more than ten; it should be applied for but half a minute at a time, and repeated once or twice a day. Giddiness is very apt to be produced unless it be used with great caution.

Epilepsy.—Epileptiform fits accompany the onset of variola and other exanthemata; they occur toward the last, when life is being extinguished by hemorrhage; they result from Bright's disease of the kidneys, and they are produced by various organic affections of the brain.

But, in remarkable contrast with the various cases just referred to, in all of which the fits are accidental and occasional, or even solitary fits of precisely the same kind return again and again in many patients for years together, and often without being traceable to any cause except an inherited tendency to nervous disorder. One cannot but regard such cases as essentially distinct from the others, and as requiring a special name; and it accords both with custom and with common sense that we should speak of them as *epileptic*, and call fits which are of accidental origin *epileptiform*. To the latter, indeed, we may (if we choose) apply the designation of "eclampsia," which is commonly employed by obstetric writers for the fits that are apt to occur after childbirth.

But in limiting the use of the word epilepsy to a recurrent paroxysmal neurosis, one must carefully avoid an error into which it is easy to fall, namely, that of imagining that underlying the attacks there is an essential disease which can be conceived to exist independently of them, and of which they are only symptoms. The fits themselves constitute the disease, the only other element in its definition being the clinical fact that they tend to return at more or less regular intervals for an indefinite length of time. We shall soon see that the distinction between *epileptic* and *epileptiform* attacks is not always easily applied, and that some cases of very long standing should properly be classed with the latter rather than with the former. And, on the other hand, a single fit, which is never repeated, may belong to epilepsy in the strictest sense, although the tendency to recurrence is overcome by treatment, or is never called into activity.

Epileptic attacks vary greatly in severity. And, in accordance with the universal practice, I may divide them into two principal groups, the one constituting what the French term the *petit mal*, the other what they term the *haut mal*. In Latin these have respectively been called epilepsia minor and epilepsia major, and it would be convenient to adopt similar expressions in English, since our language has hitherto been deficient in names for them. The *petit mal* is, indeed, commonly spoken of as *epileptic vertigo*, but we shall presently see that there is more than one reason why the use of the term in such a sense should be avoided.

Epilepsia Minor.—A paroxysm of the *petit mal*, or minor epilepsy, may be a mere suspension of consciousness, quite sudden in its onset, and scarcely more than momentary in its duration. For two or three seconds the patient is lost, but he quickly recovers himself and goes on with what he was doing. Dr. Wilks mentions the case of a shopman who often had a seizure of this kind while serving a customer, and who believed that no one noticed it. In some instances such attacks are attended with muscular rigidity. Dr. Chambers relates that a well-known lecturer would sometimes be seized while addressing his class, and would stop in the middle of a sentence, remaining perfectly still, with mouth open and arms extended; after a minute or two he would go on just where he had left off, without knowing anything that had happened. Dr. Chambers, indeed, puts this case as one in which the *petit mal* assumed the characters of *catalepsy*, but it does not seem clear

to me that it really deserved that name. At any rate, its close relation to epilepsy was shown by the circumstance that the gentleman in question afterward suffered from that disease in its ordinary form.

In other cases, if the patient should happen to be speaking at the time when he is attacked, he loses the thread of his discourse, and afterward he cannot remember what he was saying or what was said to him. Or he may stagger and lean against something, or grasp at it for support. He may experience a sensation of giddiness and lie down to avoid falling. He may even fall. Dr. Wilks mentions one instance in which a little girl would be sitting in her chair stitching, when she would suddenly fall, but before the nurse could reach her to pick her up she would be in her seat and again at work. To such cases the name of "epileptic vertigo" would be fairly applicable, if it were not undesirable to separate them from the rest, and if there were not another *paroxysmal vertigo,* which bears no close relation to epilepsy. I have already had reason to remark that some writers call any nervous affection epileptic which recurs paroxysmally; consequently the expression "epileptic vertigo" must necessarily be ambiguous.

If one carefully investigates an attack of minor epilepsy, one may notice that the pupils become slightly dilated, and that the patient no longer seems to be looking at anything. The face often becomes pale, and afterward slightly flushed. Dr. Reynolds says that the pulse may falter and become irregular. Some years ago, while I was one day listening with the stethoscope to the heart of a man whom I had never seen before, its beats suddenly ceased. I looked up and saw that his face had turned deadly pale. He said that he was going to faint, and reclined back on the chair from which he had risen. I could feel no pulse at his wrist, and for an instant I thought that he might be going to die, but I had hardly time to ring my bell when the color returned to his face, and I found that his heart was beating again. Presently there was a little twitching of the muscles in one or both hands. In a minute or two he was able to tell me that he often had "fainting fits," and that some years back he had been subject to epileptic seizures. It appeared clear that the attack which I had witnessed was one of the *petit mal.* In a large majority of cases, if not always, the so-called fainting fits which are so apt to occur in children and others are really of an epileptic nature.

It is, however, certain that in some instances the circulation in the face and limbs goes on without interruption during an attack of the *petit mal.* Dr. Reynolds says that he can testify to this from repeated observations.

Not infrequently, an attack which is in all other respects one of "minor epilepsy" is accompanied by some slight convulsive movement. There is a transient strabismus; the mouth is drawn to one side; the whole head is turned toward one shoulder; or the body generally becomes for an instant rigid. Cases of this kind form links between the two main varieties of the disease, and show how closely they are related to one another. A further proof is the fact that in perhaps the majority of cases in which attacks of the *petit mal* occur again and again, they are after a time replaced by those of the *haut mal.* Or a patient may suffer alternately from the one and from the other.

Prodromata.—Before I describe the phenomena which constitute a regular attack of the *haut mal,* I must allude to certain sensations which some patients experience at the commencement, and which may even precede the other symptoms so as to afford a warning of the approach of the seizure. To all such phenomena the name of *epileptic aura* is commonly given by a convenient extension of its original meaning; it was first applied to a feeling, as of a draught of air, passing over the surface until it reached the head,

whereupon the patient became insensible. Sometimes a creeping or a dragging sensation is experienced, which begins in the fingers of one hand, or the toes of one foot, and rapidly moves upward. Dr. Bazire relates a case in which it first affected the wrist and then extended downward to the tips of the fingers, the nails feeling as though they were pulled off at the roots. Dr. Radcliffe had a case in which it was a painful sensation, always referred exactly to the foramen cæcum at the base of the tongue. Other cases have been recorded in which an aura has consisted of a painful sensation at the epigastrium, attended with nausea. Or again, there may be loss of sensation in one side of the face, or in one limb. Nothnagel mentions an instance in which numbness always began in the right shoulder and side of the head, and spread downward into the right arm, the right leg, and the right half of the body. When an aura passes up a limb its path seldom corresponds with the course of any particular nerve. In some rare cases it seems to start from the seat of a former injury. Sir Thomas Watson quotes a case in which it proceeded from an old cicatrix in the side. Dr. Wilks alludes to one instance in which it was referred to a painful corn on the toe. He also states that a little girl under his care localized her aura in a sore spot on her face, and that her father assured him that the application of laudanum to that part would sometimes arrest the fit.

In other instances it would seem that an epileptic aura is due to an affection of the vaso-motor nerves of some part of the body. The patient, perhaps, experiences a sensation of coldness or weight in a limb; and the part is found on examination to be pale and cold to the touch, and to have its sensibility distinctly blunted. Trousseau says that when an aura occurs in a finger it is sometimes a little swollen, so that the rings on it which before were loose suddenly become tight.

In other cases, again, an epileptic attack is preceded by a profuse secretion of tears, or of saliva, or of sweat, as in several cases that have come under Nothnagel's observation.

Or, what has been termed a "motor aura" may occur, in the form of tremor or of light spasms. The eyelids may twitch, or some muscles of the face, or of one limb. Or complex movements may be performed, the patient, perhaps, turning round, or running some distance. It is said that a transient paralysis of one limb may take place under the same circumstances.

Or, yet further, the epileptic aura may be referred to one of the special senses. Joseph Frank is said to have met with an instance in which it always took the form of a sweet taste. Still odder is Gregory's case of a man who always fancied he saw a little old woman in a red cloak; she seemed to come up to him and to strike him a blow on the head, whereupon he lost all recollection and fell down. Sometimes the only warning is a vague sensation of fear. Dr. Reynolds was told by a gentleman that what always passed through his mind was, "This is what I had foreseen. I knew it would come on here; I ought to have avoided it by remaining away,"—although in reality he had not before had any suspicion that a fit was impending.

An epileptic aura may last for a few seconds or some minutes, or even (it is said) for two hours. Nothnagel mentions that when its duration has been more than ten minutes he has been able to ascertain that there was distinct loss of sensation in the part affected by it. Sometimes it occurs and passes away without being followed by a fit. In one of Nothnagel's patients a vaso-motor aura in one leg is said to have come on as many as six or even ten times daily, whereas she had only about one epileptic attack in a week.

Symptoms of the Fit.—An actual attack of the *haut mal* begins by the

patient suddenly, almost instantaneously, falling unconscious, with strained and rigid muscles. As he is seized, he may utter a sharp, shrill cry or yell; or he may be perfectly silent, the respiratory movements being arrested by spasm; or he may emit a smothered, groaning sound. In many cases the face becomes pale; but in some its color remains unchanged. I believe that the heart always goes on beating and that the carotid arteries continue to throb. But the radial pulses are sometimes imperceptible, this being, in all probability, due to the contracted state of the muscles of the upper limbs.

The *tonic spasm* which occurs at this period of an epileptic or other kind of fit is peculiar; it usually affects one side of the body more powerfully than the opposite side; the head is turned round toward one shoulder, and the eyeballs are strained in the same direction. The pupils constantly become dilated; but Dr. Reynolds says that in one instance he observed a momentary contraction before dilatation began. They are generally, if not always, insensible to light. Touching the conjunctiva commonly excites no reflex movements, but according to Romberg it is sometimes followed by closure of the lids; this observer also states that sprinkling the surface of the body with cold water may in some cases cause shrinking movements. Soon the face flushes, and acquires a dull red or dusky hue. This appears to be in great measure due to the fact that the large veins of the neck undergo compression, and that the flow of blood through them is interrupted by the spasmodic contractions of the sterno-cleido-mastoideus and neighboring muscles. Spasm of the glottis seems also to occur.

The stage of tonic spasm may last only two or three seconds, or it may be prolonged to thirty or forty seconds. Trousseau says that in some rare cases it extends over two or three minutes until the patient dies asphyxiated. It is followed by a stage in which *clonic spasms* form the most striking feature. These also are generally more marked on one side than the other. The fingers of one hand are alternately flexed and extended; the like movements simultaneously occur in the other joints of the arm, and even in the corresponding lower limb, and the eyes are twitched violently toward the affected side. The opposite limbs may escape entirely, or be affected with less powerful convulsive movements. The pupils may now oscillate between a state of contraction and one of dilatation. Dr. Reynolds says that they are not always altogether insensible to the influence of light. The jaws are forcibly contracted, and the tongue is often caught between the teeth, and gets bitten on one side. The blood which comes from it is mixed with a secretion that is poured abundantly into the mouth and air passages; and a red foam is blown out through the clinched teeth. The face is now always of a livid, purple hue. There is often profuse sweating. The urine and fæces are apt to be involuntarily ejected, and emission of semen may occur.

This second stage—of clonic spasm—may last two or three minutes; according to Dr. Reynolds, even ten minutes. Trousseau, however, says that it scarcely ever continues as long as from four to six minutes; as he points out, one is very apt to be deceived as to its duration, unless one actually reckons the time by a watch. As the convulsive movements pass off they become slightly altered in character. They are no longer wholly meaningless. The patient often draws a deep sigh; he may endeavor to change his position and may look at those about him with a bewildered, or suspicious, or sorrowful expression. Dr. Reynolds makes of this a third stage, which, however, seems to be hardly needed. He says that the pupils are now contracted.

When the fit passes off, the patient may at once regain his consciousness; or he may be more or less confused for a time; or, more frequently, he becomes drowsy and passes into a deep sleep or stupor, which may last several hours, and is often attended with guttural stertor of the breathing.

During this time, if he can be roused at all, he is generally very irritable and peevish. Slight clonic spasms not infrequently occur.

Exceptional Forms.—An attack of major epilepsy, however, does not always conform strictly to the above description. It is sometimes attended with but very slight convulsive movements, or, perhaps, even with none at all. Such cases were formerly described as examples of a form of apoplexy, or under the name of "apoplectiform cerebral congestion," as I have stated at p. 527. Even when an epileptic fit occurs during sleep, one can very often make out its real nature by the fact that the tongue next morning is found to be sore, or that the urine has been passed involuntarily, or even the fæces. As Trousseau points out, if a patient who had never before had difficulty in retaining the contents of his bladder at night, should now and again find that he has wet his bed, this mere fact should arouse the fear that he may be an epileptic. A similar inference may sometimes be drawn from the presence of minute scattered red petechial spots, very like flea-bites, which occur chiefly on the forehead, throat and chest. These may frequently be observed after an ordinary fit of the *haut mal;* and sometimes they follow a masked attack, such as I am now describing. A point which may be mentioned incidentally is that when petechial hemorrhages into the skin occur after an epileptic attack in a person who has not before been subject to fits, they may be wrongly regarded as an evidence that the patient is suffering under blood poisoning or that an exanthem is about to appear. Dr. Wilks has related a case in which this mistake occurred.

We have seen (*vide supra*, p. 550) that tumors and other local organic diseases of the brain frequently give rise to fits which are unattended by loss of consciousness, and consist mainly of convulsive movements, perhaps limited to certain parts. It might be expected that similar fits should sometimes occur in genuine epilepsy, either alternating with ordinary attacks of the *haut mal* or preceding them, as is so commonly the case with those of the *petit mal.* All observers, however, are agreed that this is exceedingly rare. The only two instances that I have read of are recorded, the one by Trousseau, the other by Nothnagel. The former occurred in a young man, aged eighteen, who was liable to convulsions of the facial muscles, affecting only the left side, and not accompanied by loss of consciousness nor by any other epileptic phenomena. But the clinical history was that the disease had first set in six years previously, with violent fits of the *haut mal*, and that these had gradually become milder and passed into those of the *petit mal*, which (I suppose) occurred alternately with the attacks of mere partial clonic spasm. Nothnagel's case was in a boy, aged sixteen. Dr. Reynolds, indeed, who gives to this form of the disease the very appropriate name of abortive epilepsy, furnishes a list of references to various writers as having described it. I have looked up most of them, but it is not clear to me that the writers in question took care to exclude cases of cerebral tumor and the like. Thus we scarcely meet with an exception to the rule that there is local disease of the brain in all cases in which attacks of clonic spasm recur paroxysmally without loss of consciousness.

Recurrence.—The frequency with which the attacks of epilepsy return varies greatly in different cases. A patient may have one fit without ever having a second; and yet, as I have already remarked, it is possible that it was epileptic in the strictest sense of the word. Or after an interval of two or three years he may be again attacked. In some cases the paroxysms return once, or twice, or three times a year; in other cases more or less regularly once a month. In women they are on the whole more apt to occur at the catamenial periods than at other times. But Dr. Reynolds says that monthly recurrence is actually more common in the male than in the female sex; and it is very rare, indeed, for a woman to be liable to fits of an epileptic character only

while menstruation is in progress. More frequently the interval at which the attacks recur is less than a month. According to Dr. Reynolds, in half the cases that he analyzed the average period of return was between fourteen and thirty days. Lastly, there may be one paroxysm, or even more than one, every day. A common thing is for two or three or more fits to occur on the same day or within two or three days, and then for the patient to be free from them for several weeks. They are then sometimes said to recur in *series*. When they return with very great frequency during a lengthened period, they almost always belong to the *petit mal*. For attacks of the *haut mal*, if they repeat themselves several times a day, must, with the stupor that follows them, take up a considerable part of the twenty-four hours, and this necessarily impairs the patient's health, so that it cannot go on indefinitely.

There is, indeed, a special modification of the disease in which the fits follow one another in rapid succession, so that before the patient has recovered completely from one of them another comes upon him. This has by modern French physicians been called the *état de mal épileptique*, and in England some writers have made use of the equivalent expression, *status epilepticus*. When it is at its height, the convulsions follow one another with extraordinary rapidity. Dr. Crichton Browne, in describing such cases, says that the limbs are scarcely laid to rest after one fit before they are tossed and contorted by another, and even in the intervals there are frequent muscular twitchings. The patient lies perfectly unconscious; his heart beats rapidly and tumultuously; his respiration may either be quick and shallow or slow and labored. His features are swollen and livid, and his lips purple. His body is bathed in profuse perspiration, and the temperature is raised to 105°, or even higher still; as Dr. Merson has noted, to 107.8°. This condition often terminates fatally; indeed, it constitutes one of the principal ways in which epileptic patients die. Charcot observes that in such cases bed sores over the sacrum develop themselves rapidly. He also mentions that a temperature as high as 105.8° is sometimes reached without death ensuing.

Complications.—Certain occasional results of an epileptic fit have yet to be described. One is a loss of muscular power in one arm, or in both the arm and leg on one side, generally that side which was the more convulsed. Dr. Todd described this under the name of epileptic hemiplegia; it may either pass off in a few hours or last some days. One must not forget that diminished mobility of the arm after a fit is sometimes due to a very different cause, namely, to dislocation of the shoulder, produced sometimes by muscular spasm, sometimes by direct injury, the patient having struck that part in falling. I myself once nearly overlooked this accident. A patient came with her arm hanging helpless; I was pointing out to the students that a transient paralysis often follows an epileptic attack, when I happened to notice that touching the limb gave pain. I grasped the deltoid muscle, and found that the head of the humerus was out of place. At one time a woman, who was liable to epileptic fits, used frequently to come to the hospital to have her shoulder set; in her case the fact that the same dislocation occurred again and again, showed that spasmodic contraction of the muscles was its cause. Still more serious injuries sometimes arise during a paroxysm. The skull may be fractured by the patient dropping down on the pavement or on a stone floor; or he may be severely burned if he should happen to fall against the bars of the grate; or he may, in a crowded thoroughfare, be run over. Persons who are liable to epilepsy cannot be too closely looked after.

Delirium has not hitherto been mentioned as accompanying epileptic fits, but in some cases it is one of their most important features. Indeed, in a person subject to the disease, the attack may be wholly replaced by one of

paroxysmal insanity, such as will be described in the next chapter. Or, again, the epileptic character may be so far maintained that the patient shall fall down, but he shall quickly get up again and attack those about him with the utmost violence. An instance of this is recorded by Trousseau, who remarks that if there had been no witnesses of the commencement of the attack, the patient would have been the subject of a criminal prosecution, since he not only assaulted the passers-by in the street, but repeatedly insulted and spat in the faces of the soldiers who held him. In other cases the epileptic stupor is succeeded by maniacal delirium, during which suicide may be committed, or even murder. Still more frequently a fit is followed by loss of memory, incoherence of ideas, and perversion of intellect, which may last some days. It is not surprising, therefore, that when the paroxysms recur very frequently and at short intervals, the mental powers should become permanently impaired. Patients who have been subject to the disease for some time commonly acquire a peculiar dull, heavy aspect, and this, with the circumstance that they almost always have widely dilated pupils, often enables one to tell at the first glance what is the matter with them. They are apt to be exceedingly irritable in temper, or morose, gloomy, and desponding. Sometimes the mental state is worse immediately before the epileptic attacks than at any other time; when a fit occurs, it seems to afford temporary relief to the brain, and the patient afterward feels lighter and more cheerful than for a long period before.

Impairment of the intellect is by no means confined to patients who have already suffered for a long time from epilepsy. In children it often happens that a series of fits, continued for a few successive hours, produces a permanent state of imbecility, or even of mania. A considerable proportion of those who are admitted into asylums for idiots are children who were, in reality, born with full powers of intelligence, and learned to talk as soon as others; but, having been attacked by epilepsy when, perhaps, four or five years old, they have since lost all sense and intellect, have become dirty in their habits, passionate or violent in temper, and unable to recognize their parents. Such cases are frequently brought to the out-patient departments of the London hospitals.

In adults, on the other hand, I believe that permanent impairment of intelligence occurs only when the disease has been of some standing. And it is indisputable that some individuals retain their full vigor of mind after having been liable to fits for years. Julius Cæsar, Mahomet, and the first Napoleon are commonly referred to as illustrations of the fact that the repeated occurrence of epileptic attacks does not necessarily injure the intellectual powers, but in each of these cases the fits were only occasional; indeed, the proof that they really occurred is far from being complete. Dr. Reynolds has recently endeavored to combat what he terms the "prevalent belief" that mental deterioration is necessarily associated with epilepsy, and he states that in rather more than one-third of all the cases which he examined there was average intellectual power, but his investigations, of course, apply only to the state of the several patients when he saw them. One cannot infer from them that the intelligence remained perfect during the whole of the patients' lives. And the records of every lunatic asylum afford abundant illustrations that dementia ultimately shows itself in many persons who had been epileptic for a long time before the mind gave away. According to Dr. Reynolds, the later the age at which the fits commence the greater the probability that the intellect should suffer. The severity of the individual attacks has less influence in bringing about such a result than the frequency of their recurrence. Esquirol long ago pointed out that dementia more often occurs in persons who are liable to the *petit mal* than in those who have attacks of the *haut mal*.

Pathology.—With regard to the pathology of epilepsy much uncertainty of opinion still prevails. So soon as an attempt was made to distinguish the functions of different parts of the nervous centres, it was suggested that in a fit there was a torpor of the brain, associated with excitement of the spinal cord. But the regular order in which the phenomena of the paroxysms succeed one another is evidently inexplicable under such a simple hypothesis. And with the advance of physiology the theory arose that the disease might have its seat in some particular part of the brain, which would be the starting point of the attacks, and which might fairly be termed the "epileptic centre." I remember that Sir William Gull used to speak of epilepsy as a "function;" and by this I understood him to have meant that the orderly development of the various symptoms which constitute the seizure must depend upon structural nervous arrangements, like those involved in the more complicated physiological actions. Van der Kolk suggested that the medulla oblongata was the seat of epilepsy, and recent writers, including Reynolds and Nothnagel, have expressed a similar opinion, only including within the area of disturbance more or less of the pons Varolii, or of the cervical part of the spinal cord.

I think, however, that we must either confine within very narrow limits the supposed "epileptic centre," or else include in it the entire length of the spinal cord. We cannot imagine that spasmodic movements of the eyes or of the face bear to such centre any closer relation than do those of the upper or even of the lower limbs. And, if we once give up the idea of fixing the seat of the disease in a definite spot within the medulla oblongata, a little consideration will lead us to include in the affected area the mass of the cerebral hemispheres. There is no other way in which we can so simply explain the facts that consciousness is suspended and that delirium and excitement are often present. I may mention Dr. Wilks as one writer who has always strongly expressed the opinion that epilepsy is an affection of the whole of the brain.

The views entertained by Dr. Hughlings Jackson are widely different. This observer has studied with the greatest care those epileptiform and other convulsions which are caused by local diseases, such as tumors of the surface of the brain. He has laid great stress on the fact that whereas a "destroying lesion," affecting a particular convolution, is incapable of causing paralysis, a "discharging lesion" of the same part gives rise to convulsions, which may implicate the opposite face, arm, and leg in a definite order. His way of accounting for this is to suppose that in particular convolutions movements are "represented," which involve the action of many different muscles.

In 1873, Dr. Ferrier performed a series of experiments on the lower animals with the express object of throwing light on Dr. Jackson's theories of epilepsy. Fritsch and Hitzig had shown in 1870 that, instead of the surface of the brain being insensible to the galvanic current, its application to some of the anterior convolutions gave rise to definite muscular movements; and they had laid down the seat of cortical centres for the muscles of the neck, of the face, and of the upper and the lower limbs—at least, so far as dogs were concerned. Dr. Ferrier's observations were made on cats and rabbits also; and, still more recently, on monkeys. His results do not altogether accord with those of the earlier experimenters. (Compare p. 551.) Still, his observations on different animals present so complete a correspondence in their broad results that one may fairly place a general reliance on his conclusions.

More than one writer has endeavored to throw discredit on these investigations, on the ground that the galvanic stimulation may have passed downward to the basal ganglia, and so have caused the movements observed.

But it seems to me that the general conclusions at which these observers have arrived have not been invalidated, and that we may fairly regard as established the physiological doctrine that certain convolutions of the hemispheres contain a series of motor centres in which definite parts of the body and limbs are represented (see p. 551).

Apart from these experimental results, it would seem, from clinical observations, that, when convulsions are set up by local disease of the surface of the brain, the fact that certain parts are especially implicated in the spasms may sometimes enable us to indicate the seat of the lesion. Thus, in his earliest paper, Dr. Ferrier quoted two cases of Dr. Jackson's, in each of which fits occurred limited to the right arm, while in each of them the disease was discovered after death to be situated in the hinder part of the uppermost frontal convolution of the left hemisphere. In the second case, in which innumerable fits of the same character occurred, Dr. Jackson had said beforehand what would be the seat of the lesion. I may, however, add that I recently made an autopsy in a case in which a small glioma was situated in precisely the same spot, on the right hemisphere, and in which it had been noted that the fits usually commenced in the left foot.

It is quite another question whether any similar conclusion is warranted, when there is no other evidence of local disease of the brain. Dr. Burdon Sanderson has found by experiment that stimulation of the medullary fibres between the convolutions and the corpus striatum produces effects exactly resembling those of stimulation of the various convolutions themselves; and there seems no reason to doubt that equally definite results might be produced by morbid processes starting in different parts of the basal ganglion itself, even if it should be practically impossible to exhibit them experimentally.

It must also be borne in mind that the spasmodic movements produced by disease—such as are observed in a convulsive attack of whatever kind—are very different in character from the slow and orderly actions to which galvanic stimulation of the brain gave rise in those of Dr. Ferrier's experiments, from which alone conclusions as to the functions of particular convolutions could be drawn. It is true that spasmodic contractions, and even complete epileptiform fits, were often observed; but these received further explanation, as will presently appear. The cases to which I now refer are those in which we read, for example, of a cat raising the shoulder and adducting the forepaw, exactly as if it was striking a ball; of a rabbit munching with its lips and jaws; and the like. But in a fit the affected part is violently jerked backward and forward, in a way altogether different from what occurs in its natural movements. I think that this would of itself suggest the view that convulsive movements do not depend merely upon the "discharge" of the cortical centres, but are the results of impressions transmitted downward from them to the corpus striatum, or even, perhaps, to ganglia situated still lower in the cerebro-spinal axis.

But the case is far stronger, when, instead of localized spasms, a complete epileptic paroxysm occurs, in which all parts of the body are convulsed, and consciousness is for the time suspended. I confess that I am at a loss to understand how Dr. Jackson can regard this as the result of the mere discharge of one or more cortical centres.

Dr. Ferrier has discussed this question very fully in the fourth volume of the "*West Riding Lunatic Asylum Reports*," and in his treatise on "The Functions of the Brain." His theory is that in the cortex of the brain there are individual centres for each separate muscular action involved in the epileptic convulsion, that they are related to each other in a constant and definite order, and that the attack is due to the discharge of these centres in a tolerably uniform manner. Much, he says, depends on the

primary source of the irritation; but a little further on he adds that from whatever part of the hemisphere this proceeds, whether from a motor centre, or from the sensory areas toward the back of the brain, the order most commonly observed is that the centres discharge from before backward, beginning with those of the head and eyes, and ending with those of the leg. He goes on to say that epileptic convulsions can be produced with as great readiness by application of the irritation to the sensory areas as to the motor centres themselves; and, again, that it is doubtful whether consciousness becomes lost when the motor centres of the brain are alone implicated.

It appears to me that Dr. Jackson's views, in their most special form, are by these statements of Dr. Ferrier deprived of all the support which they might seem to have gained from the experimental facts adduced by him. And Dr. Ferrier's own hypothesis seems to me far less likely to be correct than the older opinion that, even when irritation of the surface of the brain causes an epileptic fit, the actual motor impulses start from the basal ganglia or from centres still lower, in the pons, the bulb, or the cord. Surely, the orderly development of the phenomena of the paroxysms which he points out, and on which I have already laid stress, is much more easily explained in this way.

Again, it is well known that there are cases in which epileptic fits are set up by irritation of spinal nerves, by diseased teeth, or (as in Brown-Séquard's experiments) by pulling the hairs on the face of a guinea pig, of which the spinal cord has previously been injured. All such cases are obviously much more easily explained on the view that the parts which are the seat of "discharge" in the attacks are the lower centres, than on the theory that they are the highest centres of all, situated in the convolutions.

It seems to me that a further argument on the same side may be found in the analogy of another paroxysmal neurosis. I have already pointed out that a tumor in the brain, at a distance from what one can suppose to be the seat of migraine, may excite repeated attacks of it. One certainly cannot imagine that these are due to "discharge" of the part which is immediately affected by the tumor; they must be due to an influence transmitted downward to the thalamus or to other sensory ganglia.

At the present time a view is widely prevalent, according to which all but the initial phenomena of an epileptic attack result from vaso-motor disturbance. It is supposed that the cerebral arteries undergo spasmodic contraction, and that the conseqnent anæmia of the brain causes the patient to fall down insensible. It is well known that the face commonly turns pale at the commencement of a seizure. There is ophthalmoscopic evidence that the retina, which derives its blood supply directly from the internal carotid artery, also becomes anæmic. Dr. Jackson and Dr. Charles Aldridge have proved that the optic disc is pale or even white, and that the arteries which traverse it are much diminished in size. It is to be noted, however, that on the single occasion on which the ophthalmoscope has been used before the clonic convulsions ceased, the disc was at first pink, so as to be undistinguishable from the surrounding choroid; and that it only afterward became white, and then slowly.

But I believe that the most substantial support of the notion that the brain is essentially anæmic during an attack of epilepsy has been afforded by the well-known essay of Kussmaul and Tenner. It had before been known that both in animals and in man loss of blood was followed by convulsions. These observers showed that the like result could be brought about by ligature or compression of the four great arteries supplying the encephalon. In reality, however, their experiments only proved that a deficient supply of blood to the brain might be one cause of attacks of an epileptiform

kind. They themselves pointed out that ligature of the trachea, rendering the arterial blood rapidly venous, had the same effect; and in either case they referred the convulsions to sudden interruption of the nutrition of the brain. Their theory evidently does not in any way confirm the more modern opinion that when an epileptic fit has commenced in the medulla oblongata, the patient becomes unconscious as the result of some reflected influence upon the cerebral arteries, rendering the brain anæmic.

For I think that, after all, the feature of epilepsy most difficult of explanation is the tendency of the fits to recur at more or less definite intervals. And no way of accounting for this seems to be so satisfactory as that which refers it to a gradual accumulation of energy in the nervous centres, which is dissipated during the attacks. As van der Kolk points out, it often happens that after a severe fit an epileptic patient remains free longer than usual; but if he should have only a slight attack, he soon afterward has another, perhaps on the following day. Again, in some cases each fit is preceded by a gradually increasing irritability of temper and restlessness, which disappear or are notably diminished after it has taken place. Other patients, as Trousseau remarks, become gay, loquacious, and excited for some hours before an attack; and yet others complain of failure of memory, of torpor, and of physical and mental prostration. Nothnagel mentions the case of a lady, generally a light sleeper, who always knew that she was about to have a paroxysm, when she happened to sleep more heavily and longer than usual; nevertheless, she would wake up feeling quite well, and would not be attacked until later on in the day.

We are thus brought, in regard to epilepsy, to the same point which we reached in discussing the nature of migraine; that it is essentially a paroxysmal neurosis, recurring at more or less regular intervals. And, as in the case of migraine, it appears to me that in all probability the alterations in the blood supply to the brain, which undoubtedly occur during the epileptic attack, are accidental concomitants rather than essential to the development of any of its symptoms. This view is not inconsistent with the fact that the inhalation of nitrite of amyl is sometimes of service in the treatment of the disease; for when there is an aura the attack can sometimes be overcome by a decided impression on the part to which the aura is referred, and the inhalation may fairly be supposed to act in a similar way, and to cut short the paroxysm by arresting one of its phenomena. Moreover, venesection is sometimes useful, and, so far as I know, under the same circumstances as those in which the nitrite does good. Yet they must produce contrary effects.

To sum up, then, I would adopt the language of some modern writers, and say that epilepsy is "dependent upon an unstable condition of the nerve tissue in some portion of the nervous system, permitting occasional discharges." This, in reality, is not stating more than that the disease is a "nerve storm." Just as in migraine, teichopsia may be followed in succession by numbness in the fingers, by headache, by vomiting, by sleep, so in epilepsy tonic spasms give place to clonic convulsions, and these, again, to stupor or coma. To me it appears more satisfactory to refer this sequence of phenomena to the gradual extension of some morbid condition from one part of the nervous centres to another, than to ascribe it to modifications in the blood supply.

This account of the pathology of epilepsy can hardly be regarded as complete without some reference to the remarkable experiments of Brown-Séquard, who found that in guinea pigs and some other animals section of the spinal cord, or even of one or both sciatic nerves, was followed, after some weeks, by well-marked epileptiform fits, which returned again and again, and could at any time be excited by slight irritation of the

cheek (as, for instance, by gently pulling the whisker) or of the side of the neck. The exact bearing of these interesting observations, however, seems even now to be doubtful, so far as concerns their applicability to the disease with which we are acquainted in the human subject. I do not think that they afford any support to the idea that the epileptic aura really starts from a distal part of the body and travels along sensory nerves, setting up the paroxysm when it reaches the encephalon. There is every reason to believe that the aura is itself part of the attack, and due to a change in some region of the brain which has close connections with the roots of certain cutaneous nerves; the disturbance is accordingly referred to the peripheral distribution of these nerves.

Ætiology.—In passing on to consider the causes of epilepsy, one has, in the first place, to mention influences inherited from parents or other relations. Dr. Reynolds made inquiries which led him to the conclusion that in 12 per cent. of his epileptic patients the same disease had occurred in other members of their families. But *hereditary predisposition* really plays a still more important part than this. In a much larger number of cases, other affections of the nervous system could be traced in persons derived from the same stock as the patient. Sometimes it was one of the other paroxysmal neuroses, such as migraine; sometimes insanity, hysteria, hypochondriasis, or mere nervousness. Thus in a family in whom nervous disorders prevail, one child may be epileptic, another insane or idiotic, another hysterical, and so on. Dr. Reynolds says that in rather less than one-third of his cases of epilepsy he obtained a history of some nervous disease as having occurred at least in one near relative. Drunken habits in parents are also believed to be often concerned in inducing epilepsy in their offspring; but then the inclination to intemperance is itself often a sign of the neurotic tendency. Some writers have supposed that a scrofulous or tuberculous diathesis, rickets, or general malnutrition may be concerned in the causation of epilepsy; but this is rendered very doubtful by the exceeding prevalence of such conditions. The intermarriage of blood relations does not seem to have any tendency to cause this disease in the offspring.

Epilepsy appears to affect males and females in about equal proportions. The *time of life* at which it is most apt to begin is between the tenth and the twentieth years. Dr. Reynolds found that in 106 out of 172 cases the first fit occurred between these limits of age, and in by far the larger number it was within the still narrower period of from thirteen to seventeen years. The more marked the inherited predisposition, the earlier is the average age at which the disease develops itself, and it commences in girls sooner than in boys. Most of these facts seem to point clearly to the conclusion that the development and commencing activity of the sexual organs are in some way concerned in the causation of the disease. It has been said that in women the time at which the menses cease to appear is again apt to be attended with the development of epilepsy, but Nothnagel says that this is not the case. However, according to Dr. Reynolds, the period between twenty-five and thirty-five years of age is one at which there is a comparative immunity from first attacks; and that they often occur in persons about forty. Exceptional instances are recorded in which epilepsy has begun at an advanced age; one, for example, by Trousseau, which began at about sixty-nine.

In other cases, instead of the predisposing cause of epilepsy being an inherited tendency it is an acquired condition.

Prolonged anxiety of mind, grief and destitution have been supposed to lead to it. Habits of *intemperance, sexual excesses*, and the practice of masturbation are believed to be more directly concerned in bringing it about. Indeed, both a first fit and the succeeding ones have in some

individuals occurred only when they were actually intoxicated, and in others only during coitus. With regard to masturbation, all modern writers speak very guardedly, so far as concerns the development of this particular disease; although there is no doubt that the genital organs and the corresponding nervous centres of youths still immature are not infrequently kept by it in a state of habitual excitement, which has a most depressing effect upon the health, and is capable of causing a variety of nervous symptoms, some of them of a serious kind. (In the present state of society it is, perhaps, impossible for any one to say whether there is a real difference between the two sexes, save that in boys the sexual instinct awakens of its own accord, instead of remaining latent, as it often does in girls, until the period of marriage.) If this is the case, it is infinitely to be regretted, from every point of view. And nothing can be more injurious to the minds of the young than the habit of concentrating their thoughts and conversation upon sexual topics, which really ought not to concern them at all, as upon matters of engrossing interest and importance. What I have already said will suffice to show that I do not believe in the existence of any unfelt irritation starting from the genital organs of modest young women, and giving rise to epilepsy or any similar disease. And I regard as an abomination the operation of clitoridectomy, which was some years ago practiced, upon a theory of that kind.

We have seen that in a very large proportion of cases of epilepsy no definite predisposing cause can be discovered. And even where there is a marked inherited tendency, our knowledge of other hereditary complaints would lead us to expect that in the majority of cases the first attack, and perhaps even the subsequent ones, would be immediately traceable to some definite *exciting cause*. Experience, however, hardly confirms such an anticipation. There is, indeed, much difficulty in eliciting the real facts in many instances. On the one hand, the parents of children affected with epilepsy are much disposed to conceal a family tendency to that or any other serious neurosis; and patients themselves are apt to be unacquainted with the real state of health of relatives older than themselves. And, on the other hand, there is a powerful tendency to attribute the disease to any accidental circumstance which can by possibility be brought into relation with the first attack. Thus, Trousseau says that when a fright was assigned as the cause of epilepsy he often found, on inquiry, that it had really occurred months or even years before the fits began, or that it was of too trifling a character to account for the development of the complaint. He himself, however, relates a case in which it appeared clear that the original cause of epilepsy was the terror caused by the sight of a quarrel between two men, one of whom was wounded and fell down dead. In that instance the first attack, one of the *petit mal*, occurred within a few days, and subsequently the *haut mal* developed itself.

Now, according to Dr. Reynolds, it is only in one out of every eight cases of epilepsy that the inquiry as to the ætiology of the disease fails altogether, by neither any predisposing nor any exciting cause being discovered. And since an inherited tendency (which is the great predisposing cause) could be traced in but one-third of the cases, there must evidently be a large number in which a supposed exciting cause, such as fright, is the only one that can be detected. We have, then, a very important point to determine, namely, why the first fit should be followed by others. Now, it is to be observed that in some cases the disease has been consequent upon attacks which are commonly regarded as purely accidental in their origin. Thus Dr. Hughlings Jackson mentions a case in which a convulsive fit at the onset of scarlet fever proved to be the forerunner of habitual epilepsy.

Again, it is said that the simulation of the disease by impostors has

ended in their becoming really subject to it; and, if true, this is a still stronger fact in the same direction. Brown-Séquard found that the guinea pigs in which he artificially set up epilepsy transmitted it to their offspring.

On the other hand, some very remarkable cases have occurred which show that even when epileptiform fits have occurred at intervals for a very long period, as the result of irritation of the nervous centres by some cause acting on a distant part, the liability to their recurrence may cease entirely when the cause in question is removed. Thus, Mr. Tomes relates the case of a farm laborer suffering from epilepsy, who had the usual remedies administered to him in the Middlesex Hospital for six weeks without effect. His mouth was then examined, and the molar *teeth* of the lower jaw were found to be decayed, the fangs of some of them alone remaining. Although he complained of no pain, they were removed, and they were found to be enlarged and bulbous, from exostosis. During the eighteen months that followed he had not a single fit, although for many weeks before the operation he had had two or three daily. Another case, recorded by Dr. Ramskill, is that of a boy who for eighteen months had had epileptic fits, and in whom it was noticed that before the fits he used to rub his left cheek, on account of an indefinite uneasiness, not amounting to pain. On examination a molar tooth considerably decayed was found; this was removed, and from that time the boy did not have another fit, although he remained under observation for four months. It is to be said, however, that during that period belladonna was administered. But the most extraordinary case of all is one related by Trousseau, of a young clerk, who for several years had been subject to monthly attacks of epilepsy; remedies had been tried in vain at the Hôtel Dieu, when Dr. Foville suggested the extraction of some carious teeth which ached constantly. The suggestion was acted on, and from that day the fits disappeared.

Trousseau also relates the case of a man, aged forty, who on several occasions, at very short intervals, was seized with violent epileptic attacks. Dr. Monnier found that he had been passing fragments of *tænia*, and gave him large doses of castor oil; a whole tapeworm came away, and from that time the convulsive fits ceased.

Again, an *injury* to the head may be the starting point of habitual epilepsy. Nothnagel gives the case of a boy who, when eight years old, fell from a height of twelve feet, upon his head, upon a hard floor. He was stunned for a quarter of an hour, and ten minutes after recovering consciousness he had a characteristic epileptiform fit. There was a little scalp wound, which healed in a few days. After six weeks he had a second attack, and from that time they recurred at periods which became shorter until he had them at intervals of from four to twelve days. He was twenty-one years old at the time when Nothnagel wrote, and his intellect and memory were already somewhat impaired. A slight scar remained, but this was not painful nor adherent. Nothnagel seems not to have thought that surgical interference would have done any good; and perhaps he was right. But in the "*Lancet*" for 1873 two cases will be found recorded, in each of which a piece of the skull was removed by the trephine, on account of epileptic fits following an injury to the head. Both of them occurred at Guy's Hospital, the one under Mr. Cooper Forster, the other under Mr. Bryant. The former patient had had a blow on the head four months before his admission; it left a slight swelling, from which a little pus exuded when it was incised. His first fit occurred the day before he came into the hospital. But a week later he was having four or five fits every hour, and his temperature was 103°. The operation was then performed, and the piece of bone which was removed was very dense, three-eighths of an inch thick in one

place, and rough on the outer surface. A fortnight afterward he could walk the length of the ward, and he never had another fit—at least, until the time when the report of the case ceases, which was two months from the date of the trephining. In the other case the accident had occurred five years before, and attacks of the *petit mal* had occurred at intervals of about a week during the whole period. The cicatrix was still tender, and occasionally painful. Medicinal treatment having been tried without any good result, the trephine was applied and a piece of bone, which was much thickened, removed. The fits at once became less frequent and seemed to occur only when he was depressed from want of food in his miserable home, or when he was exhausted by diarrhœa. The very same medicines which he had before taken with no benefit were resumed. After a time he became able to earn his living, and when the case was reported (sixteen months later) a confident hope was expressed that he would remain free from attacks for the future.

Relation to Other Convulsive Fits.—One point in favor of a real distinction between epilepsy and eclampsia (in the meaning given to that term at p. 695) is the observation of Dr. Tyler Smith that puerperal convulsions are by no means of very frequent occurrence in women who are habitually subject to epileptic fits. But it is clear, from the foregoing paragraphs, that there may in practice be great difficulty in applying the distinction in question. And another instance of the same difficulty is afforded by infantile convulsions. On the one hand, it is said that persons subject to epilepsy in adult life are often found, on inquiry, to have had fits in early childhood. Nothnagel expressly states that this is often the case with children born of epileptic parents, and with those who, in later years, themselves become epileptic. But, as Dr. Jackson points out, there are at present no facts to show what is the proportion of those who having had infantile convulsions afterward escape epilepsy. It can hardly be doubted that they form the immense majority. The fits of infants were formerly attributed simply to irritation of the nervous centres, from teething or disorder of the alimentary canal. But the more closely such supposed causes are inquired into, the less clear does their relation to the convulsive attacks appear to be. The tendency of modern observation is rather to associate infantile convulsions with rickets, just as is the case with laryngismus stridulus. And whatever part in their causation one may suppose to be taken by external sources of irritation, it is certain that another very important part is taken by inherited or acquired conditions of the nervous centres, disposing them to convulsive discharge. It may well be that in infancy the brain, being imperfectly organized, yields to influences which in after life it successfully resists. One would commit a serious error if one were to say that the occurrence of fits in childhood involved danger of epilepsy in adult life. And yet it seems to me clear that no absolute line of distinction between them can be drawn; nor, in general, between habitually recurrent epilepsy and the various forms of eclampsia, or of epileptiform fits due to external irritation.

But I think that the difficulty is, in a great measure, avoided if we regard all these affections as members of the large group of paroxysmal neuroses. In the case of migraine, also, we have variations in the gravity and clinical significance of the attacks, according to the time of life at which they occur.

Anatomy.—The only morbid changes which are to be found in the brain in epileptic patients must be regarded as *effects*, and not as *causes*, of the fits. Perhaps the most important are dilatations of the capillary blood vessels in the medulla oblongata. These were first described by van der Kolk, who maintained that there was a definite relation between their seat and the symptoms observed in the paroxysms; the nucleus of the hypoglossal nerve and the

cordus olivare presenting dilated vessels when the tongue had habitually been bitten; whereas in cases in which the tongue escaped he found them chiefly in the nucleus of the vagus. He also described an albuminous exudation into the medulla, causing at first induration of its substance, but afterward fatty degeneration and softening.

The skull is often exceedingly thick and dense in those who have long suffered from epilepsy; and the membranes may be opaque and the brain generally indurated, but these changes seem to bear a closer relation to the impairment of intellect, which is so generally present in cases of long standing, than to the mere paroxysmal affection.

Diagnosis.—This involves several different questions, some of which have already been dealt with, or will be considered in other parts of this work. Thus one may have to distinguish an attack of the *petit mal* from one of cardiac syncope, and an attack of the *grand mal* from one of hysteria.

But when a person is in a fit, which, if really epileptic, would belong to the *grand mal*, there is often a preliminary matter to be taken into consideration, namely, whether it is possible that he is feigning or malingering with the object of exciting sympathy, or, perhaps, of gaining admission into a hospital or of getting discharged from military service. Now, an impostor is likely to choose for his fits a frequented spot, where he is sure to be seen. When he throws himself upon the ground it is in such a way as not to hurt himself; he may even put out his hands to break his fall. He probably overacts his part, crying out many times instead of only once, throwing his limbs violently about, and making the attack last much longer than a genuine paroxysm of epilepsy. There is, of course, no pallor of his face at the commencement; his skin, instead of being cold, is warm and covered with sweat; his pulse, even if quickened, does not become irregular; his pupils are not dilated, still less are they insensible to light. He does not keep his eyes wide open, showing the eyeballs distorted to one side; generally he prefers to shut them, perhaps separating the lids a little from time to time, so as to watch the effect on the bystanders. If an attempt should be made to raise the upper eyelid he resists it, and instead of his conjunctiva being insensible, the slightest contact excites forcible contraction of the orbicularis muscle. On the other hand, he may readily allow the thumbs to be drawn away from the palms, and afterward close them again. But, according to Dr. Marc, in a person really attacked by epilepsy the thumbs require force to unbind them; but when once extended they remain so until the end of the attack, or at least until a fresh convulsion begins. Again, in a simulated fit the tongue is seldom or never bitten; there is no foam about the mouth, unless, indeed, this can be imitated by means of a piece of soap placed within the cheek. The muscular strength of an impostor is that of an ordinary individual; but in an epileptic attack it is said to be much augmented, so that a delicate lad may require four or five strong men to hold him. If a little snuff be blown into the nostrils of a malingerer he cannot help sneezing; in a real fit no such effect is produced. Another test is the absence of sensibility to pain. A method commonly adopted by policemen and others is to press the thumb nail forcibly beneath that of the person supposed to be in a fit; or some melted sealing wax may be dropped upon the skin. I have already mentioned that there is reason to believe that repeated attempts to feign epilepsy have sometimes ended in the development of a genuine attack. An educated medical man may, of course, succeed in imitating a fit better than one who has no professional knowledge. Thus Trousseau relates that Esquirol, who did not believe that an attack could be simulated so as to deceive, was once talking this very matter over with him and with Calmeil, at the asylum

at Charenton, when the latter fell on the floor in violent convulsions. Esquirol examined him for a moment, and then said, "Poor fellow, he is epileptic!" upon which Calmeil got up and asked him whether he still retained his opinion.

But assuming the *epileptiform* character of our patient's attacks to be established, one has still to consider whether they really belong to *epilepsy*. This question is often settled by the clinical history; the case may, perhaps, be of many years' standing, and the fits may have gradually developed from the *petit mal* into the *grand mal*. But one must bear in mind that attacks may recur at intervals for a great length of time, and present all the characters of true epilepsy, and yet not belong to that disease in a strict sense of the term. I have already alluded to Trousseau's case of the clerk who had been liable to epileptiform seizures, for several years, at monthly intervals, and in whom they ceased when some carious teeth were removed. The same writer relates the case of a lady, aged seventy-one, who for thirty-one years had been subject to attacks recurring with daily increasing frequency, so that she at length had as many as twenty-one in the twenty-four hours. Her forehead and nose presented characteristic signs of former syphilitic mischief; and the administration of mercury and iodide of potassium greatly checked the return of the fits; from the time when it was commenced she had only one of them.

Generally, however, the question of syphilis need hardly be entertained unless the fits are comparatively of recent origin; and the same may be said of Bright's disease, lead poisoning, chronic alcoholism, and the various organic affections of the nervous centres. The diagnosis between these diseases and true epilepsy must depend mainly upon the presence or absence of the other symptoms which severally characterize them, and upon the condition of the patient between the attacks.

Treatment.—In the treatment of epilepsy two things have to be considered: 1, the management of the attacks themselves; and 2, that of the intervening periods, with a view to prevent their recurrence.

So far as concerns the actual paroxysm, the most important point is to ascertain whether the patient has any warning of its approach. If there should be a distinct aura, starting, perhaps, from the hand or foot, one can often, by compressing the limb above, arrest the fit after it has commenced. Some years ago I had under my care, in the Evelina Hospital, a girl who constantly wore round her wrist a piece of cord; this was pulled tight as soon as she felt the sensation which indicated that she was about to have an attack, and not a single one developed itself during several weeks; after a time bromide of potassium was administered, and the aura then ceased to recur. Many instances of a similar kind have been placed on record by different observers. Dr. Bazire mentions the case of a woman whose fits were always preceded by spasmodic closure of the left hand; by forcibly extending the fingers, and keeping them open, an impending attack could be warded off. A patient of Dr. Reynolds had jerking of the left leg, which was drawn up behind him when his attack began; it was arrested by extension of the muscles. Another plan, which has been recommended, consists in cauterizing the surface from which the aura proceeds.

Even when an epileptic fit is not ushered in by any symptoms beyond pallor of the countenance and tonic spasms, it would appear that the prompt inhalation of nitrite of amyl is sometimes capable of arresting it. Dr. Crichton Browne has related some instances of this. One is that of a man who started up suddenly in bed, with his eyes fixed and his head turned to one side; these symptoms were known to indicate the approach of an attack; but the nitrite was administered, and the patient at once fell back on his pillow in a half-fainting state, but without the slightest agitation of the

muscles. In another case, a fit had actually begun in the ordinary way, with rigid stretching of the hands by the side, and turning up of the eyeballs, when the nitrite was held to the mouth and nostrils, and in twenty seconds complete recovery had taken place.

Even when an epileptic attack has fully developed itself, there is evidence that it may sometimes be cut short by compression of the carotid artery in the neck. This procedure must be supposed to diminish to some extent the blood supply to the brain. It was first suggested by Dr. Parry, of Bath, toward the end of the last century. He relates a case in which it proved successful. A man who had been liable to epilepsy for two years was one day beginning to have a fit: his eyes were assuming a vacant stare, and convulsions were beginning about his throat, when Dr. Parry made strong pressure over the right carotid artery; upon this the convulsions ceased, and the attack proceeded no further. He instructed the patient how to compress the vessel, and the latter afterward assured him that when he had sufficient warning he was often able to prevent the epileptic paroxysms. At Guy's Hospital I have often seen this practice adopted by Mr. Stocker, and sometimes with striking results. I think, however, that it has been especially useful in cases in which there was a strong hysterical element; and I have never been able to satisfy myself that the success which has now and then attended it has really been due to arrest of the flow of blood through the carotid artery. The plan which Mr. Stocker used to adopt was to press both thumbs into the neck, one on each side, toward the spine; in doing so he doubtless compressed many other parts besides the carotid arteries, and the pain which he must have caused may well be supposed to have been concerned in the rapid restoration of the patient to consciousness, at least when the case was of an hysterical character.

When one is called to a patient in an epileptic seizure, one must see that his clothes are loosened, especially about his neck, and one must take care to prevent him injuring himself. It is sometimes well to endeavor to keep the tongue from being bitten by putting a piece of india rubber between the teeth, but this involves the risk of its falling back into the throat and causing suffocation. I may remark, incidentally, that epileptics who are liable to attacks in the night should be very careful to remove false teeth from the mouth before going to bed, lest they should become impacted in the pharynx during a paroxysm.

In the *status epilepticus* it would appear that the best remedy is the inhalation of the nitrite of amyl. Dr. Crichton Browne has recorded ten cases in which he employed it, and eight of them terminated in recovery. The effects of the remedy were of the most striking character. For instance, a man, aged thirty, from May 6th to the 10th had from twelve to sixteen fits a day; on the 11th he was in a most critical condition; he lay on his back, breathing stertorously, with livid, purple features, and streaming with perspiration; the pulse was 140, the temperature 103°. It seemed useless to make trial of the nitrite, but, as a forlorn hope, he was made to inhale five drops every hour. His breathing at once became less labored, and he had only three more fits that day, and on the 12th there were gleams of consciousness; the pulse and temperature fell; on the 14th he could answer questions, and by the 17th the fits ceased, and he passed into his usual state of health, and was able to take part in domestic work.

Dr. Browne adds that he has found no other plan of treatment of nearly the same value in the status epilepticus as inhalation of the nitrite of amyl; but he speaks of several measures as having occasionally been useful. The withdrawal of a few ounces of blood has sometimes suddenly restored to consciousness patients who were in a state of profound coma.

Some years ago a very striking instance of the same kind occurred at Guy's, in the practice of Dr. Wilks.

For the *prevention* of epileptic fits in those who are liable to them—in other words, for the cure of the disease, epilepsy—one remedy appears to surpass all others in efficacy—this is the *bromide of potassium*. Sir Charles Locock, in 1857, was the first to recommend it, and he spoke of it as being especially serviceable in women in whom the attacks coincided with the menstrual periods. But subsequent observations have shown that there is no such limitation of its curative power. It is given in doses of from ten to thirty grains three times daily, and it must be continued for some months, or even for two or three years. Its effect is sometimes to free the patient forever from the liability to recurrence of the attacks. In other cases it suspends them for a time, or diminishes to a marked extent their frequency and their severity, but when, after a time, its administration is suspended they become as bad as before. In yet other cases it does some good for a time, but seems to lose its power, although the patient may go on taking it without interruption. Lastly, in a very few instances it appears to be altogether useless. No explanation has yet been found for the varying effects of bromide of potassium in different cases. Dr. Duckworth Williams has stated that it is comparatively ineffectual when the seizures occur only during the night, but according to Dr. Reynolds this is by no means universally the case.

My own experience would lead me to believe that most persons can take twenty grains of bromide of potassium three times daily without suffering any ill effects from its administration. But when it is given in doses twice as large, it causes in some patients very striking symptoms after about ten days or a fortnight, and the condition so produced has been called *Bromism*. According to Dr. Bazire (Syd. Soc. Translation of "Trousseau," vol. i, p. 100) it is characterized by headache, apathy, impairment of the special senses and of common sensations, loss of sexual appetite and vigor, enfeeblement of muscular power in the limbs, tremor of the hands, and impaired action of the heart. A more definite symptom than any of these, however, is anæsthesia of the velum palati, uvula, and pharynx, which parts may be tickled without the production of any efforts of deglutition. And another remarkable effect is the production of a cutaneous eruption. This commonly resembles acne more or less closely; it consists of pustules surrounding hair follicles, but these are arranged in patches or groups, and dry up into large scabs, beneath which the skin becomes red and thickened, and which may remain for a long time adherent. One case of this kind is depicted on the forty-third plate of the Sydenham Society's "Atlas." In that instance the scalp and the extensor surfaces of the limbs were especially affected by the eruption, but the face and the legs are commonly said to be the principal seats of it.

One is not infrequently consulted by a patient who has just had a first epileptiform attack, and one is then generally altogether unable to say whether there is any liability to its repetition. If it were essential that the influence of medicine in such a case should be absolutely ascertained, one would have no alternative but to wait for a sufficient length of time to enable the rate of recurrence to be determined. This, however, would be injurious to the patient, since each fit that is allowed to occur probably increases the tendency of the nervous centres to convulsive paroxysms. I think it is one's clear duty to prescribe the bromide without any delay. The probability of its usefulness may fairly be inferred from its ascertained efficacy in so large a proportion of the cases in which its value can be fairly tested. If one were to lay the matter in all its bearings before the patient himself, he would certainly wish to take the medicine regularly for a considerable length of

time, even though, in the probable event of his remaining free from all further attacks, it must always remain doubtful whether or not there was a real necessity for such treatment.

But sometimes the bromide of potassium fails to check the recurrence of attacks of epilepsy, although given in the largest doses and with the utmost perseverance. One must then have recourse to some other remedy, and I think that the best is *belladonna*. This was especially recommended by Bretonneau and by Trousseau. At first a quarter of a grain of the extract should be given twice or three times a day; and the dose should be gradually increased. At one time I had a patient who took two grains three times daily for a great length of time, with marked benefit. Trousseau insists on the necessity that this medicine should be continued for a long period, if its value is to be fully tested; a year, he says, is sometimes scarcely sufficient for the discovery of its influence; and if in the second year there should be some improvement, it may be worth while for the patient to go on taking it for three or even four years. He speaks of it as completely curing the disease in some very rare cases; but Dr. Reynolds says that he has never known it do more than diminish the frequency of the seizures.

The salts of *zinc* are useful in some cases of epilepsy. Dr. Wilks speaks of having had under his observation more than one case in which the patient was always better when under their influence. Dr. Reynolds speaks favorably of the oxide, but has seen no good result from the sulphate. The nitrate and the oxide of silver have been recommended, and I believe that they have sometimes been of service. But one must not forget that if either of these medicines should be taken continuously for a length of time there is a risk of the skin becoming permanently stained of a bluish-black color. It is believed that these preparations may be administered with safety, if the course is not allowed to be continued for more than six weeks.

In some cases the introduction of a *seton* at the nape of the neck has led to the suspension of epileptic fits, at least for the time. About this Dr. Wilks speaks decisively. The application of ice to the spine, as recommended by Dr. Chapman, has been tried in numerous cases by Dr. Reynolds, who reports that it did no good whatever.

The food of patients suffering under epilepsy should be digestible, and should be taken with regularity. Dr. Wilks speaks of having seen cases in which reducing the quantity of meat has been followed by a decline in the number and severity of the fits; but in other instances a generous diet has been advantageous. Some definite experiments in regard to the influence of animal food have been made at the West Riding Asylum by Dr. Merson, who kept a number of patients for a month on a diet including much meat, and then for the same period on one in which there was no meat, or *vice versâ;* there was no very marked difference in the number of fits, but several of the patients were much more dull and stupid and languid when taking animal food than they were when kept on a diet mainly farinaceous in character.

Both the mind and the body of epileptic patients should be kept in exercise, short of fatigue. The limbs should never be allowed to get cold, particularly at night. Dr. Reynolds says that in many cases nocturnal seizures have been prevented by a simple mechanical contrivance placed under the upper half of the mattress to keep the head and shoulders well raised.

Paroxysmal Vertigo.—Another affection which may occur paroxysmally is vertigo or giddiness. The expression "paroxysmal vertigo," indeed, is not

altogether free from ambiguity; for the analogous one, "epileptic vertigo," is commonly used as a synonym for that less severe form of epilepsy which is also known as the *petit mal*. The latter, however, is not always, nor even usually, attended with any giddiness, and it is distinct from the complaint of which I am now speaking. Two forms of this are recognized by systematic writers. In one the patient feels as though he himself were made to turn round and round, or were against his will impelled forward, or backward, or to one side. In the other he fancies that objects are revolving round him. In each case he remains perfectly conscious, and all that occurs is duly registered in his memory. If the same thing should have happened to him before, he may be perfectly well aware that his sensations are devoid of foundation; yet by the strongest effort of his will he may be incapable of freeing himself from them. But in a first attack he may be completely deceived. I was told by a patient, who happened to be a railway official, that on a particular occasion, when he was traveling, one side of the carriage suddenly seemed to rise four or five feet and to throw him into the opposite corner. Having never experienced a similar sensation, he was under the belief that there was a serious accident; but in reality he had not moved from his seat. Dr. Ramskill relates that a patient of his, who was attacked while in the street, felt the pavement uneven, with alternate depressions and elevations over which he seemed to be obliged to lift his feet. At the same time the shop windows seemed to him to be moving forward, and the passers-by to be racing after one another. He also felt giddy in himself; and as a matter of fact the two forms of vertigo to which I have above referred cannot be regarded as distinct affections. In most cases both of them are experienced, either at the same time or in succession. The gait is unsteady or reeling; the patient feels afraid of running against other people or surrounding objects; he catches hold of some support; he may even lose his balance and fall to the ground. Sometimes the act of closing the eyes removes the sensation of vertigo completely for the time. Nausea very commonly accompanies the attacks, and even vomiting.

Paroxysmal vertigo is often connected with impairment of the sense of hearing, the patient being more or less completely deaf on one or both sides, and generally experiencing sensations of buzzing or singing in the ears. In 1861, Ménière recorded in the "*Gazette Médicale*" some remarkable instances of this kind; and of late much attention has been drawn to such cases under the name of Ménière's disease; but I am not myself prepared to accept the prevalent interpretation of their pathology.

In the first place, it is certain that affections of the middle, and even of the external, ear may give rise to attacks of giddiness, faintness, sickness, etc. For example, in the "*Archives of Ophthalmology and Otology*" for 1871, both Knapp, of New York, and Bremner, of Zürich, mention cases of aural catarrh, in which such symptoms showed themselves; and Toynbee many years ago asserted that cerumen accumulated in the external meatus might by its pressure on the membrana tympani produce similar effects.

But in the great majority of cases all the more accessible parts of the organs of hearing are free from disease. Generally speaking, if the deafness is of one ear, a tuning fork is not heard on that side, even when placed upon the teeth or upon the top of the head. It is inferred that the seat of mischief must be the internal ear.

At this point some very interesting physiological observations appear to find their application. Many years ago, Flourens discovered that in pigeons and rabbits section of the semicircular canals causes strange disturbances of equilibrium. And recently Crum Brown and others have shown good reasons for the belief that the function of these structures is to furnish the

impressions which form the principal basis of our knowledge as to the relation between our movements and those of surrounding objects. They have even shown what are the several disturbances of equilibrium which irritation or destruction of each canal may be expected to produce. And accordingly it has been proposed to employ the name "labyrinthine vertigo" as synonymous with Ménière's disease. Charcot observed a case in which the lesion was chiefly in the left ear, and in which the direction of reeling was principally forward, but sometimes backward, while occasionally there was a sense of rotation on a vertical axis, always from left to right. This last would, according to recent writers, be due to irritation of the left horizontal ampulla, while movements forward and backward would respectively answer to irritation of the posterior and superior canals. Destruction of the same parts would, however, produce precisely the converse effects, and thus there is no difficulty in accounting for the fact that some patients have shown a tendency to reel toward the side on which they were deaf. In either case the actual movements are supposed to be the reflex results of the impressions conveyed to the co-ordinating centre from the various canals, which under normal conditions balance one another, but which no longer do so when some of the canals are diseased or injured.

Even when morbid changes in the meatus or tympanum are obviously present, the writers whose views I am endeavoring to expound suppose that the direct cause of vertigo is disorder of the labyrinth. As they suggest, pressure upon the fenestra ovalis can easily be conceived to cause increased tension in the semicircular canals. Thus they regard all instances of "auditory vertigo" as alike examples of Ménière's disease.

But it would be a great mistake to suppose that Ménière himself merely wished to draw attention to the fact that vertigo was apt to occur in those who suffered from deafness or from some disease of the organ of hearing. What was really new in his paper was that he endeavored to show that sudden apoplectiform symptoms (including, at least, a transient loss of consciousness) might occur in a person previously healthy, and be followed for the first time by deafness, and that the cause of such attacks might be an affection of the internal ear. He relates several cases of patients who fell down insensible, and who, when they recovered, were found to be deaf; and a similar instance has been recently recorded by Knapp.

The only one of Ménière's cases in which a post-mortem examination was made is the tenth and last of his series. A young woman, while menstruating, undertook a night journey outside a coach. She suddenly became completely deaf, and was admitted into Chomel's wards. The principal symptoms were constant vertigo and vomiting. She died on the fifth day. At the autopsy no disease could be discovered in the nervous centres; but the semicircular canals in each ear contained a reddish plastic substance. I cannot say that this observation commends itself very powerfully to my own mind. Cases in which an autopsy fails to reveal a satisfactory explanation for cerebral symptoms that had been present during life are, after all, not very rare; and it seems rash to assume that the state of the labyrinths was the real cause of the fatal illness in Chomel's patient. Moreover, as Bremner points out, even if full value were allowed to the case in question, one could hardly take it as demonstrating the nature of those other cases in which cerebral symptoms come on suddenly and rapidly pass off. In these it has been supposed by some writers, including Knapp, that hemorrhage takes place into the semicircular canals. They do not seem to have found any difficulty in the fact that the blood must be effused on both sides at or about the same time, since the deafness often comes on simultaneously in the two ears. But this appears to me to render the explanation very improbable. It is true, that hemorrhage into both retinæ occurs in

cases of Bright's disease; but surely not so as to cause sudden and total blindness.*

A different view has been suggested by Dr. Wilks; namely, that when there is no affection of the meatus or tympanum, the deafness and the cerebral symptoms are both in some cases due to changes in the nervous centres. It is evident that such an explanation is particularly applicable to cases of Ménière's disease in the stricter sense of that term; the sudden loss of hearing in both ears may fairly be attributed to an affection of the auditory centre; and the giddiness to a similar affection of the centre for equilibrium, which is probably adjacent, since its most important afferent nerves are those which come from the semicircular canals. It seems to me that whatever peculiarities in the direction of the vertiginous tendencies may be observed, such as have been supposed to depend upon affections of particular ampullæ, they can all be referred to corresponding changes in the centre, for in this the functions of each canal must necessarily be fully represented. The analogy of the other paroxysmal neuroses seems to support very powerfully Dr. Wilks's view. We have seen that impairment of sight is a frequent symptom of migraine, and that it is certainly due to an affection of the brain and not of the eyes. Indeed, "cloudiness before the eyes" and "obscuration of the visual field" are mentioned as having been present with the vertigo in some of Ménière's and Knapp's cases; and it may be that in these instances the attacks presented a combination of the two neuroses, the nerve storm spreading beyond its usual limits and encroaching upon the area concerned in migraine. So also it seems to me that, in the "apoplectiform" cases, such as Ménière described, the simplest way of accounting for the loss of consciousness is to suppose that the disturbance diffused itself over the hemispheres, as I believe that it does in epilepsy.

Another strong argument in favor of Dr. Wilks's view is afforded by the fact that (as I have often had occasion to observe) bromide of potassium may remove both the giddiness and the loss of hearing at the same time; and Mr. Hinton recorded under the name of Ménière's disease a case in which paroxysmal vertigo and sickness had been associated with only transient deafness, and in which all these symptoms together were brought back by the administration of quinine, after having been removed by treatment of a different kind.

Knapp has observed that in certain cases the impairment of hearing is particularly marked for certain musical tones, those of the middle octaves being distinctly perceived, while those of the lower, and still more those of the higher, octaves are heard very imperfectly. He suggests that this is a proof that the seat of the affection is in the labyrinth; but I fail to see the force of the argument. Much more weight, however, must be allowed to an observation of Charcot's, that some patients experience sensations of vertigo and buzzing in the ears, only so long as the deafness is partial, losing these symptoms as soon as it becomes complete. But even if we should have to admit that in these instances the internal ear is really the part primarily affected, it would by no means follow that the same thing is true of the "apoplectiform" cases, nor that the vertigo is anything but a neurosis. It is one of the advantages of the theory which I advocate, that it admits of the production of the same symptoms in many different ways, the symptoms themselves being, nevertheless, always the result of one particular kind of nervous disturbance. Just as migraine may be excited by a variety of causes, so may paroxysmal vertigo.

Indeed, the analogy between these two neuroses must now be carried a

* I once saw retinal hemorrhage in the course of chronic Bright's disease cause sudden and total blindness; but in that patient the other retina had been long affected without his knowledge.—ED.

step further. I have discussed in detail the question whether migraine is ever due to disorder of the digestive organs; and I came to the conclusion that such disorder certainly plays a part in its causation. Now writers describe *vertigo a stomacho læso* as distinct from other varieties of giddiness. I think, indeed, that the stomach is not really so often concerned in the production of vertigo as the liver, the most potent of all morbid states of the abdominal organs being the condition which, following Dr. Murchison, I would call lithæmia. This, however, is a matter on which I cannot enter. What I am now concerned with is the question whether the giddiness due to disorder of the chylopoietic viscera is different in kind from that which depends upon other conditions, such as deafness. Dr. Wilks has made the remark that the vertigo caused by derangement of the liver, occurs chiefly when the patient stoops or lays his head upon the pillow, and ceases when he stands upright. But it will presently appear that this distinction cannot be upheld; and my own belief is that in many cases there is nothing in the character of the nervous symptoms themselves, nor in the circumstances under which they arise, to show that they depend upon one rather than another of the various causes to which they might possibly be attributed.

In some cases, the ingestion of food which disagrees with the patient leads so quickly to swimming in the head that the connection could not be overlooked. Dr. Murchison speaks of a medical friend of his, who has long suffered from gout, and who, whenever he drinks a cup of tea or a glass of champagne, is seized with sudden giddiness. His head feels empty and neighboring objects seem to whirl about him; he would fall did he not lay hold of something to support him. After a few seconds or minutes the attack passes off. In other patients, as Dr. Murchison remarks, the vertigo lasts longer. Dr. Ramskill relates the case of a merchant who was one day quietly walking in the city from one office to another, when he was seized with giddiness, so that he reeled, and had to lay hold of a post which was near at hand. In a few hours, after a free evacuation of the bowels, he became better, but he felt weak and shaken, and complained of a heavy, diffused headache. About three hours before the attack he had eaten hastily, and with imperfect mastication, a breakfast of which sausages and Devonshire cream formed a part; and to this the vertigo was ascribed, no doubt with justice. Yet, during the following month, the same patient had five similar attacks, not one of which could be traced to any such cause, he having in the meantime become very particular as to his diet. Dr. Ramskill even goes so far as to say that in "stomach vertigo" it is the exception for one to be able to trace any positive signs of stomach disorder. The proof is that the complaint is cured by treatment directed to the regulation of the digestive organs. Thus, a medical friend of Dr. Murchison's, who had never had gout, and in whose case the only recorded indication of lithæmia was that his urine was often loaded with lithates, was seized with dimness of sight every night while writing. He took iron and quinine and other tonics, but without any benefit. He was advised to give up practice for a time, and try the effect of a change of air; but while he was making up his mind to so serious a step, he took a few grains of blue pill, whereupon his symptoms at once disappeared. So, again, Boerhaave's commentator is quoted by Trousseau as relating the case of a man who, during two years, was always seized with vertiginous symptoms whenever he attempted to stand up. In vain had the ablest practitioners attempted to cure him. Quite suddenly he had an attack of gout, of which disease he had before shown no indication, and from that time the giddiness ceased.

To complete the chain of evidence which proves that vertigo is one of the paroxysmal neuroses, I must next point out that it may replace other

members of this group of affections. Giddiness is occasionally present during the paroxysms of migraine; and Dr. Liveing refers to two cases, in each of which an attack of intense vertigo, of short duration, appeared several times to replace the ordinary sick headaches. The connection of vertigo with epilepsy is, perhaps, closer still. As I have already mentioned, giddiness is one of the symptoms of many cases in which fits take the form of the *petit mal;* and that affection is very often only the precursor of the *haut mal.*

Still, there are cases in which this neurosis remains unchanged in type for many years. Dr. Ramskill speaks of such under the name of "essential" vertigo. He even states that he has met with two instances in which the complaint appeared to be transmitted by direct inheritance. One of his patients suffering from vertigo had a father living, and then aged seventy-one, who had himself been subject to it for thirty-five years; he also had asthma. Another patient of Dr. Ramskill's complained for three years of giddiness, for which no cause could be discovered, and which resisted all kinds of treatment. It is true that in that case the giddiness after a time became almost continuous; and I suppose that *persistent* vertigo is more commonly due to anæmia from disease of the arteries of the brain than to any other causes. But it seems probable that in exceptional instances any one of the paroxysmal neuroses may cease to present intervals, or to occur in distinct attacks, when the patient has been subject to it for a considerable length of time.

For example, a case is recorded by Charcot ("*Progrès Méd.*, ii), of a woman, aged fifty-one, who had for six years suffered from a continuous vertigo, which did not intermit even at night, and which was so severe that she could not walk, nor even stand, and that the slightest movement of her head made her clutch at surrounding objects for support. In her the complaint had begun at least twenty-six years before; for a long time it was purely paroxysmal, and the attacks were comparatively slight. She had disease of the tympanum on each side.

The *treatment* of vertigo is not different from that of other paroxysmal neuroses. Bromide of potassium has always appeared to me of more service than any other medicine, and, indeed, I think its value is manifested even more strikingly than in epilepsy itself. I believe that the aural surgeons use chloride of ammonium for those cases which are associated with deafness. A careful inquiry must be made for symptoms of dyspepsia or of lithæmia, and if any such are present the appropriate remedies must be employed. Dr. Ramskill, indeed, recommends that alkalies and vegetable bitters should be used in all cases, on the chance of their being of service; and I have often found it a good plan to give the bromide with a few grains of carbonate of soda, and with equal parts of the infusions of rhubarb and calumba. Charcot's patient was cured in from two to three months by quinine, in a dose of fifteen grains daily.

Paroxysmal Insanity.—I have now to describe a paroxysmal neurosis which is, perhaps, more interesting than any other member of the group—that in which the attacks take the form of a transitory mania or of some form of mental disorder.

Of this I could not possibly give a more striking instance than one recorded by Dr. Maclaren in the "*Medical Times and Gazette*" for 1876. The patient was a slight, gentle-looking lady, aged forty-three, with winning manners and a soft, quiet voice. She was characterized by exalted religious feelings and an excessive and morbid sensitiveness. She would be reading her Bible and talking gently to her attendant when suddenly, without a moment's warning, she would throw the book out of the window and make a rush to

run her head into the fire; or she would, perhaps, turn on the attendant and try to strangle her. She would then struggle on, keeping all the time perfectly silent or uttering only an occasional word of Scripture until she was exhausted, or until by a kind of awakening she became restored to her former condition. Sometimes in the attacks she would expose her person. During the intervals she had no recollection whatever of what she had done; at the most she could slightly recall the impulse which led her to attempt some act of violence.

One peculiarity of the paroxysms in Dr. Maclaren's case was that in each of them the patient made special efforts to get at one particular picture, which at other times used to excite in her no emotion whatever. Thus it seemed that she followed out in successive attacks trains of thought of which she had no knowledge during the intervals; and her condition might so far be fairly termed one of "dual consciousness." Falret has laid stress on the fact that in this form of mania all the paroxysms in the same patient may be alike in every detail. He has pointed out the suddenness with which they come on and the extreme violence with which they are attended. He also remarks that it is characteristic of this affection that repeated blows are struck by the patient, and several wounds inflicted or several persons injured.

By Falret the affection now under consideration was described under the name of epileptic insanity—*furor epilepticus*. But the former expression is ambiguous, since it has been used as a general name for all mental disorders which may accompany or follow epileptic attacks. Moreover, it would appear that a liability to that disease is not always present. Dr. Maclaren's patient had never had epilepsy; she was, however, a member of a family in which there prevailed marked tendencies to neurotic affections. It, therefore, seems to me that paroxysmal insanity is a better name; but there is no question that the affection is one which bears a very close relation to epilepsy. As a rule, patients who have seizures of this kind have before suffered from attacks of the *petit mal*, if not of the *haut mal*. Dr. Hughlings Jackson, indeed, has recently expressed the opinion that a transitory epileptic paroxysm really occurs each time before the mental symptoms develop themselves. In other words, he thinks that the affection is identical with that form of mania which I have described as sometimes following the *haut mal*. This view, however, rests upon a highly theoretical basis. He supposes that the mental disorder is essentially automatic, and that a necessary condition for its occurrence is the removal of the control of the highest centres, which are exhausted by having discharged themselves during the fit. My reasons for not accepting this conclusion are those which I have adduced at p. 702, *et seq.*

Even when paroxysmal insanity occurs in those who are really subject to frequent epileptic fits, one may be unable to ascertain the fact at the time when the case comes under observation. It often happens that a patient in this condition is brought to a public hospital by the police, and, as Dr. Jackson remarks, it may be impossible for one to say whether he is an epileptic, or drunk, or suffering from meningeal hemorrhage. Dr. Jackson records a very interesting case of a woman who was brought to the London Hospital in a maniacal state, with a deep gash in the left arm, by which the elbow joint was opened, and all the soft parts in front of it were cut through. She accused different people of having inflicted this wound upon her, but it was ascertained beyond doubt that she had done it herself. She had been cutting bread for her children's tea, when she suddenly sent them all out of the room. A short time afterward she was found lying in a pool of blood on the floor. On the following day she was rational, but furious mania returned several times during the next week. On inquiry it was

ascertained that she had been subject to epilepsy, both the *petit mal* and the *haut mal*.

The patient doubtless received the suggestion of the idea which led to the infliction of the wound on her arm from the circumstance that she had a knife in her hand at the time. Thus the action was in one sense automatic. But it is to be noted that she sent her children out of the room, which at first sight looks like intention.

The sufferer from this form of insanity is, in fact, peculiarly liable to find himself in the hands of the police for some offence committed during the paroxysm; and although to any skilled medical observer it may be perfectly evident that he is irresponsible, there may be much difficulty in making this clear to unprofessional minds. Dr. Jackson has done a great deal toward the elucidation of such cases by studying other instances in which the acts performed are not criminal, but absurd; they may be characterized by precisely the same degree of adaptation of means to ends, and yet they leave no trace on the memory. Thus one of his patients had been talking about supper, and it had been agreed that he and his wife should have some cold fowl, and her sister some cocoa. Soon afterward he felt the symptoms of an attack and sat down on a chair against the wall of the kitchen, where he happened to be. He remembered nothing further, but his sister-in-law came in and found him standing by the table mixing cocoa in a dirty gallipot, which was half filled with bread and milk for the cat, and stirring the mixture with a mustard spoon, which he could not have obtained except by going to the cupboard for the purpose. If the object fetched had been a knife, and if he had inflicted some injury with it, this purposive action would have seemed a strong point against him.

Dr. Jackson, however, himself admits that the form taken by a man's mental automatism during the paroxysm depends very much on his natural disposition. A savage and suspicious man would, when his highest faculties were temporarily in abeyance, be more likely to kill some one than to mix cocoa for his sister-in-law. Indeed, the actions performed during a state of unconsciousness are sometimes exactly those which would have been performed if the individual had been in full possession of his faculties. The patient last referred to had on another occasion ordered dinner at an eating house, when his mental condition underwent a change, and he remembered nothing more until he found himself at his desk in the office, feeling rather confused. He had to go to the place and ask whether he had had his dinner, and he then found that he had eaten it and paid for it, and that neither landlady nor waiter had noticed any peculiarity about him. In other cases the patient goes through actions of which the form is correct, but of which the details are absurd. Thus another of Dr. Jackson's patients while in an omnibus was one day observed to blow his nose upon a piece of paper, and when he got out he gave the conductor £2 10*s*. instead of the usual coppers.

So, also, when some foolish act is committed, it is quite an accident whether the nature of the impulse which gave rise to it can be guessed from circumstances that have previously occurred. The same physician had another patient, who one day found the extinguisher of a candle in his waistcoat pocket. It had been known that for some years he invariably looked at his watch after each fit, and the supposition is that he must, in an attack, have mistaken the extinguisher for the watch.

Falret long ago remarked that an impulsive tendency to wander about is characteristic of this form of insanity. Trousseau gives some instances. A gentleman was attending a literary meeting at the Hôtel de Ville, when he quickly ran out and walked for some minutes on the quays, avoiding with success both the carriages and the passers-by. When he

recovered, he found that he had neither great-coat nor hat; he returned to the room and resumed, with a perfectly lucid mind, the historical discussion in which he had before taken an active part. The same gentleman, being a magistrate, was presiding at a provincial tribunal, when he suddenly got up, muttered a few unintelligible words, and went into another room. He was followed by the usher, who saw him make water in one corner, after which in a few minutes he returned to his seat. Another patient of Trousseau's, an architect, used often to have an attack while walking across a narrow plank at a height from the ground. He never met with any accident, although he would run rapidly over the scaffoldings, shrieking out his own name in a loud, abrupt voice. A quarter of a minute afterward he would resume his occupation and give orders to his workmen, without any recollection of what had occurred to him.

In attacks of this kind the state of the patient is often said to be one of somnambulism, and the expression "*day*mare" has been invented for them by way of contrast with that of *night*mare. And I believe that true somnambulism (at least, when it is not a manifestation of hysteria) has every claim to be regarded as one of the members of the group of paroxysmal neuroses which we are now considering. The same is the case, too, with the "night terrors" to which some children are liable, who start up an hour or two after going to sleep, screaming with fright, and unable for some minutes to recognize the parents or nurse. That these various attacks should occur only at night is, after all, nothing more than occurs in many cases of epilepsy itself.

HYSTERIA.

HYSTERIA A REAL AND DISTINCT DISEASE—MENTAL AND MORAL PERVERSIONS—HYSTERICAL AFFECTIONS OF SENSATION AND OF MOVEMENT—CONTRACTURES — GLOBUS — FLATULENCE — THE HYSTERICAL FIT — CATALEPSY — SLEEP WALKING — ECSTASY — CONVULSIONS — MANIA — HYSTERO-EPILEPSY—ANOREXIA AND MARASMUS—OTHER HYSTERICAL SYMPTOMS—PATHOLOGY—PROPHYLAXIS—TREATMENT.

Hysterical affections, disorders of function, or pains, or perverted sensations of various kinds, are seated in the most diverse organs of the body. In books on surgery many other complaints are mentioned under the same name, occupying the joints, the mammary glands, or different regions of the trunk or limbs. Such a use of the term "hysterical" implies that there is one disease which is common to all the affections in question; and that disease—*hysteria*—I have now to describe, so far as its general characters are concerned, but without stating all the details of particular manifestations of it, which will be given elsewhere.

But, first, I must justify myself in speaking of hysteria as a distinct and definite malady. For there are some who refuse to admit its claim to be so regarded. In this they find a partial support from two sides. On the one hand, the name derived from the Greek word ὑστερα, a womb, suggests the opinion, which these observers believe to be erroneous, that the organs of generation in the female play the principal part in the causation of the affections which are called hysterical. This, indeed, would not in itself matter, for many complaints have names for which no etymological justification exists in the present state of science. What creates the difficulty is rather the circumstance that a fundamental difference of opinion exists at the present time between physicians of different schools as to the extent to which uterine or ovarian complaints are to be regarded as the cause of the disease now under consideration. Those who see little or no connection between them have been tempted not only to do away with the name, but to ignore the clinical association of the symptoms as manifestations of a single malady. On the other hand, it must be admitted that, more than most other diseases, hysteria is difficult to define. As a neurosis, independent of any appreciable structural change, it would naturally have its definition based either upon its causes or upon its symptoms. But the former are still the subject of discussion, and the latter are, to a peculiar extent, variable and inconstant. Moreover, the curability of most hysterical affections, and the fact that they are generally contrasted with organic diseases, which are severe in comparison with them, have created a tendency in the minds of many practitioners to apply the term vaguely to cases of the nature of which they have themselves no definite conception, but which they regard as of a trifling character and as likely to be of transient duration. And in this way much has been done to deprive the words hysteria and hysterical of all real meaning.

I think, however, that very little study at the bedside is needed to convince one that the various affections called hysterical are really the expression of

a special morbid condition, for which the name of hysteria is as convenient as any other name would be, besides having the sanction of antiquity and established usage. One finds that two or more of these affections are very commonly present in the same patient at the same time, or that a person who has suffered from any one of these is exceedingly apt to be afterward attacked by others. Then, again, there are marked peculiarities in the age, sex, disposition, and other conditions of such patients. And, above all, there is in most cases a particular mental state, which can easily be recognized apart from any other indications of the disease, and which may be regarded as its fundamental character.

Mental Condition.—The principal features of the mental state which characterizes hysteria are an exaltation of the emotional faculties, and a perversion of the will or a loss of the proper balance between it and the judgment. The patient—she is so generally of the female sex, that I may use the feminine pronoun—seems to have little or no power of controlling her own feelings. The most trifling occurrences excite her to rapture or plunge her in despair. She may conceive a violent affection for some persons, and an equally irrational aversion for others. She, perhaps, exhibits great attachment to some pet animal, while other creatures, no less harmless, excite in her repugnance and disgust. She torments all those about her with her caprice and her egotistical claims upon their attention and forbearance. Her will may seem to be altogether in abeyance; she may appear to be unable to make up her mind to the smallest effort, and may lie in bed all day, from sheer want of energy. But in some particular direction she may, perhaps, show the most obstinate determination and tenacity of purpose. She often has a great craving for sympathy, and is exceedingly anxious to have her delicacy of health recognized by those who visit her. In pursuance of this object she will apply corrosive acids to her skin, swallow needles day after day, or run them into all parts of her body, make abortive attempts to poison or drown or hang herself, or even actually commit suicide. The stoical endurance of pain which she exhibits under such circumstances is sufficient to show that a mere defect of volitional power cannot be the essential element of hysteria. Perhaps the best way of expressing the facts is to say, with Dr. Reynolds ("Syst. of Med.," vol. ii), that the will is no longer called into exercise by the judgment or reasoning faculties, but only by some one dominant idea or emotion. Thus, as Jolly observes (v. Ziemssen's "Handbuch," vol. xii), if one can, by stimulating her enthusiasm or spirit of emulation, supply a hysterical patient with a sufficient motive to undertake any good work, such as nursing, she will often carry it out with more than ordinary perseverance and energy. In regard to the will, as well as to the emotions, the peculiarity of hysteria is that there is a loss of the due balance and proportion between the several faculties.

Moral Perversion.—The moral tone of a hysterical woman is not uncommonly impaired to a serious extent, particularly in certain directions. Morbid sexual inclinations have often a strong hold upon such a patient, and she will plan and scheme to induce her medical attendant to make a vaginal examination or otherwise to gratify her perverted feelings. Sir Thomas Watson relates a case in which a young woman contrived to make a hospital surgeon believe that she had a stone in the bladder; and the imposture was not detected until she had been tied up in the position for lithotomy in an operating theatre full of students. I have met with an instance in which a patient, who had been supposed to have a hydatid in the liver, and who had before been punctured without result, allowed the operation to be repeated, and almost immediately produced a piece of the stomach of a rabbit (or some other small animal), which she declared she had vomited, and which she, no doubt, thought would be mistaken for a hydatid membrane; a few hours

later she sent for her medical man again, to remove from her vagina another piece of the same substance. Other persons have been known to drink their urine and then to bring it up again, while they pretended that none was passed in the natural way. The fact that such cases have occurred makes one hesitate to accept as authentic such cases as that which I have described on the authority of Charcot under the name of hysterical ischuria. There is often great difficulty in detecting impostures of this kind. Charcot himself relates a case of Boyer's in which a woman pretended that urine came from her umbilicus, ears, eyes, and breasts, and that she vomited fæces; and it was not until she had been placed in a straight waistcoat that little balls of fæces ready prepared for swallowing were found in her bed.

Some of the factitious affections of the skin are by no means easy of diagnosis. In the museum of Guy's Hospital there is a model of the right breast of a girl, which is reddened and has on it a number of large raised tubercles, some of them bigger than peas. The first time that patient was an inmate of the hospital the cause of the affection remained undiscovered, although it was evident that some irritant was being intentionally applied. But more than a year afterward she was again admitted, and Mr. Birkett, happening to visit the ward at an unusual hour, found a piece of lint, strewn with powdered cantharides, which she had put on the breast. Sir William Gull related a similar case in which he detected with a lens a glistening fragment of the same powder on the skin itself. Some years ago a girl was admitted into the clinical ward under my care who had scattered on her chest and breasts a number of whitish, gangrenous-looking patches, of irregular sizes and forms; these successively shriveled up into brownish scabs, which soon became detached, leaving the skin beneath reddened and rather scaly. On one finger, in the groove between the skin and the nail, there was found a bright yellow stain, which affected both structures to about the same extent, and which seemed indisputably to have been caused by a drop of nitric acid. I therefore concluded that the patches on her chest were likewise produced by the acid, although I am still unable to understand why they did not show the characteristic yellow color. The detection of the imposture was never carried further, and I heard some months afterward, from Mr. Roper, of Blackheath, that the girl (who had left the hospital) was still troubled with the complaint. It happened, strangely enough, that shortly before she came under my care I had had another patient who was affected in exactly the same way, and in whom there appeared no reason to doubt that the patches were the result of a spontaneous gangrene. She was a girl, aged eighteen, who seemed entirely devoid of self-consciousness. The museum of Guy's Hospital contains models taken from each of these patients.

Other forms in which hysterical patients exhibit their craving for notoriety and their willingness to deceive are illustrated by the cases of the Welsh fasting girl, and Louise Latour, the Bleeding Nun.

Among the particular manifestations of hysteria some are frequently met with, some are very rare. One, the "hysterical attack" or "fit" has always been regarded as especially typical of the disease; and two others, the "globus" and a flatulent distention of the alimentary canal, are, perhaps, still more common and scarcely less distinctive. One must make diligent inquiries after all these in any case the nature of which appears doubtful. It would be natural that I should describe them before the rest; but I think it will be more convenient that I should leave them until after I shall have mentioned some of those symptoms which are less often seen.

Perversions of Sensation.—Not the least curious of these are perversions of the special senses. The patient, perhaps, complains of intolerance of

light and insists on having the room kept darkened. Here, however, imagination often plays a great part. Dr. Reynolds relates the case of a woman who had for weeks been lying with her hand before her eyes, to keep out the light of a dull London sky. When he brought a candle close to her in order to examine the pupils she shuddered, knit her brows, and held both hands between it and her eyes. But as soon as her attention was distracted to the state of her front teeth, the brows became relaxed, the hands were removed, and she bore the light without inconvenience. In other cases the patient is distressed by the slightest sound and will allow no one to speak above a whisper. Yet such a person may herself exclaim in a loud voice, or make a great noise in stirring the fire, without seeming to mind it. In other cases there appears to be an actual exaltation of perception. A hysterical girl may seem, as Dr. Reynolds says, "to hear through stone walls;" but in such matters deception is often practiced, as when such a patient pretends that she can see with her eyes shut.

Jolly quotes from Amman the case of a woman who discovered the presence of some cherries in another room by their odor, and who could distinguish one individual from another with her nose; and such persons often detect by their taste the presence of a perfectly infinitesimal trace of any flavor that happens to be disagreeable to them. It is perhaps a perversion of the gustatory sense that leads girls affected with hysteria to eat cinders, sealing wax, lead pencil, and the like; they are sometimes fond of repulsive odors or flavors.

The sense of touch, again, may be unduly acute. In almost every case of hysteria there is at some part or other of the body an over-sensitiveness to painful impressions—a *hyperæsthesia*, or rather, in strict language, a *hyperalgesia*. Sometimes the patient complains that it hurts her very much to have the skin over one or more of the spinous processes pressed upon or even touched; sometimes she has extreme tenderness of the breast, or of the edge of the rib cartilages on one side.

Another frequent effect of hysteria is impairment of common sensation, "dysæsthesia" or "anæsthesia." This may either be limited to ordinary tactile impressions, or it may include those of heat and cold; in other instances it concerns only the perception of pain, and then the name of "analgesia" is sometimes applied to it. Gendrin went so far as to declare that sensation was more or less defective in every case throughout the whole course of the disease; and other writers have asserted that loss of feeling in some part of the skin is invariably left behind by hysterical fits. Their statements, indeed, are too absolute, but the symptom is undoubtedly often present, and Lasègue and Charcot are probably right in saying that it would be more frequently observed if it were carefully looked for, a person being sometimes unaware that she is unable to feel pain in some particular part, until her sensibility is tested. In many cases, however, subjective sensations of tingling, pins and needles, etc., are experienced and complained of by the patient. It is important to note that there is no opposition between hyperæsthesia and anæsthesia, such as might appear from the names. A part of the skin may be exquisitely tender, and the seat of burning pain when touched; and yet at this very part the power of transmitting definite tactile impressions may be greatly impaired.

Anæsthesia, or defective sensation, is much more frequently observed in some regions than in others; Jolly mentions the backs of the hands and feet, and the parts above the outer malleoli, as favorite seats of it. Sometimes it affects exactly one-half of the body, leaving the other free. To such cases Charcot gives the name of "hemianæsthesia." He quotes Briquet as having stated that this occurs on the left side more often than on the right side, in

the proportion of seventy cases to twenty. He says that the limitation of the parts in which there is loss of feeling is often remarkably sharp, corresponding almost precisely with the median line of the face, neck and body. The mucous membranes are affected in a similar way. Taste may be wanting in one-half of the tongue, the sense of smell may be impaired, and there may be a considerable degree of amblyopia, with limitations of the visual field for the several colors—a symptom which Galizowski calls achromatopsia. According to Jolly, reflex excitability is also wanting; sneezing cannot be induced by irritating the nose, nor retching by tickling the fauces on the affected side. Lastly, Charcot says that the side on which sensation is impaired is cold and pale, and that there may even be a difficulty in drawing blood from the skin by pricking it with a needle.

Altogether apart from hemianæsthesia, loss of feeling in the mucous membranes is common. Anstie laid considerable stress on the frequency with which it may be found, if looked for, in the back of the pharynx. According to that writer ("*Lancet*," ii, 1872), whenever a person who has not been taking a bromide can, without retching, let one pass the finger down to the epiglottis, the diagnosis of hysteria is exceedingly likely to be correct. Dr. Reynolds mentions that he has known several cases, occurring in married women who were still bearing children, in which there was absolute anæsthesia of the vulva and vagina. Impairment of sensation in the bladder is supposed to be sometimes the cause of retention of urine in hysterical patients, but in other cases it appears to be due to a prurient desire to have a catheter introduced by the medical attendent. Jolly relates the case of a patient who inflicted terrible burns on herself by taking live coals out of the fire and pressing them with both hands into her vagina, but who seemed to experience no pain whatever.

I believe that hysterical anæsthesia is never permanent. It may last for several weeks and even for some months, but sooner or later it always passes off.

Paralysis.—Another manifestation of hysteria may be paralysis, particularly in certain of its forms. Aphonia and dysphagia often result from hysterical loss of power in the laryngeal and the pharyngeal muscles respectively; and I may here add that the former is highly characteristic of the disease, its presence giving very great aid toward the diagnosis of some cases, the nature of which might otherwise have been doubtful. Not uncommonly, hysterical paralysis takes the shape of paraplegia or of hemiplegia. I have already, at p. 409, described the former affection as accurately as I am able. The latter likewise presents characters of its own. I believe that Briquet first pointed out how much more frequently it occurs on the left side than on the right. The proportion in his cases was as forty-six to fourteen. An important distinction between it and the paralysis due to disease localized in the opposite side of the brain, is that in hysterical hemiplegia the side of the face and tongue always remain entirely unaffected. This was long ago remarked by Dr. Todd, who also pointed out that the patient in attempting to walk "drags the palsied limb after her, as if it was a piece of inanimate matter, and uses no art of circumduction, nor effort of any kind to lift it from the ground; the foot sweeps the ground as she walks." Dr. Reynolds supplements this by saying that a woman suffering from hysterical hemiplegia cannot help lifting the great toe, like a healthy person, when she endeavors to put the foot forward; and, again, that a paralytic patient looks at her feet, an hysterical patient at the persons who are watching her. But I think that his point about the great toe can apply only to cases in which the impairment of power is but slight.

In some cases one limb is alone affected with hysterical paralysis; or,

much more rarely, only part of a limb. I do not remember to have seen any one muscle affected singly, with the exception of the levator palpebræ; but hysterical ptosis is not uncommon.

Hysterical paralysis may be very transitory, lasting only a few weeks or days, or even not longer than a few hours. Sometimes it afterward reappears, either in the same limbs or in others. But there are cases in which it persists for months or even years. I do not know that it is ever permanent, except in some of the cases in which rigidity sets in, and to which I shall allude in the next paragraph. It may disappear suddenly, under the influence of a sudden emotion or shock; or it may pass off very gradually. The removal of the patient to a hospital ward—away from sympathizing friends and relations—is often followed by a more or less rapid recovery. I have seen several cases under Dr. Wilks's care in which this result has been brought about rather by moral influence than by any special treatment. The patient is told that she is expected to begin to move the paralyzed limbs; day by day she is encouraged to do more and more; after a time she is taken out of bed, and dressed, and put into a chair; and before long she walks as well as ever.

Contractions.—Another and a very curious effect of hysteria is a chronic spasm or rigidity of one or more of the limbs, associated with a complete loss of power of voluntary movement. This has recently been studied with much care by Charcot, who has published in his "*Leçons*" several capital drawings illustrative of the distortions which it produces. Some English writers have since adopted into our language the French word *contracture* for such cases. The affection very often follows immediately upon a severe hysterical fit, such as I shall presently describe; and the paralysis and the rigidity then generally develop themselves simultaneously. But in some cases the latter does not come on until the former has already lasted for a considerable time. In certain cases only one leg or one arm becomes contracted; but sometimes both legs are affected, sometimes both the arm and the leg on one side, sometimes three of the limbs, or even all four. When the affection assumes a hemiplegic type, and the rigidity follows the loss of power after some interval, it might be thought that there would be a difficulty in distinguishing it from the "late rigidity" of hemiplegia due to organic disease of the brain; but in the hysterical affection the contraction comes on, not gradually, but suddenly, and as the immediate result of a fresh hysterical fit. Moreover, the positions assumed by the individual limbs are peculiar. The arm becomes semi-flexed. But, according to Charcot, the leg always falls into a state of rigid extension, the knee and ankle being straightened and the toes stretched down to the utmost extent possible, while the sole is turned inward. Thus the condition of the foot resembles that which exists in talipes equino-varus. Charcot also mentions that the adductors of the thighs are forcibly contracted. Jolly, however, relates a case in which one leg became powerfully flexed, especially at the knee joint.

The rigidity in these cases is not relaxed in the most profound sleep; nor does it undergo any variations in degree at different times or periods of the day. The spasm is by no means confined to a single set of muscles, for one is not able to force the patient's arm (which is semi-flexed) into a position of complete flexion, any more than to extend it. By making the patient inhale chloroform, however, if its action is carried far enough, one can temporarily remove the spasm, the affected parts becoming perfectly supple in all but very exceptional cases. Anæsthesia of the affected limbs is generally present. The muscles remain well nourished, and retain their electro-contractility at least for a considerable time. But when a limb has been contracted for some years without intermission, a little general wasting may

occur, and the reaction of the muscles to galvanism may be somewhat impaired. The termination of hysterical "contracture" is almost always in recovery; and in many cases this takes place quite abruptly. One of Charcot's cases is very instructive. A woman was first attacked, at the age of thirty-four, with loss of consciousness after a moral shock; she then fell into the fire and burned her face severely. After this she had several seizures, some of them hysterical in character, others rather epileptiform. Four years afterward she had a very severe fit, attended with convulsions and followed by an apoplectiform stupor with stertorous breathing; this was at once followed by left hemiplegia. Rigidity of the left limbs set in abruptly after an interval. In the following year the right limbs also became contracted; and later still the jaws were fixed, so that an œsophageal tube had to be used. Her right arm, however, became free; and Charcot maintained that recovery was still possible. One evening, six years from the commencement of the contraction of the left arm and leg, she had an attack in which she imagined herself to be about to die. She cried out, became agitated, and with her right arm pushed aside those who held her. Her strong desire to get to the window for air was resisted; she became more and more angry; and first her right leg lost its rigidity, then her left leg, and finally her left arm. She got up and walked; and in eight hours her cure was complete. A slight crackling in the joints was all that remained of the complaint Such a case has an obvious bearing upon those in which cures are attributed to miraculous agencies. They are seldom related in such a way as to enable one to speak positively as to the exact form of paralysis which had been present. But Charcot refers to an article by Littré in which detailed histories are given of certain persons who were cured at the end of the thirteenth century by pilgrimages to the tomb at St. Denis, where the relics of Louis IX had recently been deposited. Three of these were young women who had been suddenly attacked with contraction of one leg, or of the arm and leg on one side, and who also had anæsthesia. They were cured suddenly, under circumstances certainly very likely to act upon the imagination. In fact, the analogy seems to be complete. In some cases of hysterical "contracture," however, recovery never takes place. Charcot mentions several instances which he believed to be hopeless. One is that of a woman, aged fifty-five, whose legs had been contracted for sixteen years. Under chloroform her knees could still be relaxed; but her feet remained in a condition of equino-varus. This observer has made an autopsy in one case in which all four limbs had been contracted for ten years; and he discovered sclerosis of the lateral columns in nearly the whole length of the cord. The patient was a hysterical woman, and the rigidity passed off several times and returned again, before it became permanent. There seems no reason to doubt that the case originally belonged to the same class as the rest. Charcot accordingly supposes that sclerosis may come on secondarily in such cases. The points on which he would lay stress as indicating the probability of an unfavorable termination to a case of long-standing contraction of a limb are that particular groups of muscles should be wasted in an extreme degree and affected with fibrillary tremor, like what is seen in progressive muscular atrophy; that there should be a great diminution in the contractility, as tested by faradization; and that the rigidity should remain to a very considerable extent when the patient is under the influence of chloroform. On the other hand, he does not attach any importance to a little general loss of substance in the affected limbs, to a slight impairment of the reaction of the muscles for electricity, nor to the presence of spasmodic movements, which last are not uncommonly manifested in such cases, as in those of ordinary hemiplegia (see p. 508).

Globus.—Among the most common and characteristic symptoms of hysteria there is one—the so-called *globus hystericus*—of which there is a difficulty in saying whether it is merely a morbid sensation, or depends upon spasmodic contraction in the œsophagus and pharynx. The former opinion has been maintained by Eulenberg and Reynolds, the latter by Jolly. To the patient herself the globus is a sensation, most commonly a feeling as if there were a ball or lump in the throat, which she cannot swallow or get rid of in any other way. She may even put her finger into the pharynx, and make herself sick, although she knows there is not really anything that can be dislodged; or she may drink mouthful after mouthful of water to wash it down. The exact nature of the sensation varies a good deal in different persons. In some it is likened to that which would be caused by a bean sticking in the throat; others feel as if a worm or some other living animal were creeping about in its interior. With others, again, it is rather a feeling of constriction about the neck, as if the clothes were too tightly fastened. The relation of such a feeling to the state of emotional excitability which characterizes hysteria is shown by the fact that a precisely similar sensation is felt by healthy persons when suddenly seized with grief or terror. Indeed, children before a fit of crying experience a sensation of a lump in the throat, which is probably identical with the more marked form of globus. In some hysterical women the lump is not merely felt in the throat, but seems to rise up from the chest, or even from the epigastrium. Rumbling movements in the bowels are also complained of by many of these patients, and, perhaps, this is the best argument in favor of the opinion that the globus is also due to peristaltic contractions in the gullet.

Flatulence.—Another frequent symptom of hysteria is a rapid distention of the intestines with gas. The abdomen may suddenly swell up, so that the patient is obliged to take off her stays and undo all the fastenings round her waist. Jolly remarks that this form of tympanitis bears no relation to meals. In several instances he has seen it return each morning regularly and attain its maximum about midday. The gas may ultimately escape, either by the mouth or from the rectum. Jolly says that it is commonly quite free from odor; and this, he adds, explains the fact that the distention sometimes subsides without any apparent escape of wind in either direction. How such an enormous quantity of gas can be accumulated in the stomach and bowels is as yet altogether unknown. In some cases hysterical tympanitis persists for years together, the abdomen being as tense as a drum. Bamberger alludes to a case of this kind, in which the patient passed through an attack of cholera without alteration in the size of the abdomen, but in which an abundant flow of saliva, coming on spontaneously, once led to its temporary subsidence.

The Hysterical Fit.—There remains to be described that which (as I have already remarked) was for a great length of time regarded as the chief symptom of hysteria, the hysterical attack or fit. But the truth is that in the actual majority of cases no attacks occur during any part of their course; according to Briquet in three cases out of four. Moreover, when they do occur, they present infinite varieties of symptoms. Sometimes they are of so trifling a character that it is obviously unnecessary to send for medical advice. The patient, perhaps, has an unusually painful globus, and then begins to cry and sob, or falls into violent laughter. Or she may laugh and cry alternately, until she is exhausted. Such an attack, for which the vulgar name is that of "hysterics," may occur singly, or there may be several in the course of two or three days. A very constant feature of them—and, indeed, of all kinds of hysterical fits—is that when they pass off the patient voids a very large quantity of colorless urine, of low specific gravity, and, in fact, almost pure water.

But in many cases an hysterical attack is accompanied by violent movements of the body and limbs, which may be distinctly convulsive in character. Perhaps the patient screams out, and then falls into a state of opisthotonos, remaining supported for some minutes together only by the back of her head and by her heels. Or she may throw her limbs in all directions, beating them on the ground, or striking her own chest with her closed fists, tearing her hair, kicking those about her, and struggling with all her might to release herself from restraint. Or, again, she may thrust one arm high in the air; or forcibly bend her arm over the chest, and her thighs across one another; some of the fingers and toes being at the same time stretched apart to the furthest possible extent, while others are as strongly flexed. The respiration is much quickened, and it may be very noisy, but it is not actually stertorous. Her features undergo the strangest contortions and grimaces. Her eyes are generally closed. The eyelids quiver, and the eyes themselves, the pupils of which are of natural size, are often turned upward, particularly if an attempt is made to separate the lids by force. The state of the eyes thus differs altogether from that which belongs to an epileptic fit, for in it they are commonly fixed wide open, and have their pupils greatly dilated.

There appear to be all degrees of impairment of unconsciousness in hysterical attacks. At the commencement the patient almost always knows what is happening, so that she is able to place herself out of the way of injury. Not rarely she keeps on screaming during the greater part of the fit, or shouts out "fire," "thieves," or "murder," or pours forth a continuous torrent of the foulest language. Sometimes she has visual hallucinations, addressing furious invocations to imaginary persons, or fancying that she is surrounded by wild beasts or spectres. Sometimes she seems to be altogether unconscious, but even then by careful watching one may often make out that she really notices what is going on, casting occasional furtive glances from between the half-closed eyelids, or modifying her movements under the influence of remarks made by the bystanders.

Catalepsy.—In some instances a hysterical attack is attended with very remarkable symptoms, which have long been regarded as belonging to a special neurosis named catalepsy (*κατάληψις* = a seizure or arrest). Its characteristic features are that the patient, although she may be deprived of sensibility and of voluntary motion, remains fixed in whatever position she occupied at the commencement of the fit, and yet that her muscles offer no such resistance to external force as would prevent the limbs from being easily bent or extended by another person, or the body from being placed in any posture. The condition of a cataleptic patient may be compared with that of a lay figure, such as artists use; if she is sitting up her arms can be put at the most awkward angles with the trunk, and will remain without falling, at least for a time; if she is recumbent, her spine may be bent upon the pelvis so as to form an obtuse angle with the thighs, and will retain that posture. The name of *flexibilitas cerea* is sometimes given to this peculiar state of the muscles; they have been found by Rosenthal to have their electric sensibility and contractility either normal or decidedly increased. It is, however, a mistake to suppose that during the cataleptic state the muscles are capable of resisting the force of gravity for an indefinite length of time. On the contrary, the limbs, if extended, slowly fall again to the side. Dr. Chambers quotes a case in which an impostor was detected by the simple plan of attaching a weight to the extended hands. The patient (as she was supposed to be) supported it without moving, and this was taken as showing that she was not laboring under a genuine attack of catalepsy; ultimately she confessed the fraud. The eyelids of a cataleptic patient may either be widely open or shut; in the latter case, if

opened, they perhaps very slowly close again. The pupils may contract under the influence of light.

I have gone into these details with regard to the "cataleptic" paroxysm in this place, because I cannot learn that it ever occurs in a well-marked form except in persons who either are obviously hysterical or who, at least, may be strongly suspected of suffering under this disease. In speaking of the *petit mal* of epilepsy I have quoted a case in which it was said to have assumed a cataleptic character; but in that case the *flexibilitas cerea* does not seem to have been observed. I do not know of any instance in which a fully-developed catalepsy has been shown to have been a modification of epilepsy in a person of the male sex, or in a woman presenting no hysterical tendencies. The attacks seem almost always to be traceable, at least in the first instance, to a fright or to some other powerful emotion. Dr. Chambers mentions the case of a girl, a patient in St. Mary's Hospital at Paddington, who, when Covent Garden Theatre was burned down, was awakened by the flashing into the ward through the uncurtained windows; on the following morning she was attacked with catalepsy. It is noticeable that the most striking instances of catalepsy which stand recorded are to be found in the medical works of a former generation, published at a time when the modern complex conception of hysteria was not dreamed of, and yet the reports of these cases often contain clear proofs that the patients were in the highest degree hysterical. The celebrated history related by Dr. John Jebb nearly a century ago is a case in point. The subject of it was a young lady who suffered from hysterical risings in the throat and flatulence, and who was highly susceptible to every change in the weather. A very suggestive feature is that although she was prepared for Dr. Jebb's visit when he first went to see her, she was seized with the disorder as soon as his arrival was announced. "She was employed in netting, and was passing the needle through the mesh, in which position she immediately became rigid, exhibiting in a very pleasing form the figure of death-like sleep. . . . The positions of her fingers, hands, and arms were altered with difficulty, but they preserved every form of flexure they acquired, nor were the muscles of the neck exempted from this law, her head maintaining every situation in which the hand could place it, as firmly as her limbs. . . . About half an hour after, the rigidity and statue-like appearance being yet unaltered, she sang three plaintive songs in a tone of voice elegantly expressive, and with affecting modulation." The seizures sometimes lasted as long as five hours.

In other cases similar attacks have been of still longer duration. I believe that the condition of the muscles has then generally been different from the *flexibilitas cerea* of catalepsy proper; either the limbs oppose considerable resistance to all attempts to change their posture, or they are completely flaccid, falling into any position whatever under the mere influence of gravity. Such patients may most fairly be said to be in a state of trance. Writers describe this condition as sometimes lasting for days and weeks at a time. The face may then be perfectly pale, the breathing so feeble as to be imperceptible except on the closest examination, and even the heart's pulsations and pulse at the wrist discoverable with great difficulty. These are the cases which have led to the popular belief that death is sometimes only apparent, and that there may be a danger of persons being buried alive. I am not aware that any trustworthy case stands recorded in which this actually occurred, but it cannot be denied that a patient in such a condition might easily be allowed to die by careless or ignorant attendants.

Sleep-walking.—In other hysterical persons the attacks assume the form of somnambulism or of ecstasy. The former is a condition which may be compared to an acted dream. I have already referred to it (at p. 722) when speaking of

certain remarkable transformations that epilepsy and the other paroxysmal neuroses sometimes undergo; and I believe that when not allied to those diseases it is a manifestation of hysteria. The somnambulist, without seeming to wake from a state of sleep, walks all over the house and even out of doors. She balances herself without difficulty on narrow planks, and manages to avoid all obstacles in her path. She takes no notice of any one whom she may meet, there is much difficulty in rousing her, she remains for a time bewildered, and she has no recollection of what she has been doing. Marvelous stories are told of the feats which are accomplished by persons in this strange condition. But one must not forget how untruthful hysterical patients often are, how prone to take a pleasure in mystifying and deceiving those about them, and how apt to be encouraged in such a course by the admiration and wonder which their performances call forth, particularly when they are sufficiently exaggerated.

Ecstasy.—The state of ecstasy is one in which a person becomes regardless of all external circumstances and engrossed with some particular emotion or idea. She may remain motionless, with staring eyes and fixed expression, or she may repeat a few words with ceaseless monotony, or dance or spin round and round with extraordinary vigor and persistency. In the "Tarantulism" of the Middle Ages, and in the epidemics which have more recently affected religious enthusiasts in Scotland and America, men suffered as well as women. But there seems to be no doubt that underlying the attacks there was a loss of balance between the different faculties of the mind, with an exaltation of the emotional susceptibilities exactly like what we have seen to be essential to hysteria. Certain cases, in which movements of one particular kind are repeated incessantly for an extraordinary length of time, have had the special name of "malleation" invented for them. Some years ago Dr. Wilks had a girl under his care, who sat by her bedside and kept thumping at something with her fists all day long. But the most remarkable instance of this kind on record is probably that which was related by Mr. Kinder Wood ("*Med.-Chir. Trans.*," vol. vii). The patient, a young married woman, who had suffered severely with pain in one side of the face, began to be troubled with involuntary movements in the eyelids, which were opened and shut with excessive rapidity. After a time the hands were beaten rapidly upon the thighs, and the feet upon the ground. Then she became half raised from her chair and seated down again, these movements succeeding one another as quickly as they possibly could. Other modifications of her attacks occurred from time to time, until at last she took to skipping about the room, regulating her movements by a series of strokes on the furniture as she passed, or by movements of her lips, as though beating a tune. Some one thinking he recognized the air as "The Protestant Boys" began to sing it, and she suddenly turned and danced up to him, and continued dancing till she was out of breath. Then a drum and fife were procured, and she immediately danced up as close to the former as possible, and went on until she lost the step, or until the measure was changed, or was made so rapid that she could not keep up with it. It was presently found that a continued roll of the drum also had the effect of putting an end to her movements; and this instrument being used as soon as the attacks threatened to begin, their occurrence was at length prevented. The explanation which the patient gave of them was that "there was always a tune dwelling on her mind, which, at times becoming more pressing, irresistibly compelled her to commence the involuntary motions."

Still more extraordinary movements were performed by a lady who came under Dr. Abercrombie's observation, and who had suffered for two years from various nervous affections. She, while lying perfectly quiet, would suddenly with her whole body make a kind of convulsive spring, by which she

was jerked entirely out of bed. Or, if sitting or lying on the floor, she would fling herself into bed, or leap, as a fish might, on to a wardrobe fully five feet high. After a time the muscles of the back and neck became affected with a wonderful semi-rotatory movement, which sometimes went on without interruption night and day for several weeks together. If the head or neck were touched, the motion was increased to a most extraordinary degree of rapidity. She was cupped in the temples, when the affection suddenly ceased, with a general convulsive start of the whole body. But it returned again and again, and finally disappeared only when treatment was used to correct constipation and menstrual disorder from which she suffered. Some years ago there occurred in Guy's Hospital a case very similar in some respects. A little girl, aged nine, had been knocked down by a boy five days before her admission. She was insensible at the time, and seven hours afterward she had a fit. Subsequently she had nine other fits. They began by her making a low, sighing or moaning sound, after which the upper extremity became contracted, the teeth were clinched, and with a sudden bound she threw herself completely out of bed. When she recovered consciousness she had no remembrance of what had occurred. On the day of her admission she had fourteen fits, and at one time she remained insensible for two hours. A day or two later she had two fits during the visit of the physician, Dr. Owen Rees; in these she clapped her hands, and her face went through a most extraordinary series of contortions. In one attack she struck a part of her head to which a blister had been applied, whereupon she at once became conscious. After she had been in the ward six days one of the fits was commencing, when the nurse told her that she must be tied down; she immediately began to cry and recovered, saying that she would have no more fits. From that time she remained well.

The final manifestation of the disease in Dr. Abercrombie's patient—when the head revolved from side to side—has been dignified by certain systematic writers with a special name, that of "rotation." Several instances of it are related by Sir Thomas Watson as having been recorded by different observers. The most striking of them is one ("*Med.-Chir. Trans.*," vol. v) which occurred in Scotland in 1813. A girl, aged ten, who had before exhibited other nervous symptoms, was seized with a propensity to turn round on her feet like a top, with great velocity, always in one direction. This continued a month and then passed off. Afterward she began to roll over and over on her bed, moving rapidly from one end to the other, for six or seven hours every day. Having been taken into a garden, she quickly rolled along the whole length of a walk, and even when she was laid in the shallow part of a river, she began to turn round as usual, although she seemed to be on the point of being drowned. She made little or no use of her arms in performing the rotations, of which there were about sixty in a minute. At the end of another month or six weeks she began an entirely new set of movements. Lying on her back she could, by drawing her head and her heels nearly together, bend herself like a bow, then she would relax her muscles, and fall with considerable force on her seat. This she repeated ten or twelve times a minute for several hours daily. After a fresh interval, she became possessed with a propensity to stand on her head; and as soon as her feet were elevated perpendicularly in the air, to let herself fall again, her knees striking the bed first. This movement, also, she continued for fifteen hours a day, at the rate of twelve or fifteen times a minute. The native treatment of blistering, purging, and leeching was employed without result; but having been taken to Glasgow, from her home in the country, in an open gig, and brought back after three days, she was seized with diarrhœa, and she soon afterward recovered entirely.

Other Varieties.—In other cases, again, maniacal excitement with halluci-

nations and delusions complicates the hysterical attack, or even constitutes its main feature. Such symptoms may be transitory, the patient becoming perfectly rational as soon as the fit passes off, but in many instances they last for a considerable time, and then it may be very difficult, and even impossible, to distinguish the case from one of insanity. Dr. Blandford remarks that the points specially indicative of insanity are that the patient should betray strong amatory feelings, that she should pretend to be unable to speak, and that, while seeming to be violently delirious, she should nevertheless be thoroughly alive to all that is going on, and display an exercise of volition in what she does. But I believe that many very doubtful cases find their way into lunatic asylums, and the truth seems to be that no actual boundary line between hysteria and insanity exists, the one disease being very prone to pass into the other. Dr. Savage, in the twenty-first volume of the "*Guy's Hospital Reports*," says that a large number of the female insane patients admitted into Bethlem Hospital have suffered from severe hysteria at former periods in their lives, and that many cases, at first regarded as examples of hysterical insanity, end in death or dementia. Some observers have made it a sort of test for hysteria that the mental faculties should be found unenfeebled when recovery takes place, even after a considerable length of time. And thus many doubtful cases have been at last set down as hysterical because of the suddenness and completeness with which the patients have regained their senses. Dr. Savage speaks of one woman who imagined that she had the itch, and who refused to shake hands with any one; she awoke, one morning, quite free from delusion and full of wonder at her fancy; and he alludes to other patients who have gone to bed believing themselves eternally lost and have awakened full of hope and faith.

Hitherto I have made no allusion to the occurrence, in an hysterical attack, of rhythmical clonic spasms at all resembling those which are seen in an ordinary epileptic fit. But the truth is that spasms of this kind are not infrequently present, the other phenomena of the seizure being such as to leave no doubt as to its nature, the patient, for example, keeping the eyelids forcibly closed and turning the eyes upward. In 1876, a girl was under my care in Guy's Hospital, whose main symptom was a rhythmical clonic spasm recurring at intervals of a few seconds, in the neck, shoulders and arms, and who had transitory fits in which she seemed to lose consciousness, while these jerking motions became much intensified and succeeded one another with greatly increased frequency. One feature in that case was the perfectly bilateral character of the spasms, and this accords with the statements of writers to the effect that in hysteria the convulsive movements are rarely limited to one side, or even greatly more severe on one side than on the other, whereas in epilepsy each of these conditions is well known to be of frequent occurrence.

Epileptiform Hysteria.—In some cases of hysteria, however, fits occur which in all respects resemble those of epilepsy. The loss of consciousness is complete; the eyes are widely open and the pupils dilated; a bloody foam escapes from between the lips, and the tongue is bitten; the movements are clonic rhythmical spasms of the simplest kind, and repeated again and again with but little variation. Perhaps this should not surprise us, for there are few diseases of the higher nervous centres (whether functional or organic) which may not be accompanied by similar attacks, of the most typically epileptiform character. But writers on hysteria, having probably in view the necessity of laying down a line of distinction between such fits and the ordinary hysterical seizures, have created for the cases in question the special designation of "hystero-epilepsy;" and there have been many discussions as to whether they are transitional links between the two neuroses, or instances of the co-existence of both of these in the same individual. But, as I have

remarked at p. 695, the disease epilepsy is characterized not merely by peculiarities in the fits themselves, but by the fact that they recur again and again, without any cause, unless it be an inherited tendency to nervous disorder. If it is theoretically inaccurate to regard as epileptic the fits to which some patients are liable for months or years as the result of the irritation of a carious tooth, it must be equally incorrect to give such a name to the fits which occur in a woman only while she is under the influence of hysteria. And Charcot has recently pointed out that the so-called hystero-epilepsy differs altogether from true epilepsy in some important particulars. However closely the attacks may follow one another, they do not, as in the status epilepticus, entail danger to life with the development of a high bodily temperature. He relates the case of a woman who for more than two months labored under a constant repetition of epileptiform fits. Once they succeeded one another without a break from 9 A.M. to 8 P.M.; and then again from 9 P.M. onward for an equal space of time. He estimates that she had from 150 to 200 attacks on that one day alone. As he remarks, a patient suffering under epilepsy would soon have succumbed. But as to the issue of his case there was at no time the least anxiety. The woman never passed urine nor fæces involuntarily, and got out to relieve herself in the brief intervals that occurred between the attacks. Her temperature rose occasionally for a short time to 101.3°; but the mean was 98.6°. As further distinctions between hystero-epilepsy and epilepsy, Charcot alleges that the former never assumes the type of the *petit mal;* and, lastly, that, however perfectly epileptiform a fit may be, it can always be arrested by compression of the ovary, in a way that I shall presently describe.

Fatal Event.—The rule that hysterical fits are devoid of danger to the patient's life is not altogether without exceptions. Wunderlich met with a case in which a servant girl, aged nineteen, who had for eight weeks been laboring under a succession of epileptiform fits unattended with pyrexia, fell suddenly into a state of *collapse,* and died in two days, with a temperature of 109.4°. Other cases have been recorded by Meyer, in which excitement of a nympho-maniacal character, followed by cramps and spasms in various muscles, led to collapse and death after an illness of some weeks.

Again, the more chronic forms of hysteria sometimes prove fatal by *marasmus.* Dr. Wilks has related in the "*Guy's Hospital Reports*" for 1866 the case of a young lady who lost her sister rather suddenly, by heart disease, whereupon she began to experience palpitation and sickness and pain over the heart, would take no food, declared that she had her sister's complaint and should soon follow her to the grave, and did actually die, in a sort of hysterical convulsion, exactly five weeks afterward. He also gives the case of another girl who would eat nothing, but merely nibbed a biscuit and drank wine by drops. The bowels ceased to act, but masses of scybala were from time to time removed from the rectum. She became thin, and Dr. Wilks at length renounced the opinion that the complaint was merely hysterical, and thought that there must be some partial obstruction in the small intestine. But when, at the end of more than two years, she died rather suddenly, he could find nothing to account for her illness. Her body was then fatter than when he first saw her, and she had, in fact, taken rather more food toward the last.

A somewhat similar complaint has been described by Sir William Gull ("*Clin. Soc. Trans.*," vol. vii) under the name of *anorexia nervosa vel hysterica.* It is attended with extreme wasting, and is commonly supposed to be the result of some latent tubercular affection. But Sir William Gull remarks that the emaciation is actually too great for this opinion to be correct, for persons with organic disease do not usually become so thin until they are no longer able to get about, whereas it is rather characteristic of the hysterical anorexia

that those affected with it display an excessive restlessness and bodily activity. In this complaint the pulse and the respiration are slow, and the temperature is generally below the normal. Sir William Gull's patients were chiefly young women between the ages of sixteen and twenty-three. It does not appear that any of them showed definite indications of hysteria. Some of his cases terminated fatally,* but in others a complete recovery took place, the main points in the successful management of them being the avoidance of fatigue, the administration of nourishing food at very frequent intervals, and the use of warm clothing.

Ætiology.—In passing on to consider the causes of hysteria, we find ourselves face to face with a question which even now can hardly be said to be settled, although it has been discussed for centuries—I refer to the question as to the part played by affections of the generative organs in the ætiology of the disease. As I have already remarked, its very name involves etymologically the doctrine that it is the result of uterine disorder, and the ancient Greek writers—including even Plato and Aristotle—actually supposed that the womb left its place in the pelvis and wandered about the body, setting up disturbances in different parts. From their time to the present there have never been wanting theories which, according to the different standpoints afforded by the science of the age, have referred hysteria to a similar cause. The physicians who have held such views in extreme forms have, indeed, been generally professed gynæcologists rather than men acquainted with the pathology of nervous diseases. But Romberg, the great German authority on these diseases of forty years ago, defined hysteria as the reflex neurosis proceeding from sexual irritation.

By different writers different disorders of the female sexual apparatus have been made the starting point of hysteria. Some have endeavored to trace it to unsatisfied desire, citing in support of their views the frequency of its occurrence in young widows and in the wives of impotent husbands, others have referred it to masturbation or to excessive venereal indulgence, and others again to the common menstrual disorders. Addison published a clinical lecture in 1830, to show that its phenomena depended upon "uterine irritation." By Dr. Henry Bennett ulceration and induration of the cervix uteri were represented to be morbid conditions of great potency in causing such effects. More recently Dr. Graily Hewitt has assigned the principal place to flexions of the womb.

Charcot has laid great stress on the frequency with which hyperæsthesia of the ovary is present. When lecturing on this subject at the Salpêtrière he showed to his class five women (almost all the hysterical patients who were then in the hospital, each of whom had pain in, or tenderness on pressure over, one or both of the ovaries. According to him the pain is sometimes very acute, and widely diffused over the abdominal surface, with special hyperæsthesia of the skin over an area two or three inches in diameter, corresponding with adjoining parts of one iliac and of the hypogastric regions. But in other cases no spontaneous pain manifests itself, indeed, the skin is anæsthetic, and the muscles can be pinched up without the patient being hurt, but on making deep pressure toward the brim of the pelvis one can feel the ovary as an egg-shaped body, which slips beneath the fingers. Further pressure upon it gives rise, not exactly to pain, but to a peculiar sensation which she at once recognizes, having felt it many times before as the starting point of hysterical attacks; and this, passing up to the epigastrium, is followed by pain in that neighbor-

* [I once saw a case of this form of hysteria with Dr. Dabbs, of Newport. Extreme emaciation preceded death. At the autopsy there was confirmation of suspected vaginal irritation, but the organs were like those of starvation. The cord was normal to the eye, and histological examination revealed no lesion.—Ed.]

hood, and sometimes by nausea and vomiting. Next, if the pressure on the ovary be continued, the patient experiences palpitation of the heart with extreme rapidity of the pulse, a sensation of globus in the neck, a hissing noise in the ear of the same side, a feeling as if blows were being struck with a hammer on the temporal region, an impairment of vision, especially in the corresponding eye, and even a perturbation of consciousness, leading to a regular hysterical fit. Charcot also says that when there is hemianæsthesia, or paralysis, or contraction of the limbs on one side of the body, it is always the ovary on the same side which is hyperæsthetic; and that, if such symptoms are bilateral, both ovaries are affected. Lastly, he has found that when a patient is seized with a hysterical attack, even if it is epileptiform, it may be cut short by energetic compression of the ovary continued for three or four minutes; sometimes, when this is the case, the patient says that the pressure hurts her, sometimes that it does her good.

The writers whose opinions I have been quoting do not all suppose the sexual organs to be the starting point of hysteria. For example, Charcot, although he speaks of ovarian hyperæsthesia as "playing a predominant part" in the *clinical history*, nowhere speaks of it as the *cause* of the disease. There are, in fact, proofs that Romberg's definition, already quoted, is inaccurate. Thus hysteria is occasionally met with in males; according to Briquet in as many as five per cent. of all cases, and Dr. Reynolds says that in them its development seldom bears any relation to the generative functions; he has sometimes observed it in boys at puberty, but the most marked cases which he has seen have been in men from thirty-five to fifty or upward, who have been mentally overworked or greatly reduced in physical power. So, also, in women, although probably more than one-half of those who become affected with it show the earliest indications of it between the twelfth and the twentieth years, about the time when menstruation was first being established, there are yet a considerable number of girls who manifest hysterical symptoms at a much earlier period of life. No fewer than one in five of Briquet's cases occurred in girls under the age of puberty.

It is, indeed, one of the most important facts in the ætiology of hysteria that, even among children, it is far more common in girls than in boys. We have seen (at p. 655) that this is the case with chorea likewise, and in both diseases the preponderance of cases in females is probably due to the same cause, namely, their innate emotional susceptibility and their want of power to resist external influences. It has been correctly remarked that hysteria seldom, if ever, shows itself in such women as would be called "masculine," whereas the men and boys who are attacked by it are those who would be said to be of a "feminine" disposition. One influence which does much to favor the development of the disease is the want of a proper education. If the girl is fanciful and capricious, her parents yield to every whim. If she is morbidly self-conscious, she is allowed to indulge her vanity, and to nurse the belief that she is gifted and misunderstood. If she is studiously disposed, she is left to pore over books, and never gets proper exercise in the open air. If she craves for precocious excitement, she is taken to balls and parties when still young, she is permitted to sit up late at night, to spend the morning hours in a soft bed, to live on a rich and stimulating diet, and to feed her imagination with romances. The influence of example may also induce a liability to hysteria; the daughter of a hysterical mother is very likely to acquire the disease in her turn; so potent does this cause appear to be, that according to the best authors there is at present no need to suppose that hysteria is ever transmitted by descent. In comparison with the emotional excitability in a girl, her intellectual

capacity has but little influence in rendering her susceptible of hysteria. As regards bodily constitution, some hysterical women are robust, with fresh-colored cheeks and a vigorous circulation, but by far the larger number are weakly and delicate.

The real relations between hysteria and affections of the generative organs are, I think, best elucidated by the aid of the analogy afforded by some of the paroxysmal neuroses. We have seen that in many persons the attacks of migraine are constantly excited by disorders of the visual apparatus, but that in others this is not the case. We have found that vertigo is in some individuals brought on by diseases of the ear, and in some by diseases of the digestive organs, but that in others it is traceable to neither of them. So it seems to me clear that the neurosis, hysteria, cannot essentially depend upon any one set of external exciting causes. In some cases it, perhaps, develops itself spontaneously. Very often anæmia or chlorosis appears to play the most important part in its causation, and it has sometimes been observed as the direct result of the deprivation of food. In other instances—probably in the immense majority—its manifestations are directly called forth by impressions which reach the nervous centres from without. But these are by no means always derived from the generative organs. Emotional and psychical influences—a sudden alarm, the shock caused by the death of a relative, or the unexpected loss of property—may bring on hysteria in any of its forms in a person who had before seemed perfectly healthy. A similar result may follow a gunshot wound (as has been pointed out by Mitchell), or it may be brought about by a railway collision, even by one of so slight a character that no bodily injury can be detected. In some cases a spirit of imitation suffices for its production; the disease has been known to spread like an epidemic from one woman to another among the inmates of a hospital or a workshop. In other instances, as was pointed out by the late Dr. Anstie, indulgence in alcohol seems to be the main exciting cause of hysterical complaints.

Again, it often happens that several of the conditions which may bring forth manifestations of hysteria are present together in the same individual. A person who is attacked after a sudden fright may before have had her health depressed by misery and exhaustion of mind and body, or may have been laboring under religious excitement, jealousy, wounded self-love, or remorse. The liability to hysterical symptoms of all kinds is always increased at the periods of menstruation, and there is reason to believe that it is also augmented in many women at the climacteric age, although this does not appear from the statistical data which certain observers have collected.

But, no doubt, there are many cases in which disorder of the generative organs is really the main cause of the development of this neurosis, and I am quite disposed to accept Addison's teaching, to the effect that such disorder should be carefully searched for whenever it can possibly be supposed to exist. It is fortunate that we are able to investigate the state of the ovaries by pressure above the brim of the pelvis without offence to a patient's delicacy, but writers are by no means agreed as to the actual facts in regard to the frequency of morbid conditions of the sexual apparatus in hysterical women. I find Jolly already disputing the accuracy of Charcot's recent statements as to the proportion of cases in which hyperæsthesia of the ovary is present. He quotes Scanzoni and Amann as having found among their hysterical patients as many as 19 or 20 per cent. in whom the generative organs were perfectly healthy, and, as he remarks, the fact that these observers were professed gynæcologists renders it certain that their figures in regard to this point are too low. Speaking from his own impressions, he is disposed to state the proportion at more than 50 per cent. (Ziemssen's "Handbuch," xii, 2, p. 461.)

Diagnosis.—That this is often difficult must be sufficiently apparent, not only from what has been said in the present chapter, but from the repeated references to it that I have had to make when speaking of organic diseases of the nervous system. What I would in this place insist on is the importance of careful inquiry and search, in all doubtful cases, for the different positive indications of hysteria which have been described at p. 730. Anæsthesia, limited to small portions of the cutaneous surface, is probably present in many cases in which the patient is not aware of it, and its discovery may go far toward clearing up a doubtful case.

At the same time one must bear in mind that a woman may be hysterical and yet be affected with organic disease, whether of the brain or of any other part of the body. This remark is true even of cases in which all the symptoms of the former complaint are present in the most marked form, but its applicability is increased indefinitely if we extend the domain of hysteria as widely as some writers have done. Sydenham went so far as to say that the majority of women were hysterical, and since the psychical characteristics of this neurosis are, after all, only an exaggeration of those which belong to the whole female sex, it is obvious that no absolute boundary line can be drawn. Some even describe a minor degree of exaltation of susceptibility under the name of the "nervous temperament."

Again, it is important to note that there is no kind of proportion in the degree to which the different symptoms of hysteria are developed in the same patient. A woman who has hemianæsthesia, or paralysis, or some local pain may present scarcely any indications of emotional susceptibility, may be quite free from uncomfortable sensations in her throat, and may never have had the slightest approach to a hysterical attack. Yet the whole course of the case may show conclusively that the complaint is of this nature. Indeed, when one has to deal with the more marked affections, such as paralysis or contraction of a limb, of which the characters can be well defined, I think that one may be justified in making a diagnosis of hysteria, even in the absence of all corroborative evidence.

So, again, I believe that one may safely attribute to the same cause all cases of hemianæsthesia if unaccompanied by motor paralysis. And even when the loss of sensation is limited to a single limb, it would probably very rarely happen that one would be wrong in coming to the same conclusion. I have, however, often heard Sir William Gull speak of a case in which he at first made a mistake, and which he has recorded in full detail in the "*Guy's Hospital Reports*," Ser. iii, v. 4. A nurse in the hospital, aged thirty-eight, complained of numbness in the left arm. Below the elbow sensation was completely wanting; but when the skin of different parts above that joint was carefully tested with the point of a needle, there were all kinds of contradictions in her statements. She would one minute say that she could feel the needle at a certain spot and the next minute that she could not feel it. Such observations led to the conclusion that the case was one either of feigning or of hysteria. But after the anæsthesia had existed for two years, the limb became gradually paralyzed. Two years later she died from the effects of an accident, and it was then found that the spinal membranes were thickened, especially in the cervical region, and that the posterior columns of the cord and the gray matter had undergone degeneration.

Treatment.—We must, in the first place, consider what degree of success may fairly be aimed at in dealing with hysteria. We can hardly expect to modify, to any great extent, the peculiar morbid constitution which constitutes its essential feature, although moral influences may strengthen the power of self-control. A complete cure of the hysterical tendency is seldom to be looked for, at least until the patient reaches the climacteric

period. The earlier the age at which the disease began to manifest itself the more unfavorable the prognosis in this respect is said to be. It is very important that a child who displays emotional excitability which seems likely to develop into hysteria should be placed under the most favorable hygienic influences possible. Exercise in the open air, a plain, nourishing diet, limitation of the hours of study, early rising, sea bathing, the use of the shower bath, at once suggest themselves as likely to aid in keeping the disease at bay.

When a hysterical patient is anæmic or chlorotic, she should, of course, be made to take one of the preparations of iron for a considerable time. If there be any uterine disorder, it is generally advisable that the patient should employ some astringent injection for the vagina, such as the compound alum lotion which Addison recommended so highly, and which contains a drachm of sulphate of zinc and a drachm of alum in half a pint of water.

In dealing with special symptoms, again, one must never overlook the importance of moral treatment. I have already mentioned how successfully this may sometimes be employed in the management of paraplegia—an affection which, more than almost any other manifestation of hysteria, is apt to persist for months, and even years, if left to itself. So, also, one can sometimes cure hysterical aphonia by urging the patient to speak and confidently assuring her that she is able. For this affection electricity, too, is very useful. Dr. Reynolds recommends that sparks should be taken from the larynx; or that a shock should be administered from a Leyden jar; or that an interrupted current should be passed through the throat. In long-standing cases Dr. Mackenzie's instrument may be employed, by which galvanism is applied directly to the vocal cords. Each of these methods often leads to the instantaneous restoration of the voice. Dr. Reynolds speaks of a strip of emplastrum lyttæ applied around the throat as having occasionally been successful when electricity had entirely failed.

Paralysis of the limbs may be treated with advantage by faradization, and passive movements and frictions should also be diligently employed. But Dr. Reynolds says that he has found no remedy so effectual as the application of a narrow piece of blistering plaster completely round the affected parts.

Hysterical anæsthesia is said to be sometimes benefited by the application of a rather powerful faradic current by means of a dry metallic brush. Stimulating liniments may also be rubbed into the affected parts.

For rigid contraction of the limbs it is said that the application of the continuous galvanic current is sometimes useful. But I have seen more benefit result from straightening the affected joints under chloroform, and placing the limb upon a splint, than from any other plan of treatment. Sir Thomas Watson speaks of cases in which a stream of cold water directed upon the contracted part, and continued in spite of the patient's complaints of pain, led at once to the relaxation of the spasm. He also mentions an instance in which Sir Charles Clarke cured by the same method a girl who had hysterical trismus and could neither speak nor eat. He began to pour pitchers of water upon her face, but before he had emptied the second pitcher she began to scream and complain, with her mouth widely enough open.

When hysteria manifests itself by producing a chronic and long-standing contraction of a limb, the administration of medicines seems to be altogether useless. But for most of the other effects of the disease, I cannot but believe that drugs are often very useful. Most writers speak of assafœtida and valerian as owing their virtues chiefly to their disgusting taste. But many hysterical women actually relish assafœtida. Moreover, pills containing valerianate of zinc ought, on such a theory, to be almost inert,

whereas, I have several times seen them (in doses of gr. j–ij) most effectual in removing aphonia, hemianæsthesia, and hysterical hemiplegia. In 1874 a woman was under my care in the clinical ward of Guy's Hospital, who had paralysis of her left arm and leg, the latter being rigidly extended; she took large doses of assafœtida for some days without benefit, but was afterward quickly cured by the valerianate of zinc, so that she walked out of the hospital within five days from the time when she first began to take it. In other cases I have obtained equally good results from assafœtida, five grains of which were ordered to be taken every three or four hours. Musk and castoreum are also said to be useful, but I have not had occasion to use them. Dr. Reynolds has found bromide of potassium altogether ineffectual in any form of hysteria. Both he and Jolly recommend that opium or morphia should be given for the relief of pain and to procure sleep; and they rightly observe that for the former purpose the subcutaneous injection of the alkaloid is especially effective. But one must always keep before one's mind the evil results which may follow from the establishment of a morphia habit, and to which I have already referred when speaking of the treatment of neuralgia. The continued use of chloral as a hypnotic is to be objected to on precisely similar grounds. And perhaps more harm is done by alcohol than by either of them. I entirely agree with Dr. Anstie in the strong protest which he uttered against the practice of advising a hysterical woman to take a glass of wine or a teaspoonful of brandy, or even a dose of sal volatile to keep up her spirits whenever they are depressed, or to enable her to bear a frequently recurring pain. One should never sanction the employment of alcohol in any form by such patients, excepting in very moderate quantities and at meal times.

When one is called to a patient who is actually in a hysterical attack, there are several methods by which one may often succeed in cutting it short. One plan, suggested by Dr. Hare, is to keep the patient from breathing for a certain time, by forcibly closing her nose and mouth. The vigorous inspiration which she makes, as soon as she is allowed to do so, is said to be often followed by a relaxation of all spasm and a subsidence of the fit. Dr. Reynolds says that he has found this mode of treatment notably useful when an attack is sufficiently prolonged to make it worth while to interfere with the natural course of events. Another method, to the value of which I can myself testify, consists in drawing the patient's head and shoulders over the edge of the bed and pouring cold water upon them freely from a large jug. Sometimes one has merely to make preparations for carrying out this procedure, by giving the necessary orders to the patient's friends in her hearing, in order to bring the fit to an end. One must always keep in remembrance the fact that, excepting in seizures of a strictly epileptiform character, the patient is perfectly alive to all that is going on, although she may seem to be unconscious. A calm and quiet demeanor, the avoidance of all expressions of sympathy or of uneasiness about the issue of the attack, may do much to shorten its duration; if one is flurried or alarmed, she is sure to notice it.

I have already mentioned Charcot's observation that compression of one ovary will sometimes arrest a hysterical fit, even although it may be of an epileptiform kind; he speaks of one patient who learned to apply the pressure herself. Another procedure, which I have often seen adopted by the late Mr. Stocker, consists in pressing upon the arteries, and other structures on each side of the neck, in the same way as has already been described under the treatment of epilepsy (p. 712).

HYPOCHONDRIASIS.

DISTINCTIONS FROM HYSTERIA—THE SUPPOSITION OF IMAGINARY DISEASES, COMMONLY ABDOMINAL—DISTINCTION FROM MELANCHOLIA—MAY MASK REAL DISEASE—TREATMENT—PROGNOSIS.

From hysteria we pass to hypochondriasis by what seems a natural transition, for these two neuroses are commonly regarded as closely allied to one another, and do in fact resemble one another in one prominent symptom, namely, their tendency to simulate organic diseases of various parts of the body. Indeed, some writers have gone so far as to declare that hypochondriasis, which chiefly occurs in men, is in the male sex the representative of hysteria in the female; and the uncertainty which still exists as to the relative frequency of hysteria in men and boys arises mainly from the fact that cases which would be at once set down as hysterical, if they occurred in women, are often wrongly attributed to hypochondriasis because the patients are of the other sex. In reality, however, there is an essential difference between the two diseases. We have seen that the mental state in hysteria is characterized by an exaltation of the emotional faculties, and an increased susceptibility to outward impressions, the will being unable to control the feelings. But in hypochondriasis the morbid sensations which the patient experiences are not dependent upon any external cause; they are almost entirely created by his mind itself. And thus Romberg was not without justification when he designated it the very antithesis of hysteria.

Moreover, hypochondriasis is unattended with any of those nervous vagaries—convulsive attacks, paralytic affections, hyperæsthesia, anæsthesia—which are so characteristic of hysteria. Its predominant symptom is always of one kind; the patient believes, without cause, that he is the subject of serious bodily disease. It is true that the full development of the complaint is said to be in most cases preceded by a period in which there is merely a general perversion of his feelings. He becomes gloomy, reserved, and wrapped up in himself; or his mental state alternates between a moody silence and high spirits with great loquacity. Presently, however, he becomes conscious of a pain which seems to have its seat in some particular region of his body. Upon this he at once concentrates all his attention, and he is now a "hypochondriac."

The part to which the morbid sensations of hypochondriacal patients are most commonly referred is, as might be inferred from the name, the upper part of the abdomen. The disease is one which has retained the same appellation since the days of Hippocrates and Galen. But they conceived the actual seat of the disease to lie in the viscera which are situated immediately below the diaphragm. And a similar hypothesis was intended to be conveyed by the equivalent English expression, "the spleen." Indeed, it is the fact that the digestive organs are generally more or less disordered. Dyspepsia is often present; the bowels are constipated; large quantities of gas are apt to accumulate in the alimentary canal, giving rise to flatulent belchings and

noisy rumblings in the abdomen. And, in all probability, the starting-point of the morbid hypochondriacal feeling is really an impression proceeding from the viscera. In describing the symptoms of disease of the stomach and of the liver, I shall lay stress on the depression of spirits and irritability of temper which so often accompany them. But what is peculiar to the complaint with which we are now concerned is the marked exaggeration of every local pain. Moreover, the region to which the pain is referred seldom, if ever, remains the same during the whole progress of the case. After having dwelt for a length of time upon a gnawing or burning pain at the epigastrium, which he felt sure must indicate cancer of the stomach or liver, he all at once loses this, and, perhaps, begins to suffer from symptoms which, if due to organic disease, would point to the throat as its seat. Or he is attacked with dyspnœa and palpitation of the heart, and feels convinced that he is laboring under serious cardiac mischief; or a slight cold leads to a cough, and he begins to collect the sputa, is certain that he is phthisical, and consults all those physicians who have the greatest reputation for pulmonary affections; or he finds himself giddy, and experiences a sensation of weight and pressure in the head; and forthwith he thinks of nothing but of the apoplectic fit which he believes to be impending over him. But, perhaps, the most miserable of all hypochondriacs are those who refer their sufferings to the genital organs. To this class belong by far the larger number of those who consult one for spermatorrhœa, or for impotence. They complain that they have dragging or stinging pains in their testes, that these organs always hang too low, that their urine is turbid and must contain semen, that they have emissions at night, during defecation, and at other times. One sees at once that their whole attention and thoughts are concentrated upon the sexual function; and, to make matters worse, they are sure to have studied with avidity a number of books and pamphlets, written for the very purpose of stimulating their apprehensions, and of inducing them to seek relief at the hands of quacks and charlatans.

If further evidence were required to prove that the disease is a neurosis, it would be afforded by the close relation which can be traced between it and other nervous affections. This was especially insisted on by Anstie, who maintained that the hypochondriac almost invariably belongs to a family of which other members have been of unsound mind. As to whether the disease tends to pass into insanity in the same individual, different writers have expressed somewhat different opinions. Melancholia is the form which bears the closest superficial resemblance to it; but Romberg points out that this is characterized by a tendency to self-negation, whereas in hypochondriasis the whole attention is concentrated upon personal feelings and sensations. And he goes on to point out that the difference is expressed in all the patient's relations with other persons. An individual affected with melancholia treats his medical attendant as an enemy or as an ignorant pretender, and constantly tries to avoid him; but the hypochondriac looks up to him as his guardian and saviour. He is constantly on the search for new plans of treatment, and expects that each fresh one will succeed in relieving him of his complaints. As Romberg puts it, "the more physicians, the better he is satisfied; he likes to change them as often as he would change a poultice." Yet, in spite of all, he is not unhopeful, and is never weary of life. A tendency to commit suicide is no part of hypochondriasis; and any doubtful case in which such an attempt is made may be safely set down as one of actual insanity. And probably the same may be said of every case in which the patient has distinct delusions, as (for example) that he is made of glass, or that the devil is playing a barrel organ in his belly, or that the bowels are about to escape

through the abdominal parietes, or that he has swallowed spiders, and that they are multiplying within his body. Griesinger, though so great an authority, seems to have entirely mistaken the boundary line between hypochondriasis and melancholia; the cases which he relates as examples of the former are almost all of them really instances of the latter disease. This, perhaps, explains the circumstance that he found hypochondriasis "extraordinarily frequent in young people," and occurring sometimes in the years of childhood; whereas Anstie was, I believe, expressing the general opinion when he said that it is scarcely ever seen in those who have not reached the age of puberty, and that the majority of those affected with it are middle-aged men. However, he also remarks that it very rarely makes its first appearance in any one who is more than fifty years old at the time.

A point which must always be kept in mind is that a person laboring under well-marked hypochondriasis may also have organic disease; of the stomach, for example; or of the liver; or an aneurism of the aorta. Many a patient has had his complaints made light of, until the signs of one or other of these diseases have become too manifest to be overlooked, or until he has died suddenly, from rupture of a large vessel, or from angina pectoris. Anstie further remarked that in certain cases the possibility of malarial poisoning or of chronic alcoholism must be thought of, before one sets down a patient's symptoms as the result of mere hypochondriasis.

The treatment of this disease is commonly difficult and unsatisfactory. An essential point is that one should not attempt to make light of the patient's sufferings, but should show one's self to be really interested in and anxious to relieve them. Exercise, short of severe fatigue, should be taken daily, and mental occupation and amusement should be cultivated with the greatest care. Sea-bathing, the cold-water cure, the warm baths of Gastein, may each be prescribed with advantage; but it is difficult to say how much of the benefit is due to change of air and scene, and alteration in the habits of life. Any disorder of the digestive organs must, of course, be carefully investigated and corrected. Anstie spoke highly of cod-liver oil; but he added that many patients cannot take it for a sufficient length of time. Valerian is said to be sometimes useful; but neither quinine, strychnia, nor phosphorus is generally of much service, nor even iron, unless there be marked anæmia. Anstie laid stress on the importance of not prescribing alcohol for hypochondriacal patients, even in the form of medicinal tinctures, lest by doing so one should encourage habits of over-indulgence in drink.

In weighing beforehand the probable success of treatment in a case of hypochondriasis, one has to take into consideration not so much the apparent severity of the symptoms, as the length of time they have lasted and the circumstances under which they began. The younger the patient the better the prospect of cure; particularly if the family history is good, so far as regards the occurrence of insanity or other nervous diseases in his near relations.

PSYCHOSES

OR

FUNCTIONAL DISORDERS AFFECTING THE MIND.

INSANITY MOSTLY A FUNCTIONAL DISORDER—CLASSIFICATION OF ITS FORMS —PRODROMATA.

Acute Delirious Mania.—ORIGIN—SYMPTOMS—PROGNOSIS—TREATMENT.

Acute Mania.—DELIRIUM ABSENT—COURSE—TREATMENT—PROGNOSIS.

Chronic Mania. — MONOMANIA — DELUSIONS — ILLUSIONS — HALLUCINATIONS.

Acute Dementia.—DESCRIBED BY ESQUIROL—SYMPTOMS—PROGNOSIS—TREATMENT.

Melancholia.—SYMPTOMS—M. ATTONITA: M. ACUTA—FORCIBLE FEEDING —DRUGS—QUESTION OF "MORAL INSANITY."

Chronic Dementia.—NOT ALWAYS FUNCTIONAL—MEMORY—PROGNOSIS. GENERAL ÆTIOLOGY OF INSANITY.

Idiocy.—MORBID ANATOMY OF BRAIN AND SKULL—SYMPTOMS—VARIETIES—IMBECILITY—DIAGNOSIS.

Cretinism.—ENDEMIC FORM — CONFIGURATION — RELATION TO ENDEMIC BRONCHOCELE—SPORADIC FORM.

The Cretinoid Condition in Adults.—DESCRIBED BY GULL—CONFIRMED BY SUBSEQUENT OBSERVATIONS—MYXŒDEMA—SECONDARY PRODUCTION OF THE CONDITION AFTER REMOVAL OF THE THYROID IN MAN AND IN THE LOWER ANIMALS.

We pass to the consideration of insanity, that terrible malady which is regarded by the law as rendering the patient irresponsible for his actions, and justifying his confinement and restraint. The legal point of view is not, however, that from which we approach this subject. We treat of insanity as a matter of clinical study, as a disease which threatens and may destroy life, but which is often curable by proper advice and treatment. In strictness, insanity is not one disease, but many,—a group of allied disorders, connected by intermediate links, but not less distinct from one another than most which find places in our nosology.

With two exceptions, all the forms of insanity—the Psychoses as they are called—must at the present time be placed with the functional diseases of the brain. They are not known to depend upon any recognizable structural changes in the nervous centres. In rapidly fatal cases, indeed, the pia mater and the cerebral substance may be found hyperæmic, and in chronic cases the brain may be wasted, the convolutions shrunken, and the ventricles dilated. But in many instances no such appearances can be detected, and when present they are not to be regarded as essential. Every pathologist has made post-mortem examinations of persons who had for years been insane, and in whom the nervous centres appeared absolutely normal. The two diseases which differ from the rest in this respect are Idiocy and General Paralysis. In these the brain presents organic defects, in the one imperfect structural development, in the other atrophy or degeneration. I have, therefore, described general paralysis with the organic affections of the brain (p. 586); but find it convenient to take idiocy after the psychoses.

A logical *classification* of the several forms of insanity has been strenuously aimed at by writers on the subject, just as has been the case with some other groups of diseases that come chiefly under the notice of specialists. Such attempts, however, have met with but little success. Morbid anatomy being unavailable, ætiology would naturally suggest itself as an alternative basis; and in 1863 Dr. Skae proposed an arrangement upon this foundation, but his scheme includes at least twenty-five varieties, few of which can be regarded as really distinct from one another, while most of the particularly important forms of mental disease are left altogether unnoticed. And I find that although later writers speak highly of its merits they do not, in fact, make use of it. The order which they really adopt is more or less based upon the very simple one long ago suggested by Pinel. The forms which he recognized have since been somewhat increased in number, or rather they have been partially subdivided. Thus, I find most writers describing separately (1) acute delirious mania, (2) acute mania without delirium, (3) chronic mania, including the so-called monomania, (4) acute dementia, (5) melancholia, (6) chronic dementia.

Each of these forms of insanity presents peculiarities in its symptoms, ætiology, course, prognosis, and treatment, of which brief clinical accounts must be given. They are not, indeed, mutually exclusive, and intermediate forms between them occur, just as is the case with the other neuroses or the various forms of dyspepsia or of Bright's disease, and between many cutaneous affections and the various forms of new growth.

Prodromata.—But before I pass to the consideration of the separate forms of insanity, I must draw attention to certain early symptoms which commonly exist for some little time before the patient can actually be said to be of unsound mind, and which may fairly be called premonitory since they suffice to reveal to a trained observer the nature of the impending disease. Of these one of the most important is sleeplessness, or at least a marked deficiency in the amount of sleep. Another is a change difficult to be defined in the moral and emotional character. The patient's friends and servants notice "something odd" about him. Generally his spirits are depressed. Very often he is duller and more quiet than natural, or he may be more restless, irritable, or excitable. If a parsimonious man, he perhaps becomes extravagant; if a modest man, presumptuous or exacting; if an affectionate parent, indifferent to his family. The appetite is often voracious, alcoholic liquors are taken to excess, or immoderate sexual intercourse is indulged in. Sometimes the head is hot and painful, the face is flushed and the eyes are suffused.

It is of great importance that a medical man should be prepared to attribute to the right cause such symptoms as these; but the diagnosis is exceedingly delicate, especially if one should happen not to have known the patient before. Some years ago, I was consulted by a bank clerk on account of sleeplessness and depression of spirits, and anxiety of mind about his duties, which had recently been changed, so as to press more heavily upon him than before. His articulation also was thick, but I did not know how much importance to attach to this, having until then never heard him speak. I allowed him to leave my house, and several hours afterward he was found by the river side. Whether he had been thinking of drowning himself could not be ascertained. He quickly became maniacal, and was insane for several months. He is now well and has resumed his duties. The thickness of speech remained noticeable until his mental powers were restored, but afterward it disappeared.

But cases occur in which the disease begins quite suddenly and without any premonitory symptoms, and as such cases run a more rapid course than any others it will be convenient to take them first.

Acute Delirious Mania, as it is called by Dr. Blandford and others, is often caused by a mental shock, such as is produced by the sudden death of a friend, a disappointment in love, and the like. Or it may set in after parturition, after a drunken bout, after exposure to intense solar heat. It may follow the fatigue of a very long walk. It may develop itself in the course of phthisis, or after a febrile disease, such as acute rheumatism. It may occur in an epileptic patient, but it is then usually very transitory, assuming the form which I have described at p. 719 as paroxysmal insanity. Dr. Blandford, however, relates the case of a gentleman who within a year had two attacks of mania, each of which passed off in an hour or two. After some time he had a third seizure, but this lasted a week, and six months later still he had a fourth, from which he had not completely recovered at the end of two years.

A patient suffering under this form of mental disease may pass hours, or even days, in singing and shouting, and may never rest for a moment. His speech is commonly a jumble of unconnected sentences, or the constant repetition of a word or phrase, which he may begin to utter in a low tone and repeat more and more loudly until the room rings with his piercing cry. He may declare that he is the Deity, or an emperor, or that he is acquainted with all the sciences, or that he possesses all the treasure of the world—in the same breath he may say that he is a chair. It is important to note that none of these delusions are fixed. Each phase of the disease brings with it fresh ideas which dispel the old ones. The patient may be in a state of terror, and may scream fire or murder; he may be furiously angry, attacking those about him, or reviling them in the most opprobrious terms; or he may be gay and hilarious, shouting and laughing with glee and mischief. But in many cases no delusion can be made out from the confused jargon which he pours out.

Maniacs are commonly supposed to be endowed with much greater muscular strength than those who are sane, but Griesinger says that this is not generally the case, although he admits that they are able to continue their violent muscular movements much longer.

Sleeplessness is one of the most important symptoms. Dr. Blandford mentions the case of one young lady who during eight days only had one hour's sleep. The sexual instinct is often intensely active. The appetite may be ravenous and indiscriminate, so that the patient will eat the most digusting food with avidity. The pulse is not necessarily much quickened. During the paroxysms of struggling it may become very rapid, but in the intervals of comparative quiet it falls to the normal rate. The temperature also is but slightly increased in most cases. There is profuse perspiration, and the secretion from the skin may be highly fetid. Sometimes, however, a "typhoid" state develops itself, the pulse remains very quick, the temperature is from three to five degrees above the natural standard, the tongue dry and brown, and the teeth covered with sordes. These symptoms are very unfavorable.

Acute delirious mania is a disease of young adults, generally between the ages of twenty and thirty. It is common in women, and there is often a difficulty in distinguishing it from hysteria.

At the commencement of an attack of this kind it is exceedingly difficult to foretell its probable duration, and yet it is very important, because one has to determine whether or not the patient should be removed to an asylum. Dr. Blandford recommends that, if possible, one should wait three or four days before taking this decided step. He observes that a patient, who would have rapidly recovered if treated at home and among friends, often experiences a much more severe and prolonged attack when forcibly removed and placed in the hands of strangers.

Acute delirious mania, however, is a disease which usually runs quickly to a termination, either in recovery or in death. Dr. Blandford says that it takes the latter course in one case out of about three or four. Much depends on the strength of the patient. It is a favorable sign when the emotional state is one of cheerfulness and gayety rather than of depression. A fatal termination occurs either by coma or by collapse. Dr. Maudsley remarks that it is often very sudden and unexpected, so as to leave in one's mind an uneasy feeling that one's treatment many not have been judicious.

A patient affected with this form of mental disease should be placed in a large room with windows out of reach or protected. It is not absolutely necessary that he should be removed to an asylum, but he must be placed in the charge of thoroughly-trained attendants. The room should be kept dark. The bed must be made up on the floor, a considerable part of which may be covered with mattresses. As he is very likely to strip his clothes off and tear them to pieces, he should have a strong suit made, of which the different parts are fastened together in a single piece, and laced up the back. Or he may have a blanket fastened over his neck and round his arms.* The patient's relatives should not, as a rule, approach him, but the occasional presence of an old friend or servant may do much to quiet him. It must be borne in mind that those who are the subjects of this form of mania often know far more about what is going on than would be suspected from their acts and gestures; attendants and others must, therefore, be careful what they say.

It is of great importance that plenty of food should be taken, and, if possible, solid food. The patient will also require an abundant supply of drink, such as lemonade or soda water. It is well to avoid stimulants, at least when the patient is young and of sound constitution, but bottled ale or stout may be allowed in moderate quantities. As the case advances, one is commonly obliged to fall back upon beef tea and broths, and it may then be necessary to give brandy or wine.

Among medicines, Dr. Blandford recommends chloral as the most valuable. He gives it in doses of a drachm in cases of acute mania; he says that he has never met with a case in which some sleep did not follow its administration, and that a patient who had previously been very violent may, after taking it, wake up perfectly free from delirium, although still affected with delusions. On the other hand, he objects to opium, and even to the subcutaneous injection of morphia. He admits that the patient may be made to sleep for half an hour by these drugs; but when he wakes, the mania is often worse than ever.

It is generally well to give a brisk purge at the commencement of the attack; but in the later stages the repeated administration of aperients is to be avoided. Bleeding and cupping and blistering are no longer employed in acute mania.

Warm baths are often employed in cases of this kind, and seem sometimes to promote sleep, particularly if an ice bag be at the same time applied to the head. Dr. Blandford recommends that the temperature should not be higher than 90° or 92°; the patient should remain in the water for half an hour at least, but not for several hours at a time, as some French physicians have advised.

Packing in the wet sheet is another method of treatment which has been especially recommended by Dr. Lockhart Robertson. He keeps the patient packed for an hour or an hour and a half, and then takes him out, rubs him well with a dripping cold sheet, and after pouring two pails of cold water upon him, places him in another wet sheet and blankets. In some cases he has pursued this system throughout the day, or has repeated the

* These suggestions are taken from Dr. Blandford's work.

treatment three or four times in the day. But Dr. Blandford says that such a practice is not unattended with risk.

In cases which terminate in recovery, the first favorable indication is generally that the patient sleeps. When he wakes, his maniacal condition remains with but partial improvement. But he sleeps again and again, and at length his mind recovers itself. The period of time required for convalescence is very variable. Dr. Blandford says that a man affected with severe sleepless delirium of considerable duration, may be well in a month from the commencement of his illness; but other patients remain for weeks or months in a condition of nervous prostration, and yet ultimately recover. In some cases all that occurred during the attack is registered in the memory, in others the patient can recollect nothing.

In certain exceptional cases the disease terminates neither in death nor in recovery, but in permanent chronic mania or dementia. It is also to be borne in mind that even in the most favorable cases there is for a considerable time a danger of relapse.

Acute Mania.—The disease just described has been designated "acute delirious mania," for the same term is applied by alienist physicians to another form of insanity, of which the symptoms and course are very different. Dr. Blandford distinguishes this as "acute mania without delirium." The patient may be extremely noisy and violent, but he knows what he is about; his acts are guided by design. He may be very mischievous and destructive, but it is for the purpose of provoking those who have charge of him; and with the same end in view he is wet and dirty in his habits. He will begin singing and shouting, or will pour forth the most abusive language; but he knows perfectly well who are present. He may talk coherently for a time, and then wander off into incoherent nonsense. Or he may even show some skill in concealing his insanity, so that there may be a difficulty in getting his certificates signed. Not infrequently he practices self-abuse openly and shamelessly; or he may expose his person with respectable persons about him.

This disease is not, like acute delirious mania, especially apt to occur in young adults; many of those attacked by it are over forty years of age. It may either begin suddenly or after premonitory symptoms have existed for a longer or shorter period. The patient's bodily health is generally good. He eats heartily or even voraciously. He sleeps badly, sometimes not at all for several days together; but he seems to be able to do without sleep. He does not waste rapidly. His tongue may be clean, and his bowels may act regularly. His insanity may last for months without imperiling his life.

Indeed, many cases of this form of insanity run so protracted a course that the name of "acute mania" seems altogether unsuitable to them—at least, if one is accustomed to consider the epithet acute as the antithesis to chronic. Dr. Crichton Browne accordingly describes such cases under the name of chronic mania ("*West Riding Asylum Rep.*," vol. v). In one sense, indeed, the symptoms are "acute" enough, for the patient may go on chattering, laughing, or shouting, for days together. This writer mentions the case of a man, who worked energetically as a navvy all day, and who for six months continuously sat up in bed talking and shouting all night, and was never known to sleep. At the end of that time he had not lost weight. The writing of such patients, like their speech, is a continuous flow of sentences, perfect as regards grammatical construction, but consisting of utter nonsense, and connected together only by the most fantastic associations. Their countenances do not betray weakness and vacuity, but rather a combination of mirth and mischief; their eyes are bright and sparkling; they are generally smiling and often laugh inordi-

nately. Some of them adopt peculiar attitudes, others collect all kinds of useless articles such as pebbles or leaves ; and others love to adorn themselves with any cast-off finery that they can lay their hands on.

In patients affected with this form of mania an asylum is absolutely necessary. The only chance of managing them consists in moral control and discipline. Much may be done for them by a well-considered system of rewards and punishments. Dr. Blandford relates the case of a man who for four months had behaved outrageously. Two men were then placed constantly with him, so as to give him no chance of being mischievous, and he was deprived of his tobacco, of which he was very fond. In a month's time he was discharged, and he continued well for some years.

It is of great advantage to such patients to be made to take prolonged exercise in the open air. Among medicines Dr. Blandford recommends chloral as the best hypnotic, but he does not object to opium or morphia in this form of mania. A remedy which has been largely employed in many asylums is tartarized antimony. In doses of from one-third to half a grain it soothes the patient; and it is said not to cause nausea nor to spoil the appetite. Hydrocyanic acid is sometimes employed with a similar object.

The prognosis of this form of mania is difficult. Dr. Blandford says that the patient seldom dies, unless his health has been impaired by previous disease. He may even recover after having been insane for a considerable length of time. Dr. Blandford mentions one case which terminated favorably after the subject of it had been in an asylum for ten years, although it was the fourth attack. The younger the patient the better the prospect of recovery. It is a good sign that there should be noisy excitement and turbulence, and that the delusions should be fleeting and frequently altered. The presence of fixed delusions is of very unfavorable augury—particularly if the patient fancies he hears noises. Many patients pass into the condition presently to be described under the name of chronic mania, into dementia or melancholia.

Chronic Mania : Monomania.—We have seen that many cases of what is commonly termed acute mania run on for several months, and that Dr. Crichton Browne has fairly enough described such under the name of chronic mania. The latter name, however, is more commonly applied to a somewhat different class of patients, namely, to those whose chief symptom is that they are under the influence of fixed *delusions*. Another designation for cases of this kind is that of Monamania. But this seems to me likely to mislead, since it suggests the notion that the mental disorder consists only in the presence of a single delusion, the mind being on all other points sound. It appears really not less objectionable than the expression "partial insanity," which has of late been disused because it is now known that even when a patient laboring under delusions concerning some one or two points is intelligent and rational with regard to all others, it is incorrect to say that any part of the mind is sound and free from disease.

It is a peculiarity of these fixed delusions of chronic mania that they are all connected with the patient himself. Commonly they may be classed under what is termed mental exaltation. He believes that he is possessed of enormous wealth, high rank, or immense strength, he is going into Parliament, or about to rise to the summit of his profession, or on the point of patenting a new invention which will make his fortune. His manner and behavior are, therefore, in the highest degree self-satisfied, arrogant, and supercilious.

On the other hand, he may imagine that wicked men are conspiring to ruin him or his family, that his food or his clothes is poisoned, that his food contains blood or human flesh, and the like. But when his delusions

are of this nature they do not make him depressed in spirits (as in melancholia), on the contrary, his alarm makes him furious or violent.

Illusions are also frequent in cases of this kind, that is to say, wrong perceptions of the objects presented to the patient's senses. To use Dr. Blandford's illustration, he may see a chariot in the sky when every other person sees a cloud, or hear a voice when those about him recognize the sound of a carriage or a distant footfall. Those who labor under this form of insanity are also very apt to make mistakes with regard to the identity of others. A man thinks his wife and children are changed, declares a stranger to be a relation or friend, or asserts that a near relative is someone else. A woman says that her husband is not her husband but a stranger, and yet she asks after all at home. Dr. Blandford, indeed, says that mistakes of this kind are not to be regarded as illusions but as delusions; he believes that the fact really lies with the patient's ideas and not with his perceptive faculties.

Again, patients affected with chronic mania are also subject to *hallucinations*—that is, to false perceptions of the senses having no foundation in any impressions derived from without. They see angels or visions of the Deity, or spirits floating in the air like birds; or, again, they are visited by the ghosts of departed friends or heroes, by fiends, or by the devil himself. Dr. Blandford, however, remarks that hallucinations of sight are far less common in the chronic than in the acute forms of mental disorder. But, on the other hand, hallucinations affecting the sense of hearing are of exceeding frequency. The patient may hear sounds of whistling or humming or singing. Or he may hear actual words and sentences, uttered in a voice that may either appear well known to him or that of a stranger. As no one is to be seen, he generally imagines that the speaker is in the next room or house, or in a cupboard or chimney. Dr. Blandford, whom I am now again quoting, knew of a lady who was so annoyed by voices coming through the wall that she purchased the adjacent house to compel them to cease, but, of course, without the desired result. He goes on to say that such patients often regulate their whole lives by the commands they receive from the voices. Even if compelled to act otherwise, they believe that they will suffer for their disobedience, and they may commit the most frightful crimes without compunction or sense of guilt. They commonly will not repeat what the voices say to them, and one may, indeed, have great difficulty in extracting from them the fact that they are laboring under this kind of hallucination. They appear afraid to reveal the secret, or seem to think it a point of honor not to do so. One may often make a guess at it by noticing that when one is talking to them they are at times inattentive and appear to be listening to some one else, or one may overhear them answering and keeping up an imaginary conversation. All authorities recognize that these hallucinations of hearing are of exceedingly grave import as regards prognosis, and that patients who are affected with them are amongst the most incurable of all lunatics. Hallucinations of smell are much less common; some persons, however, declare that they smell fetid and noisome exhalations, the scent of the dead, or of vaults or catacombs, or that their food or drink has an offensive odor. Dr. Blandford remarks that they more commonly assert that an offensive smell proceeds from their own bodies, rendering them objects of disgust to others. I may remark that at least two patients have come to consult me on such an account, but I must confess I could not satisfy myself that their mental state was more than one of hypochondriasis. The occurrence of true hallucinations of taste seems not yet to have been established.

Some curious sensations of which these patients complain are classified by Dr. Blandford as hallucinations of the sense of touch or of the muscular

sense. Thus it is not uncommon for a person to say that he feels himself touched on all parts of the body by little raps or shocks, which he may even attribute to supernatural agencies. Other patients feel what they suppose to be electric shocks.

Patients laboring under chronic mania are almost always capricious in their temper. If one doubts or attempts to disprove their delusions, they are apt to become angry and excited.

The *prognosis* of this form of insanity is often exceedingly doubtful and difficult. If the patient is removed to an asylum at an early period of the disease, recovery is not infrequent. On the other hand, when it is already of two or three years' standing, one may safely say that it is incurable. Dr. Maudsley, indeed, remarks that, as a general rule, recovery does not take place in any case in which a fixed delusion has existed for more than six months. After a time dementia may set in. But many patients go on for years with but little alteration in their condition; they may live to the extreme limits of old age.

With regard to the management of such, all that it seems necessary to say is that moral treatment is more important than the administration of drugs.

Acute Dementia.—One of the less common forms of insanity—but a very remarkable form—is that which most writers, following Esquirol, call *acute dementia.*

This is a disease of young subjects. It seems never to occur after twenty-five or thirty years of age, and those whom it attacks are commonly weakly and have outgrown their strength, or have perhaps become exhausted by the confinement and monotony of prison life, or of factory labor amid the ceaseless noise and motion of machinery. Dr. Crichton Browne ("*West Riding Asylum Reports*," vol. iv) says that it comparatively seldom depends on an inherited neurotic tendency; such a tendency was present only in three cases out of twelve, in which he fully traced out the family history. It is more common in girls than in boys. Sometimes it is directly excited by a shock of fright, sometimes by exposure to cold or by excessive fatigue. Masturbation may induce it, or protracted diarrhœa, or loss of blood from piles, or long-continued leucorrhœa or menorrhagia. Sometimes it follows an attack of some acute disease, such as enteric fever; and this even though delirium was not present when the illness was at its height. Dr. Browne thinks that it may have its origin in malaria.

Symptoms.—The commencement of acute dementia may be gradual or sudden. In the latter case its onset is attended with a brief outbreak of excitement, wild laughter, and fury. In the former case there are no such symptoms, but rather a retardation or suspension of all mental and bodily activity. The countenance is vacant; the attitude lethargic; the voice low and drawling. When spoken to the patient takes no heed; even if he can be roused to answer a few questions he soon shows that his memory is gone, and that he cannot collect or arrange ideas. A point mentioned by Dr. Browne is that such a person often repeats the questions put to him, or the last few words of them, instead of attempting to make any reply. A girl under his care, after her name had in vain been asked for at least a dozen times, at last cried out "Elizabeth" in a shrill treble, and for the following month, whenever she was spoken to, she invariably said "Elizabeth," with the same sharp accentuation, but never raising her eyes nor changing her attitude. In extreme cases of acute dementia the patient often sits or stands motionless for hours together, staring at vacancy; or he may incessantly repeat some particular movement. Dr. Blandford mentions the case of a girl who would go on snapping her jaws together for days at a time,

and presently change this action and wag her head continually from side to side. A sort of cataleptic state often develops itself; if made to stretch out the arms horizontally or to raise them over the head, the patient may keep them in such a position for twenty minutes or more. The habits are generally dirty. Such persons often require to be fed like infants and to have everything done for them. They do not usually resist taking food, but they are apt to be sick when it has reached the stomach. They may have occasional gusts of excitement, aimless garrulity, restlessness, and violence. They often perform movements of an automatic character. Thus, if set to run, they often go on until they are stopped by some obstacle. Dr. Browne relates a case in which by putting a spoon into the patient's hand and carrying it several times backward and forward from a plate to her mouth, a tendency to the continuance of this series of movements could be impressed upon her. But, when left to herself, she did not even look to see whether the food was still within reach, but carried the spoon always across the same part of the plate, so that this had to be shifted from time to time.

In well-marked cases of this kind Dr. Browne says that the surface of the body becomes anæsthetic. Pricking or pinching the skin causes no pain, tickling gives rise to no movement. Irritation of the nostrils, even, is not followed by sneezing. The mouth is kept wide open, and the saliva is allowed to run from it. Dr. Browne remarks that this secretion is sometimes increased in quantity, so that as much as a pint has been collected in five hours.

In patients suffering under acute dementia the heart's action is exceedingly feeble. The pulse at the wrist may be so small as to be scarcely perceptible. The hands and feet are cold and blue, and often swollen. Chilblains form on them, which pass into indolent sores, and these may persist even during the summer, and although the parts are kept wrapped in cotton wool. The nose, ears, and cheeks also may be of a reddish-purple color. Dr. Crichton Browne, however, says that the temperature in the rectum or axilla is nearly normal, although in the hands the thermometer remains from 12° to 15° lower. The pupils are dilated and inactive. The respiration is quiet and shallow; sometimes the patient can hardly be seen to breathe.

Prognosis.—In the more extreme cases œdema of the lungs is apt to occur as a complication, and pneumonia or phthisis sometimes proves fatal. But English writers are agreed that most cases terminate gradually in recovery, the patient having afterward no recollection of what has happened. Dr. Blandford, however, observes that some cases of which he had had hopes proved to be examples of permanent dementia. The only disease with which acute dementia can be confounded is melancholia.

Treatment.—Removal to an asylum is not absolutely necessary in this form of mental disease; but the proper treatment can very rarely be efficiently carried out in the patient's home.

The regular administration of food is of the first importance, and it may be necessary to employ the stomach pump. Dr. Browne mentions a case in which the patient (who afterward recovered perfectly) took no nourishment for three months except by its means. The plan adopted in the West Riding Asylum is to feed the patient at least three times a day and to administer during the night two enemata composed of beef tea, butter, and port wine. Moderate exercise in the open air is beneficial. Care must be taken to keep up the warmth; but shower baths of ten seconds' duration are often very useful if followed by brisk friction. Dr. Clifford Allbutt applied galvanism in some cases with marked benefit. The current from a battery of from five to twenty cells was passed from forehead to occiput, or from one lateral region to the other, for ten minutes every day. The

patients were very little sensitive to its application, but they sometimes became flushed and giddy. The coldness of the extremities was diminished, and any tendency to the cataleptic state was always lessened. One patient, who, during four months previously, had made no progress toward recovery, was convalescent in twenty-four days, another in about seven weeks. Among medicines, Dr. Browne speaks of quinine in doses of ten or fifteen grains as the most useful, but iron and other tonics may be of service.

MELANCHOLIA.—In another well-defined group of insane patients, the preponderating symptom is extreme depression of spirits, a feeling of profound misery, which is either admitted by the patient to be causeless or attributed by him to an imaginary cause. The disease is then termed melancholia; it can often be recognized at a glance by the dejected aspect of the sufferer. Griesinger describes such a one as standing motionless, or sitting in a corner all day long, taking no notice of anything, his eyes cast down or fixed widely open with an expression of suffering or astonishment, his face contracted, his eyebrows wrinkled, the angles of his mouth drawn down. Melancholic patients generally look much older than they really are. They sleep badly, but are not altogether sleepless. They may have but little appetite, or may eat enormous quantities of food. They are thinner than in health, but do not waste rapidly. Their circulation is feeble, and their extremities are cold.

Sometimes, instead of remaining without movement, patients of this class are in a state of perpetual restlessness, wringing their hands, moaning and crying, loudly accusing themselves, and bewailing their miserable condition. Griesinger mentions a great desire to wander about, to make long excursions, and to visit friends and relations, as one of the symptoms which such persons present.

There need not, at first, be any perversion of intelligence. As Dr. Maudsley remarks, the patient may, for a time, be conscious of the change in himself, and may grieve over it and strive to hide or fitfully resist it. But at length he succumbs to it entirely; he becomes more and more self-absorbed, and more and more indifferent to and distrustful of those about him. Some definite delusion now develops itself, which is often connected with religious ideas. He believes that he has committed the unpardonable sin, that he is possessed by the devil, or that he is forever damned. Or he imagines that he has committed a murder or some other horrible offence, very often some particular crime about which he happens to be reading in the newspapers of the day. Or, again, he supposes that he is ruined, and that he and his children are going to the workhouse. Or his delusions may refer to his bodily organs. He has pains in various parts of him. He is eaten up with syphilis. He has a serpent in his stomach, or spiders breeding in all parts of the body, or a burning fire consuming his vitals and reducing him to cinders. It is the last-mentioned cases that afford the transitions between hypochondriasis and melancholia, to which I have already alluded.

The cutaneous sensibility is commonly impaired in cases of melancholia; there may even be complete anæsthesia of some parts of the skin.

The period of life at which melancholia is most often observed is between the ages of forty and sixty, but sometimes it is seen in young persons and even in children. Dr. Blandford speaks of it as apt to occur in women a few weeks after parturition, particularly in those much weakened by their confinement.

Subacute Form.—The course of this form of mental disease is generally chronic. Dr. Blandford prefers to call it "subacute." It sometimes presents remissions, but very rarely complete intermissions. Griesinger, however,

mentions the case of a woman affected with intense melancholia who once had a lucid interval of nearly a quarter of an hour's duration, for which he could discover no cause, and which ended as suddenly as it began; and Dr. Maudsley has more than once seen such patients wake in the morning cheerful, and seemingly quite well, remain so all day, and yet be as bad as ever on the following day.

The prognosis of this "subacute" melancholia is by no means unfavorable. Dr. Blandford says that in his experience he finds almost every case, whether in or out of an asylum, terminate sooner or later in recovery, unless it passes into one of the more grave forms of the disease which I shall presently describe. The ordinary duration of it is said by Griesinger to be from six to twelve months. The subsidence of the symptoms is generally gradual, one after another disappearing, while the remissions become more and more prolonged. Dr. Blandford has related three instances in which patients recovered after having been inmates of asylums for very long periods, two of them for five years, and one for seven years. He observes that there is no other form of mental disease in which a favorable issue can be hoped for after so great a lapse of time. The point is one of considerable practical importance. In cases of this kind, the taking out of a commission of lunacy should, of course, be delayed as long as possible, and the relations of the patient must be cautioned not to make pecuniary or other arrangements on the assumption that he is hopelessly insane.

Four cases are mentioned by Dr. Maudsley ("Syst. of Med.," ii, p. 16) in each of which melancholia was quickly removed by the cure of a prolapsus uteri from which the patient suffered. In one instance the mental disorder is said to have returned regularly when the pessary was removed. I am not aware that any other affection of a part at a distance from the nervous centres is capable of exciting it.

Alternating Form.—In some cases melancholia passes into mania. Griesinger says that he has seen the two diseases alternate at particular seasons of the year, the patient becoming melancholic in winter, maniacal in the spring, and in the following autumn again melancholic. In other instances the transformation from one to the other form of insanity takes place regularly every few days. Falret and other French writers have directed special attention to this variety of mental disorder under the name of *folie circulaire*, or *folie à double forme;* its prognosis is extremely grave. Many cases of melancholia also pass into dementia.

Ingravescent Form.—But in other instances melancholia tends toward an unfavorable termination by its proper symptoms undergoing a great aggravation and increase of severity, and of such cases there are two distinct forms, one of which is commonly known under the name of "mélancholie avec stupeur" (*M. attonita*), the other under that of "acute melancholia."

(*a*) In *mélancholie avec stupeur* the patient seems simply lost to all that goes on about him. He lies helpless, suffering himself to be washed and dressed, and placed in a chair and fed, but rendering no assistance whatever, even if he do not offer more or less passive opposition. He takes not the slightest notice of his friends and relatives, and gives no sign of recognizing them; he may make no attempt to speak for years together. Yet even in cases of this kind recovery may take place, and Griesinger remarks that it is often sudden, the sufferer seeming (as it were) to wake from a dream. A striking instance of this has been recorded by Dr. Buzzard, of a gentleman who for four years had maintained obstinate silence, and had just suffered himself to be fed and cared for under the protest of black looks, clinched jaws, and an inarticulate grunting. One day his brother, who was visiting him at the Morscroft Asylum, talked about his eldest boy, for whom he had a strong partiality. The patient showed vehement signs

of irritation by frowning and grunting. A few days afterward, when the attendant was about to feed him in the morning, he asked for a cup of tea. From this time he spoke and took his food naturally, and he was very speedily restored to perfect health. At first he was unable to stand, but at the end of six weeks he could walk a mile. It was afterward ascertained that he remembered all that had occurred during his illness and that he had accurately kept record of the time that had passed. He said that he could not account for his behavior, but that he had felt that no power on earth could make him speak or eat. His weight had fallen from 11 stone 10 pounds, until it was at one time a little over 6 stone 12 pounds.

(*b*) The condition known as *acute melancholia* is very different. Dr. Blandford describes such a patient as "panic stricken. In violent frenzy and terror he paces the room, and dashes at the doors and windows, eager to escape from the doom that awaits him, or from the police who are on his track. He will not sit on a chair, or lie still on his bed, but is incessantly running about exclaiming that he is going to be burned or tortured. . . He resists with the utmost violence all that is done for him. He will wear no clothes, and will not be washed. . . . He refuses food not passively, but with all his might; ejecting it even when it is placed in his mouth." But unless nourishment is administered forcibly such cases may quickly prove fatal. Gangrene of the lungs often sets in, and sometimes a condition resembling scurvy is developed.

Patients affected with this form of melancholia show suicidal tendencies in the most extreme degree. They not only try to kill themselves, but will inflict on themselves the most terrible injuries. Thus Dr. Blandford speaks of them as gouging out their eyes, cramming things down their own throats, swallowing nails or bits of glass, or breaking their legs and arms in the furniture.

But it is important to note that the tendency to commit suicide is by no means limited to the more extreme forms of melancholia. It forms part of even the mildest varieties of the disease.

Thus Dr. Maudsley relates the case of a quiet man, having the delusion that his soul was lost, who had been months under care, and in whom no one suspected any mischief, but who suddenly started up from bed one night, and flung himself out of a window through which it would have been thought impossible that any man could get. And Dr. Blandford insists on the frequency with which it happens that a patient belonging to this class commits suicide at a still earlier stage in his disease, "before his friends have noticed anything like delusion in him, and when they only thought him a little low and were afraid to take any measures for his safety, for fear of worrying him. Hundreds and hundreds of inquests are held upon patients of this kind, who by the commonest care might have been successfully treated and cured." Melancholia is indeed the form of insanity in which suicide is most commonly attempted; and this may be the first symptom of unsoundness of mind. Thus Dr. Forbes Winslow related the case of a lady in whom no one had remarked anything extraordinary, but who suddenly sprang up during dinner and tried to throw herself out of the window; the attempt was frustrated, and forthwith she became maniacal. Some eminent French writers have maintained that all persons who kill themselves are insane. This opinion is certainly untenable; for those cases in which remorse for crime or a profound feeling of shame on account of real degradation supplies an adequate motive for suicide; but it is doubtless correct so far as concerns the majority of those cases in which no such explanation can be given. Children sometimes destroy themselves on account of what seem trifling disappointments. Griesinger mentions the case of a boy who killed himself because he had lost a bird; and that of

another, aged five, who threw himself into the water because his mother whipped him.

Treatment.—The tendency on the part of melancholic patients to suicide renders it necessary that they should never be left alone for a minute. Hence, unless they are in very easy circumstances, they have generally to be placed in an asylum. In the treatment of this form of mental disease the most important point is the administration of food. Persons affected with melancholia are said by Dr. Blandford to be almost invariably better toward evening, and worse in the morning, when they have been without food. In the milder forms of the disease, meat, poultry, and fish should, if possible, be given. Dr. Blandford especially insists that a foul, coated tongue, a fetid breath, and constipated bowels, are no indications that solid food will not be well digested. He recommends a very liberal diet, with but short intervals between the meals, and considerable quantities of wine and stout.

But in the more severe forms of melancholia the patient can be made to take food only by force, and, of course, liquids alone can then be given to him. There are great differences of opinion as to which is the best method of forcible alimentation for such cases. Dr. Blandford discusses the various plans at considerable length. In the first place, it is necessary that the patient's body should be fixed. One way of effecting this is to place him on his back on a mattress, on a floor, with bed clothes over him, and a bolster supporting his head. He is to be in his night dress, and his neck must be free from clothing. Two attendants then kneel, one on either side of him; they place their hands on each of his wrists and on each shoulder, pressing it down. By this method few, if any, bruises need be inflicted. The operator then kneels at the patient's head and proceeds to introduce a spoon between the teeth. He may do this, if the patient should be a woman, by getting her to talk and slipping it in when she opens her mouth. But in any case he can generally succeed by making a little persistent pressure upon the teeth, and, if necessary, by inserting a finger between the gums, behind the last molars. The best spoon to use is a small iron one with a straight handle. When it has been introduced it should be retained by the thumb and index finger of the left hand, the palm and remaining fingers firmly grasping the chin and fixing the head. A third attendant closes the patient's nostrils with one hand. The operator now with his right hand pours food into the patient's mouth, who cannot help swallowing it. It should be held in a gutta-percha bottle containing about half a pint and provided with a nozzle; through this about half an ounce is to be squeezed after each act of expiration. Dr. S. W. D. Williams, who has advocated the method, says that by its means one can in all ordinary cases administer at least a pint in from ten to fifteen minutes without a possibility of inflicting any injury on the patient.

But Dr. Blandford says that it is not possible in such a way to keep a powerful patient quiet without struggling, and that an obstinate one will succeed in ejecting by an expiration some at least of the fluid. Moreover, a part of it is apt to pass into the larynx, exciting cough and a sensation of choking. It seems to me not improbable that this may be one cause of the gangrene of the lungs which has already been mentioned as of frequent occurrence in the more acute forms of melancholia; but I do not find any allusion to such a possibility in works on the subject.

Another method by which a patient may be forcibly fed consists in the introduction of a slender œsophageal tube into the stomach through the nose. This has been especially recommended by Dr. Harrington Tuke. The tube which he employs is seventeen inches long, and of a diameter varying from that of a No. 3 to a No. 6 catheter. Sometimes difficulty is experienced in the performance of the operation from the instrument catching against the

cervical vertebræ; it is said that one can obviate this by bending it so as to give it a tendency to turn downward, and also by throwing the head of the patient backward just when the end of the tube is passing the posterior nares. Afterward, to avoid the risk of introducing the instrument into the larynx, the patient's head may be bent forward and downward, but it is said that for a practiced manipulator this is unnecessary.

The plan which Dr. Blandford himself prefers consists in the employment of an ordinary thick stomach-pump tube, which is passed through the mouth. The patient is first placed in a wooden arm-chair, and his body and limbs are then swathed in sheets drawn through its arms and legs so as to render him immovable. If the teeth are closed, they are gradually opened by means of a silver-plated wedge, which is made to expand by a screw. A wooden gag is then introduced, which is held by an attendant standing at the patient's head. The œsophageal tube is so large that it cannot enter the larynx, and it is flexible to its very end. It is passed through a hole in the gag and directed slightly to the right. The patient may press upon it with his tongue and so prevent its descent, but in a few seconds he is obliged to take breath and relaxes his hold upon it. When it has passed through the cardiac orifice an injecting apparatus is fixed to the extremity which projects from the mouth, and food is at once pumped into the stomach.

This operation has sometimes to be repeated over a very long period, in cases of melancholia. Sir William Gull has related ("*Trans. Clin. Soc.*," vol. viii, p. 41) an instance in which a patient was fed 7647 times with the tube in the course of about three years and eight months. The instrument was passed by a medical man on each occasion; the time occupied in the whole procedure was only forty seconds.

The medicines most useful in this disease are the hypnotics. Chloral is especially serviceable in the form described as acute melancholia; by giving sleep it husbands the patient's strength. Morphia administered subcutaneously is very valuable in the same class of cases. In the subacute variety Dr. Blandford recommends that some preparation of opium should be given regularly, in a full dose, at night, and in smaller doses two or three times a day. He prefers the *liquor morphiæ bimeconatis*, but Battley's solution, or Dover's powder, or solid opium may be used instead.

The bowels must be carefully regulated; a plentiful supply of food does much to keep them open, and bran bread may be given with the same object. In Sir William Gull's case, to which I have already referred, four ounces of olive oil, and afterward six ounces, were administered each day, with the result that no obstinate constipation afterward occurred.

It is of great importance that melancholic patients should be kept warm. Dr. Blandford remarks that, if not carefully watched, they are very apt to get out of bed and to lie all night on the floor. It is of great importance that this should be prevented, and he recommends that when other means fail mechanical restraint should even be employed. Warm baths are often beneficial in this form of insanity, as are also hot-air or Turkish baths. The rooms in which patients suffering with melancholia are placed should be sunny, and their clothing should be warm.

Moral Insanity.—We have seen that most cases of insanity are at their commencement characterized not so much by disorder of the intellectual faculties as by a change in the disposition and character of the patient. In the language of modern psychologists, the "affective life" is perverted before the "ideational." This is particularly marked in melancholia. But certain writers have maintained that there is a particular form of mental disease which essentially consists in an alienation of the moral feelings, the intelligence remaining unaffected; and two varieties of it have been described, of which

one has been called "emotional" or "moral" insanity, the other "instinctive" or "impulsive" insanity.

Now, in this brief sketch I am anxious, as far as possible, to keep strictly to the medical aspects of the subject, avoiding altogether those which are legal, but the theory of moral insanity derives its whole significance from the doctrines which have been laid down by lawyers. In regard to all medical questions the views held by non-professional thinkers, who have themselves no practical acquaintance with disease, are apt to clash with those held by men who possess such an acquaintance, and this is especially likely to be the case if the former class of persons deliberately ignore and refuse to study the writings of the latter class. But I believe that it will almost always be found on inquiry that the opinions held by laymen at any period are really those of a former generation of medical men, which had been cast aside and abandoned as science advanced. Now, we have not to go very far back in the history of the knowledge of mental disorders to reach a time when lunatics were supposed to be, like brutes, bereft of all reason and intelligence. That such persons should escape punishment for crime was a matter of course—they were believed to be unable to distinguish between right and wrong. Afterward, however, it became apparent that many persons were insane whose intellectual faculties were by no means thus completely extinguished. Then arose the doctrine of "partial," as distinguished from "total" insanity, which was, for instance, laid down by Lord Hale. But it is important to note that Lord Hale did not regard partial insanity as a bar to responsibility in criminal cases. All modern psychologists, however, are agreed that the mind cannot be so divided into separate parts as that a man should be sane in regard to one set of ideas but insane in regard to others. It has followed from this that if insanity can be proved in reference to any one point, the patient must escape punishment. Now, the most obvious indication of insanity is certainly the presence of delusions. And it is not surprising that the lawyers should have fallen back upon this when they found in course of time that their former position was untenable, and that they could no longer insist on a want of knowledge of right from wrong as the criterion of freedom from responsibility. But even before they have settled down into their new quarters, psychology has advanced yet further. Those who are engaged in the practical study of mental diseases have recognized the fact that however conclusive as to the existence of insanity the presence of delusions may be, they and the actions based upon them nevertheless form but a very small part of that change of character and disposition and conduct which really constitutes unsoundness of mind. So far they are undoubtedly right, but at the next step they seem to have wandered from the true path. Knowing that delusions have been regarded as the essential indications of disorder of intelligence, they have failed to see that their own more accurate appreciation of the true character of insanity required that such an opinion should be reconsidered. And, instead of their maintaining that the intellect may be gravely altered without delusions being present, the doctrine which they have formulated is that insanity may exist without any impairment of the intellectual faculties. This, for instance, was the view laid down by Dr. Prichard, who (I believe) was the inventor of the term "moral insanity," which has since led to so much discussion. That writer illustrated his opinions by a series of cases, which have been criticised *seriatim* by Dr. Blandford, and I certainly agree with the conclusion to which he arrived, namely, that even if some of the cases may be said to be examples of insanity without delusions, there are none in which the intelligence can be said to be perfect. It is absurd to say that "the reasoning faculty remained unaffected" in persons who not merely exhibited a complete perversion of all their natural feelings and affections

and propensities, but committed all kinds of outrageous and filthy actions, for which no motives could possibly be assigned that would influence reasonable beings.

When once we have given up the arbitrary notion that the presence or absence of delusions determines whether the intellectual faculties are or are not impaired, there seems to me to be no basis whatever for the doctrine that "moral insanity" is to be regarded as a separate form of mental disorder. But it must necessarily be that the cases in which no delusions are to be detected, are generally those in which the existence of insanity is most apt to be doubtful or difficult of proof. And one must also bear in mind that the doctrine that lunatics are irresponsible for crime is not necessarily applicable universally in the new aspect of the subject. This question seems to me to be, after all, one of practical expediency rather than of principle. One must remember that, according to the present conception of insanity, it is a perversion of the cerebral functions which may vary infinitely in degree of intensity, and which necessarily shades off into sanity by imperceptible gradations. It seems absurd to say that of two persons separated by an imaginary boundary line, one is absolutely responsible for everything he does, while the other is accountable for nothing. At the same time it must be admitted that lunatics do not seem to use the knowledge that they will escape punishment as an encouragement to them to perpetrate crimes. It is a striking fact that those who have the charge of asylums never dream of invoking the aid of the law to punish even the most violent madmen. They are able to protect themselves. Yet the inmates of such an establishment would only have to combine together to carry everything before them, at least for a time. The fact that they do not do so is surely a proof that even in what seem to be the milder forms of insanity the impairment of the mental powers is really greater than would appear from the positive symptoms that are present.

Again, it does not seem that there is any real necessity for the recognition of an "impulsive" or "instinctive" insanity. I have described elsewhere (see p. 720) some very remarkable forms of mental disorder, in which a person may carry out a more or less complicated train of actions while in a state which is so completely abnormal that afterward he has not the slightest knowledge of what he has done. Such instances are sometimes spoken of as examples of "dual consciousness," but the more correct statement appears to be that the actions which the patient performs are automatic and independent of his proper consciousness. But what I wish now to point out is that these are the cases, if any, to which the name of impulsive insanity is applicable. On the other hand, the cases to which this name is commonly given are simply cases in which persons in whom no delusions can be shown to exist commit criminal acts—generally acts of violence—for which no motive can be detected. It may be true that most of these persons, or all of them, are insane, but it is nevertheless wrong to suppose that theirs is a special form of insanity, or that the proof of it is to be based upon evidence different from that which is required in other cases. As Dr. Blandford points out, many of the acts of ordinary lunatics are impulsive in precisely the same sense. Those who all day long break windows, or tear their clothes or bedding to pieces, can give no reason for what they do. And when a motiveless crime is committed by a person hitherto supposed to be sane, the only way to settle the question whether he is insane is by inquiring into the circumstances of his previous history and of that of his relations, and by careful scrutiny of his subsequent conduct. There may be cases in which the utter senselessness of a criminal action, and its being entirely inconsistent with the previous character of the person who committed it, suffice by themselves to stamp its author as insane. But such

cases, if they occur, instead of being examples of a special form of insanity, are simply instances in which the insanity happens not to have manifested itself fully in the ordinary way. And, as a matter of fact, Dr. Blandford states that an analysis of the cases of so-called "homicidal mania" hitherto recorded, does not yield a single instance in which there was not other evidence of general mental disease.

Chronic Dementia.—In speaking of mania and other forms of insanity I have had occasion to mention that each of these diseases may, in course of time, lose its more distinctive characters, and the patient pass into a state of mere imbecility. A precisely similar condition often occurs in old persons as a mere result of age, and in younger ones as the sequelæ of permanent hemiplegia, or of various organic cerebral diseases. Writers sometimes call it "secondary dementia," to distinguish it from the "primary dementia" that has been described at p. 753. But for the senile cases this designation is inapplicable.

It may be a question whether one is right in dealing with all the forms of chronic dementia among functional diseases of the brain. Dr. Major states in the "*West Riding Asylum Reports*" that in all the senile cases which he examined he found histological changes. The nerve cells in the cortical substance are commonly atrophied or in a state of granular degeneration, and their branches are to a greater or less extent destroyed. The vessels are generally dilated and the channels outside them enlarged, and granules and crystals of hæmatin are deposited upon them, but there is no great proliferation of their nuclei (as in general paralysis). It is probable, however, that some at least of these changes are the mere result of old age, apart altogether from the presence of any special symptoms of mental failure. This, I believe, is certainly the case with the more obvious pathological appearances that such cases present; namely, thickening of the membranes, increase of fluid in the meshes of the pia mater, and shrinking of the convolutions.

The most striking symptom of dementia is impairment of memory. This varies infinitely in degree. In the most extreme cases the patient may not even know his own name or that of his nearest relations. He may not be able to say where he is nor how long he has been in the same place. Or he may be clear on these points, but unable to give the slightest information about his affairs. Very often he retains a full recollection of the events of former years, and particularly of his early life, and yet he cannot remember the occurrences of a few hours ago. If a relative should come to see him he may forget all about his visit before the end of the day. He may, perhaps, say that he has a balance at his banker's, and that he draws checks from time to time, when he has not really done so for many years past.

The appearance of a person affected with this form of mental disease is dull, vacant and torpid; he often looks much older than he really is. His attitude is helpless and his gait shuffling and feeble. He may be fond of standing or crouching in some one corner, or of walking backward and forward for a certain distance on a particular strip of ground, or he may be constantly running to and fro as if looking for something; or hopping, dancing, or gesticulating. His disposition is generally to be gay and happy; he is prone to laugh and to chatter; he may appear amused in the midst of the saddest circumstances; he is incapable of feeling any emotion, whether of love or of hate; and he may be indifferent to privations and even to pain. Sometimes he takes pleasure in collecting stones or pieces of paper or sticks; or he may amuse himself with toys, or be fond of ornamenting himself with feathers or flowers. Certain patients, however, are gloomy, depressed, and inclined to weep; and others are mischievous and spiteful.

In some cases there are the most extraordinary hallucinations or illusions. Dr. Maudsley speaks of one such patient as lovingly nursing a lump of wood decked in rags, and of another as busily engaged in spinning threads out of sunbeams. The conversation is often indecent, or at least devoid of all delicacy. Not infrequently the same sentence or the same question is repeated over and over again. The habits are often dirty, the fæces and urine being passed in the bed at night and in the clothes by day.

But not infrequently hallucinations, illusions, and delusions are all alike wanting in cases of dementia. And lawyers are apt to make a great point of this if the will of such a patient should be disputed after his death—the way in which this form of insanity most commonly becomes the subject of medico-legal inquiry. I need not here repeat what has been stated at p. 760, to the effect that legal authorities have been disposed to lay far too much stress upon the presence of delusions in comparison with other indications of impairment of intelligence, of all of which failure of memory is the most important. It is not to be denied that in the less marked forms of dementia the question of testamentary capacity is often exceedingly difficult to determine.

Dr. Blandford observes that one may ask one's self whether the patient would be able to shift for himself, to take a lodging or house, to come and go unattended, or to pay his accounts. If not, he must be allowed to be unsound of mind. But this writer goes on to say that the difficulty is greatly increased in the case of ladies, since many of them do not manage their own affairs even when they are well.

The bodily health in chronic dementia is generally good; there is often a tendency to corpulence. A point on which Dr. Blandford lays much stress is the importance of being prepared for the supervention of the various visceral diseases in cases of this kind. Phthisis, in particular, is apt to develop itself without the patient complaining of pain or cough or anything else. The only observable symptoms may be that he appears out of sorts, sits listless and dejected, and does not eat as he is wont. The state of the appetite affords a better test of the condition of such persons than any other. If there be frequent sickness, hernia must be thought of; and when the urine dribbles away, the state of the bladder should always be carefully watched, lest it should become over-distended.

The prognosis in chronic dementia is always unfavorable, but the patient may remain for many years without any alteration in his state. Removal to an asylum is by no means absolutely necessary, although persons suffering under this form of mental disease require constant and watchful care; and when kept in private houses they are often sadly neglected. A good attendant may do very much to check dirty habits. Dr. Blandford says that it is only when the patient is very far gone, indeed, that he cannot be taught to relieve himself at regular intervals, and in a proper receptacle. Even when consciousness is lost, "accidents" (as they are termed) may be made the exception, instead of being the rule. The prevention of bed sores is another matter of great importance. Unless there be bodily illness, the patient should never be allowed to remain in bed all day; even if he is unable to walk, he should be washed and dressed and seated in an arm chair.

Ætiology of Insanity Generally.—I have already incidentally referred to most of the causes of insanity; but there are some which I have not yet discussed. One of these is *hereditary transmission.* Its importance is universally acknowledged, but different observers have formed widely different estimates of its frequency. It would appear that about one-third of all insane persons can be shown to have had one or more relations affected with

some form of mental disorder; a proportion which is, I believe, higher than for any other equally common disease, except phthisis and gout. But this is by no means the limit of the influence of inherited tendencies; insanity is also very apt to show itself in the members of families, where other members are drunkards or affected with various neuroses, such as epilepsy, hysteria, etc. The force of transmission is, of course, increased in the offspring of marriages of consanguinity; or when the parents, although belonging to different families, are both disposed to nervous complaints. Dr. Savage ("*Guy's Hosp. Rep.*," xxi, p. 70) mentions the cases of two brothers and a sister, all of whom became insane, their parents being, not indeed mad, but both of them odd and excitable. One of the most striking instances that I have read of in which mental disease occurred in several members of the same family is related by Dr. Blandford; out of nine brothers and sisters six showed unmistakable signs of unsoundness of mind. According to Baillarger, the father of a family is less likely than the mother to transmit insanity to the children; but the former hands the complaint down to boys and girls alike, whereas the latter more often has daughters who become insane than sons. As might be expected, children born subsequently to the occurrence of insanity in a parent are more likely to be attacked by the disease than those born previously. The same form of mental disease may appear in different members of one family in succession; or the forms may be different. Acute dementia is stated to be less often traceable to inheritance than most other forms of insanity; and we observed that the same is said to be the case with general paralysis. It does not appear that the prognosis is worse in those cases of insanity in which an inherited predisposition can be found, than in the rest. Dr. Blandford at any rate says that this is true of first attacks.

Some of the points mentioned in the last paragraph have a bearing upon the question upon which the medical attendant of a family is often consulted, whether a person who has a tendency to insanity is justified in marrying. The answer to such a question must, of course, depend in part upon the strength of the taint; in other words, upon the number of relatives who have been mad or affected with other nervous disorders, but it also depends in part upon the mental constitution and character of the individual whose marriage is in contemplation. In many cases one may have great difficulty in arriving at a right judgment. The doubt as to whether conjugal relations should be entered into is, of course, much stronger in the case of a person who has actually been insane. Dr. Blandford goes so far as to say that no woman who has been out of her mind ought to marry, at least until she has passed the child-bearing period. In the case of men he speaks less positively, not only because the risk of transmission to the offspring is less, but also because the probability of relapse is not increased in a man by married life to the same extent as it is in a woman under the several conditions of sexual intercourse, pregnancy, and parturition.

Among the less obvious causes of insanity, *syphilis* must not be overlooked. Dr. Allbutt has related some interesting examples of this ("*West Riding Asylum Rep.*," vol. iii). One is the case of a patient, a scholar and a man of the world, who became harassed and depressed by religious difficulties. He lay day after day in bed, having the Bible read to him, and reproached himself bitterly on account of former doubts. After a little time his ideas underwent a change, and he became affected with horrid sexual delusions. He lost all energy and manliness of character, and seemed likely to end his days in an asylum. No treatment did any good until after three or four months the administration of antisyphilitic remedies were commenced, and in three weeks his symptoms were removed. He had slight eruptions at the time, and he had been treated for secondary syphilis two or

three years before. In cases of this kind the predominant symptoms appear generally to be fear, depression of spirits, irritability, and melancholy. I may notice in this place that Dr. Allbutt also lays great stress on the frequency with which most patients complain of insomnia, waking up night after night after only one or two hours' sleep.

Idiocy.—In passing on to consider the diseases of the highest nervous centres which depend upon organic lesions it is natural that one should begin by referring to cases in which the affection is a congenital defect and in which the appearances that are found after death are due not to ordinary morbid changes, but to arrest or perversion of development. Those in whom such defects are found are commonly idiots or imbeciles, and they are studied chiefly by physicians who have charge of asylums for this class of patients. It is, however, a great mistake to suppose that such an institution as Earlswood receives only those whose brains are congenitally imperfect. A large proportion of its inmates are individuals who were originally possessed of perfect intelligence, but epilepsy or other disease attacked them in infancy or early childhood and shattered their mental endowments. Again, injury to the fœtal cranium at the time of birth, whether from prolonged pressure against the maternal structures, or from the application of forceps, is often followed by failure of mind. Dr. Crichton Browne has related several instances of this kind in the "*West Riding Asylum Reports.*" All such are commonly included under the name of idiocy or amentia, but a strict pathology should distinguish them from those of congenital malformation of the brain. In some of Dr. Browne's cases, indeed, great deformity of the head was present, and the cerebrum was doubtless altered in shape to a corresponding extent. But when failure of intelligence follows epilepsy or some febrile disease in childhood, it is not to be expected that even the slightest structural change should be found, any more than in ordinary insanity; such patients are, in fact, properly to be called insane children rather than idiots.

I am not sure whether true congenital idiocy is not always traceable to evident structural defect in the nervous centres. Writers have described them as normal in some cases, but I do not feel confident that they have been careful to distinguish the other conditions under which mental defects in children may arise.

Morbid Anatomy.—One of the most remarkable malformations is that in which the brain is exceedingly small, the hemispheres being particularly ill developed and forming a very low arch. The brain may not weigh more than eighteen ounces in a case of this kind, even after adult life has been reached. The size of the head is very much less than normal in all directions, even the maxillary bones being imperfectly developed. Such idiots are commonly called microcephalic; they form a tolerably definite group, scarcely connected with the rest by any intermediate links. The museum of Guy's Hospital contains a model of the head and of the surface of the brain from a case of this kind. The convolutions are seen to be exceedingly few and simple, but hardly, if at all, smaller than in the normal brain.

In some cases, again, one hemisphere is much smaller than the other; and the defect is not limited to the convolutions, but is very marked in the basal ganglia, and it also involves the cerebellum and the spinal cord, but on the opposite side. Or it may be that particular portions of the brain are altogether absent. Thus the upper parts of one or both hemispheres may be wanting, the cerebral substance thinning off into an edge, above which the membranes form a sac filled with cerebro-spinal fluid, and continuous with the lateral ventricle. I once made a post-mortem examination in a case of this kind at the Evelina Hospital. Both sides of the brain were affected, so

that one looked straight down upon the corpora striata and thalami as soon as the membranes were divided. Heschl has given the name of parencephalus to this malformation, which, however, Griesinger believes to be due to intra-uterine disease rather than to a mere arrest of development. It is commonly attended with paralysis and contraction of the limbs on the side opposite to the affected side of the brain. The child under my care at the Evelina Hospital had all its limbs contracted. But it is remarkable how little evidence of defective intelligence there is in some of the less extreme cases of this kind. I once found the membranes of the right hemisphere forming a cyst which contained a large collection of fluid, and the floor of which was a thin layer of brain-matter roofing in the lateral ventricle. The patient, aged thirty-eight, had died of an accident a few hours after his admission into Guy's Hospital, so that no observations had been made as to his intelligence, but he had, at any rate, earned his living as a laboring man. Only a year before a case had occurred at the same hospital in which the greater part of one anterior lobe was hollowed out into a cavity measuring three inches by two inches in its diameters, and which communicated with the anterior cornu of the ventricle by a smooth opening. The patient, a man aged seventy-seven, had died of an operation for cancer of the lip. He had had no cerebral symptoms.

In the former of these two patients the corpus callosum was represented by only a few thin shreds. That this great commissure should have been so defective without the mental powers of the individual being markedly below the average accords with the fact that Sir James Paget and others have recorded cases in which a large part of it was wanting, apart from the presence of any other malformation, but in which no considerable failure of intelligence had been observed.

Another morbid change in the brain which has been found in some persons who had been regarded as idiots is a great enlargement of its substance, generally attended with induration, so that it feels tough, like gutta-percha, and cannot be broken down by a stream of water of considerable height. This is commonly designated hypertrophy of the brain; but in all probability it is really a diffused sclerosis, the neuroglia being the tissue in which an overgrowth takes place. Such a condition may be associated with rickets, but I do not know whether this is the case when the symptoms are those of idiocy. Hitzig (Ziemssen's "Handbuch," xi, p. 761) relates cases in which it seemed to have been caused by injuries to the head. The skull is often greatly enlarged in these cases of so-called "hypertrophy;" the membranes are firmly adherent to the bone and the ventricles have their walls flattened against one another and are empty. The affection is apt to be mistaken for chronic hydrocephalus.

In many cases of idiocy, indeed, the lateral ventricles are really found much dilated, with thickening of the ependyma. But I think it is probable that this condition is secondary, and has no more claim to be considered the cause of the failure of intelligence than it has to be regarded as the cause of epilepsy or dementia when found in a person who had long been subject to either of these diseases. Indeed, a very large proportion of idiots are subject to frequently recurring epileptic fits.

So, again, I think it is most likely that unilateral atrophy begins at one spot in the hemisphere, and that the extensive changes which are found at the autopsy are really results of the continued failure of functions of all the parts physiologically related to the original seat of disease. A striking illustration of this principle is afforded by a case of Dr. Frederick Taylor's, in which I made an autopsy in 1876. A woman, aged twenty-seven, had had paralysis of her left arm and leg from the time when she was five years of age, having then been attacked with convulsions after scarlet fever. The left

arm was smaller and shorter than the right; the fingers had during life been contracted, but in constant motion. I found that the outer part of the right corpus striatum was atrophied and contained an ill-defined cavity. The whole right hemisphere was smaller in all diameters than the left; whereas the left half of the cerebellum was smaller than the right. These must evidently have been secondary changes ("*Guy's Reports*," xxiii, p. 16).

By Virchow, on the other hand, a theory has been elaborated in great detail, according to which premature synostosis of particular sutures of the cranium is the primary cause of a variety of structural defects in the brain. Cruveilhier is said to have found all the sutures closed in an infant at the age of eighteen months; and, according to Griesinger, it is by no means rare for many of the sutures to be ossified in children of three or four years. When this is the case the skull and brain must necessarily be small, and Griesinger goes so far as to say that it is the most common cause of microcephalus, the arrest of growth in the brain being a mechanical result of the pressure on its surface. But some microcephalic idiots have all the sutures open up to the normal period of closure or even longer, and the theory in question is clearly inapplicable to any cases in which the convolutions are of the natural size, but defective in number and in complexity. Moreover, it seems to me that great caution is required in drawing the conclusion that premature synostosis is the cause rather than the result of structural changes in the brain. In Dr. Taylor's case of acquired unilateral atrophy, the cranium was much thicker on the affected side than on the opposite one. And anything which prevents the due expansion of a part of the brain seems likely to be followed by a cessation of growth in the corresponding bones. I have no wish to dispute the correctness of the opinion that peculiarities in the shape of the skull in different individuals of the same race are immediately dependent upon variations in the order in which the sutures are severally closed. But this appears to be altogether a different question. It is admitted that narrowing of the cranium in one direction is very commonly compensated for by dilatation in an opposite direction, and that when this occurs to a sufficient extent, the deformity does not interfere with the development of the brain nor with the integrity of its functions.

Symptoms.—The symptoms of idiocy vary infinitely in degree. The cases might be arranged in an ascending scale, the bottom of which would come just above the position of an "anencephalous" fœtus, whose nervous centres are inadequate to the office of supporting its independent existence. Many idiots are unable to stand, and the power of speech is entirely absent in all the more degraded among them. Not only is memory altogether wanting, but they seem to have no idea of their own personality nor of the existence of an external world. They have to be fed and clothed and taken care of in every detail of their lives. If food is not regularly supplied to them, they may be unable to show that they feel the want of it by any sign beyond a general restlessness or a grunting noise. Those a little higher in the scale may move their lips and hands in some particular way, or weep until they get something to eat. A large class utter only monosyllables; and many of those who learn the meaning of certain words when used by others attend to them in a sort of mechanical way, without looking up and without change in the expression of their faces. The power of recognizing the attendants is, perhaps, the next step in the scale, and other manifestations of intelligence occur in succession until a point difficult to define is reached, when the defect is said to be rather imbecility than actual idiocy. One curious circumstance is that those who are exceedingly deficient in all other respects sometimes possess one faculty in a very high degree of perfection. Thus, it is not uncommon for idiots to have a remarkable memory for places; at Earlswood there is a most beautiful model of a man-of-war, made by a person whose mental

capacity was very small, and who, in particular, had no idea of numbers. Some clue to the explanation of these peculiarities is afforded by a case of Morel's, of a speechless idiot who had a special gift of playing on the drum; his grandfather had been drum major and his father a drummer, while a brother had always wished to follow the same occupation.

Another remarkable fact is that, according to Griesinger, the supervention of an acute disease sometimes leads to the display of faculties until then dormant. Nièpce relates that an idiot who could only articulate a few words was seized with hydrophobia, whereupon he began to speak fluently about things which had happened long before, and of which he had at the time appeared to take no notice.

The emotional tendencies of idiots are very different in different cases. Some are always gay and joyful, with laughing features and good-humored eyes; others are ill tempered and morose. Changes of temper are apt to occur in them without adequate cause; one idiot may show a joyful excitement whenever he sees a piece of paper, another may manifest the most tender regard for one particular child, taking no notice of the rest. Many of them are in the habit of continually repeating some special movement; they are always rocking themselves, or swinging to and fro, or shaking their heads, or puffing and blowing with their mouths. Griesinger mentions one idiot who was every day and all day long pushing a drawer in and out, and rattling two keys.

The senses of smell and taste are altogether wanting in many idiots; they may eat nettles or excrement. Their hearing is often dull, but their sight is comparatively seldom impaired, although they are very apt to squint. The cutaneous sensibility may be very defective; there may even be actual anæsthesia, both for pain and for impressions of cold. Paralysis with contraction of one or more of the limbs is often present, or ordinary "club foot." The sexual functions remain undeveloped in all the more severe forms of idiocy; the genital organs are small and fail to undergo the changes indicative of puberty; menstruation may never occur, or not until after the twentieth year; in exceptional cases, however, the catamenia are normal, even though the idiocy may be extreme in degree. The body and limbs are commonly more or less stunted. The tongue, on the other hand, is apt to be thick and fleshy, and saliva may be constantly running from the mouth.

The less severe forms of congenital mental deficiency which receive the name of Imbecility vary infinitely in degree, and can hardly receive formal description, Some instances may be set down to a sort of retardation of growth: a child twenty years old resembles a child of ten or twelve, and so on. Assiduous instruction effects great improvement in cases of this kind. Others show instinctive tendencies to mischief, lying, theft, or cruelty, which cannot be controlled. Many of these persons are weak-minded, but some can hardly be shown to be deficient in intelligence, so that they are often said to be affected with a "moral imbecility." They usually pass a large part of their lives in prison, and, indeed, many of them may be said to belong to the criminal classes by inheritance. Some observers have laid stress on arching of the palate, and on absence of a free lobule to the pinna of the ear, as being important indications of defect of development in cases of this kind.

The *causes* of true congenital idiocy are obscure. It is especially apt to occur in the children of parents who themselves present neurotic tendencies, particularly if the latter are blood relations, or of too early or too advanced an age, or intemperate in their habits. It is also frequent in families other members of which are stunted, or deaf-mutes, or epileptic.

The *diagnosis* of idiocy is usually easy. But cases occur in which

it is difficult, particularly within the first two years of life. Very commonly what first arouses the parents' suspicions is their finding that their child does not begin to speak; and I have had an infant, obviously idiotic in an extreme degree, brought to me that I might say whether or not it was tongue-tied, requiring the frænum linguæ to be cut through. One must ask whether it takes notice of and smiles at its mother, and whether it seems to know how to grasp at anything placed before it. Another point which Dr. West suggests as worthy of attention is whether it can support its head properly. But sometimes one acts wisely in not expressing a positive opinion at a first visit. It is possible to attach too much importance to the fact that a child does not begin to talk, even when a considerable time has passed. I have seen a boy who was said to have been unable to speak until he was seven years old; his mother told me that he tried hard, but could not say anything that could be understood. But at the age of fourteen he was able to earn money as a painter, and was said to be very clever and sharp at his work, although he had not even then learned to read or write, and was smaller in stature than a younger sister.

The *prognosis* of this disease is, of course, unfavorable, but great improvement may be effected by careful management. Many idiots who would be troublesome and spiteful at home become good-tempered and obedient in an asylum, and learn much more than would be anticipated.

Cretinism.—In certain countries there prevails endemically a form of idiocy which is peculiar in being constantly associated with certain remarkable peculiarities of cranial and bodily configuration; subjects of it are called "cretins." That the peculiarities in question are constantly present is not, indeed, admitted by every one. Even Virchow says that he has found the skulls of notorious cretins, or of individuals from cretinous localities, possessing all kinds of abnormal forms. But it appears to me that in all probability they were really common idiots, whose presence in such localities was merely accidental. I do not see how this supposition can be refuted, and it is certain that in the immense majority of cretins there is a special type of cranial development.

The origin of the word cretin is uncertain; some would derive it from *chrétien*, others from a Romänsch word, *cretira*, of similar meaning, and others from *creta*, in allusion either to the chalky complexions of those who suffer from the disease or to the geology of the districts where it prevails, which are of calcareous formation. The mental symptoms do not appear to differ from those of ordinary iodicy. As in that condition, there are all degrees of defect of intelligence. A Royal Commission on Cretinism which reported to the Sardinian Government in 1848, divided those affected with it into three classes, for which they proposed respectively the names of "crétins," "semi-crétins," and "crétineux."

As I have already observed, it is in their bodily configuration that cretins differ from ordinary idiots. Except in the very slightest forms, they are seldom more than four feet and a half in height, and often below three feet. They have large heads, especially in the direction from ear to ear. The features are broad and thick; the eyes are wide apart; the nose is very flat at the root, and spreads out enormously toward the alæ; the mouth is very large, widely open, with thick lips, and allows the saliva to escape. The forehead and cheeks are wrinkled and the skin is coarse and rough, so that they have always an appearance of old age. A cretin twenty years old is exactly like a cretin of forty. The hair comes low on the forehead and is coarse and bristly. Cretins have narrow chests, large bellies, crooked limbs. Their hands are broad but short, with short fingers.

The observations of Virchow with regard to the dependence of malfor-

mations of the cranium upon a too early closure of the sutures find an important application in the case of cretinism. That great pathologist has, in fact, shown that all the peculiarities in the configuration of the skull and face in this disease are the result of premature ossification of the basilar process. In a cretinous fœtus he found complete synostosis of the basi-occipital and basi-sphenoid bones, a continuous layer of diploë passing from one to the other, so that no trace of the original separation could be discovered. The basi- and pre-sphenoid bones were also fused together; but this is less important, because their union usually takes place soon after birth, whereas the basilar process and the sphenoid bone should remain separate until at least the fifteenth year. The completion of this "tribasilar synostosis" (as Virchow calls it) appears to be followed by the cessation of all further growth of the basis cranii in the way of enlargement in an antero-posterior direction. It also gives rise to a marked deformity in the shape of the internal base, the most constant features of which seem to be that the sella turcica is very narrow, and that the parts in front of the posterior clinoid processes form a greater angle than normal with the clivus or basilar process. Virchow found that the slope of the clivus was unduly steep in one fœtus. But at an early age a steep clivus is normal; and Nièpce has stated that in the cretins whose bodies he examined this part was horizontal; an observation which receives confirmation from a dissection of my own, to be presently referred to. It would therefore seem that the premature closure of the spheno-occipital suture, although it arrests growth, does not prevent the occurrence of some, at least, of the changes in the form of the base of the cranium which should take place in the years of childhood.

Laterally, the cretinous skull doubtless expands in its growth to an abnormal extent, so as to compensate for the deficiency in its antero-posterior diameter. This gives to the features of the cretin their peculiar breadth. The sunken appearance of the root of the nose is, I suppose, due to imperfect development of the septum from deficient forward growth of the vomer.

But if we admit that the configuration of the head and face in cretinism is directly attributable to premature synostosis of the basal sutures, it still remains a question whether this throws any light upon the ætiology of the disease as a whole. Some of the peculiarities in the build of the rest of the body may, indeed, possibly be due to a similar interruption in the growth of other bones. Thus Griesinger suggests that the shortening of the fingers and other parts of the limbs may depend on a too early ossification of the epiphyses of the several long bones. He even hints that from this point of view cretinism may be regarded as the antithesis of rickets.

The importance of such speculations, however, is trifling in comparison with another question which must now be discussed, namely, that of the very close relation which exists between endemic goitre and cretinism. In this country as well as in others, goitre prevails in many districts in which endemic cretinism is not met with. Indeed, so far as I am aware, there is no place in England in which the latter disease has ever been observed, except in the village of Chiselborough in Somersetshire; and Dr. Petherton, who in 1847 pointed out its presence there, tells me that it has since almost died out. On the other hand, I believe that there is no district in the world in which endemic cretinism occurs in which goitre does not likewise prevail. Again, it has been noticed that when a family migrates into a place where the two diseases are both met with, goitre is first developed in them; it is only in the second or third generation that cretins present themselves. These facts at once suggest the conclusion that, both diseases depending upon a common cause, the former arises where it has been in operation for a shorter time or with less intensity, the latter when it has been in operation for a longer time or

with greater intensity. We should therefore expect that individual cretins should have a goitre of the largest size. But observation shows that this is not the case. Many of the worst cretins have no goitre at all. According to the Report of the Sardinian Commission, goitre is absent in one-third of the cases of cretinism. Virchow has, indeed, pointed out that cretins have such thick necks that considerable enlargement of the thyroid may exist without being noticed. But this does little or nothing to remove the difficulty.

Sporadic Cretinism.—Now, it is a most remarkable circumstance that in England a form of cretinism sometimes occurs which also bears a close relation to affections of the thyroid body, but which is not peculiar to one part of the country rather than to others. In papers in the "*Medico-Chirurgical Transactions*" (1871) and the "*Pathological Transactions*" (1874) I have termed it Sporadic Cretinism. In this form of the disease there seems never to be a large goitre, but sometimes a slight swelling can be detected; or, as in the case of a boy who came to me from Halden, in Kent, a near relation (in that instance a sister) may have goitre. In the great majority of cases, however, the thyroid body, so far from being enlarged, is entirely absent, no trace of it being discoverable on dissection. This fact was first pointed out by Mr. Curling in 1850. In almost every other respect sporadic cretinism is identical with that which is endemic. Some of the figures which illustrate my communications on the subject (figs. 1 and 3, Plate III, "*Med.-Chir. Trans.*," liv, and fig. 2, pl. xii, "*Path. Trans.*," xxv) show how close is the resemblance between the two diseases in their general characters. Among the patients affected with sporadic cretinism that I have seen, one, at the age of sixteen years and a half, was only two feet eight inches high; another when twenty years old was only two feet four inches high. They have the same broad, short hands and short fingers, the same dry hair, the same rough, scurfy skin. Their heads are large and broad; their noses are flat at the root, so that the distance between the eyes is increased; their mouths are large and gaping, with thick lips. In the only case in which I have had an opportunity of seeing an autopsy (it was through the kindness of Dr. Grabham, of Earlswood) the base of the skull was much altered in shape; the posterior clinoid processes were at a much higher level than the anterior, and the sella turcica was exceedingly narrow. The clivus was horizontal, and the position seemed to be part of a general elevation of the occipital bone in relation with the other bones, for the cerebellar fossæ were exceedingly shallow. The line of union between the sphenoid and the occipital bones could no longer be traced, but as the patient was twenty-one years old at the time of death this, of course, had no significance.

The intelligence of those who are affected with sporadic cretinism is ordinarily very imperfect, and many of them present an extreme degree of idiocy, being even deprived of the power of speech. Their disposition is generally mild and tranquil. In this respect they seem to differ from the subjects of the endemic disease; for Griesinger says that the latter seldom present any agreeable traits of character; they are unsociable and repugnant to each other. Probably, however, much depends on the conditions under which such patients are placed, and the amount of care and interest bestowed upon them.

One of the most curious features of sporadic cretinism has still to be mentioned. It consists in the presence on each side of the neck in the "posterior triangle," outside the sterno-mastoid muscle, of a soft, lobulated, movable tumor. This on dissection is found to consist entirely of adipose tissue, which, however, may differ a little from the rest of the subcutaneous fat in appearance, being of a pinker color. In one of my cases, these supraclavicular swellings were much larger than hen's eggs. Their size seems to

be influenced to some extent by the state of the general health. In a case of Dr. Fletcher Beach's ("*Path. Trans.*," xxv) they were well marked when the patient first came under observation, but disappeared entirely before death, which was caused by exhaustion after protracted diarrhœa. It therefore seems possible that the poverty and unfavorable hygienic influences under which the subjects of endemic cretinism labor might prevent the formation of supraclavicular tumors in that form of the disease, even if they would otherwise make their appearance. It is, at any rate, certain that their presence has not yet been noted, unless a reference to them is included in a remark of Virchow's to the effect that there is sometimes a large development of subcutaneous fat in cretins, with leucophlegmatic swelling of the external soft parts. Is it possible that less skilled observers have confounded them with the goitre?

However this may be, it would, I think, be altogether premature to conclude that an absolute distinction between sporadic and endemic cretinism is afforded by the presence of those fatty tumors which (I may incidentally remark) are sometimes found slightly developed in adults who otherwise appear to be in good health. And, if the two forms of cretinism are in any way related to one another, we have a right to expect that the sporadic cases should throw great light upon the obscure pathology of the endemic disease. Now, I have failed to trace sporadic cretinism to any external cause. Dr. Langdon Down has suggested that it is due to alcoholic intoxication on the part of one of the parents at the time of procreation. None of my cases, however, lend any support to such a theory. I have more than once seen it in several children of the same parents. Thus I know of a family of twelve, three of whom are cretins of an extreme type, while the rest are healthy, and one has rowed in an Oxford and Cambridge boat race. They were all born in London, and their parents are in comfortable circumstances. The disease probably began to develop itself in them from the time of birth. But one of my cases of sporadic cretinism was that of a girl, who was stated by her relations to have been perfectly healthy until she was eight years old, when she fell ill with what was supposed to be a second attack of measles, and kept her bed for a fortnight. After her recovery her physical development underwent a remarkable change. Her features were previously well formed; they now acquired the cretinous configuration. Her hair, once black and abundant, became light-colored, dry, crisp, and very scanty. She ceased to grow; at the age of sixteen and three-quarter years she was only four feet one inch in height. Her hands and feet were of about the size of a child six or seven years old. She had a fatty tumor of the size of a hen's egg above each clavicle; and no trace of the thyroid body could be discovered in front of the trachea.

This case is very difficult of explanation. But, taken with the fact that the thyroid body is congenitally absent in so many cretins, it certainly suggests the idea that the febrile illness at the age of eight years led in some way to atrophy of that organ, and that this was the cause of the supervention of the cretinous state. I believe that deficiency of the thyroid body has never been observed in persons who were not cretins. An obvious inference is that its presence may be protective against the occurrence of cretinism; and, if goitre be to any extent a true hypertrophy, that the enlargement of the gland may *pro tanto* serve to augment the protective influence.

Thus I think that the true relation of goitre to cretinism is that they are antagonistic effects of the same unknown cause. This theory, which I suggested in 1871, seems to me to meet all the difficulties of the question—difficulties which are so great that some writers have absolutely refused to admit that the two diseases were in any way connected with one another,

except as a matter of coincidence. As I have already observed, I think that when the cause begins to act, or acts with but little intensity, the sole effect is goitre. But if it acts with great intensity, or upon successive generations, it at length produces cretinism as well as goitre. The not infrequent absence of goitre in cretins—when not dependent upon congenital deficiency of the thyroid body—must, I think, be attributable to some local morbid change in it, by which it is prevented from undergoing enlargement under the operation of the morbific agent. It is conceivable that when goitre has existed in a family for two or three generations, the structure of the thyroid body may undergo deterioration in members of the succeeding generation. The likelihood of the occurrence of such a local degeneration is perhaps augmented by the consideration that all families in which advanced cretinism is prevalent tend to undergo complete extinction within a very few years. According to the Sardinian Commission it is rare for any family residing in the Valpelline to reach a fifth generation. If it were not for immigration into this valley it would become altogether depopulated. Such a deterioration of structure in the thyroid body as I have supposed to occur under these circumstances would, of course, favor still more the development of cretinism, according to the theory which I have ventured to suggest. And I believe it is constantly the case that large goitres are present in the non-cretinous brothers and sisters of those cretins who themselves have no goitres, or only small ones.

I have just mentioned a case in which sporadic cretinism developed itself at eight years of age. Endemic cretinism is not known to commence so late as this.

Sir William Gull, however, has described ("*Trans. Clin. Soc.*," vol. vii) a very remarkable condition which he terms *cretinoid*, and which occurs in adult women. It is characterized by a change in the features, which become broad and flattened, the eyes appearing unduly wide apart, and the alæ nasi being thick, the lips large, the connective tissue below the eyes loose and folded, and that under the jaws and in the neck heavy, thick, and thrown into folds. The tongue is large, so as to interfere with the wearing of false teeth, and even to embarrass the articulation. The hands are broad and spade-like. The texture of the skin is smooth and fine, and it is of a delicate, rose-purple color. At the same time the disposition of the patient undergoes an alteration, activity and inquisitiveness of mind giving place to a gentle, placid indifference and languor. Through the kindness of Dr. Wilks I have seen one very remarkable case of this kind. It occurred in a young lady who was at one time fairly well looking, although not, perhaps, handsome. Her features underwent a most extraordinary change, and she became so hideous that the boys shouted at her when she showed herself in the streets. Her face became elongated; and her nose, lips, and mouth were enormous. There was not the slightest infiltration or hardening of the skin or subcutaneous tissue. She also had amaurosis, but this was perhaps an independent affection.*

[Since the above lines were written by Dr. Fagge, several additional cases of this remarkable affection have been recorded. I had one very striking case in a woman under my care in Guy's Hospital. She had the coarse, scanty hair, the sallow complexion with spots of bright red on the cheeks, looking as if she painted, the subconjunctival œdema, the broad

* [Dr. Wilks has kindly informed me, in answer to inquiry as to the sequel of this case, that the patient was twenty-eight years old, had suffered from neuralgia, and gradually became blind. Her features were swollen, and her hands could not be fitted with ordinary gloves. She died, after six years, "comatose," but at a distance from town, so that there was no autopsy. The urine did not contain albumen.—Ed.]

clumsy hands, thick blubber lips, slowness in answering, and tranquil temper. The contrast with a photograph taken ten years before was most remarkable. Though the "bladders" under the eyes looked ready to burst, acupuncture failed to bring out a drop of serum. Similar cases have been carefully recorded by Dr. Ord ("*Med.-Chir. Trans.*," 1878, "*Clin. Trans.*," xiii, 1880), by Dr. Dyce Duckworth ("*Clin. Trans.*," xiii, 1880), by Dr. Cavafy ("*Clin. Trans.*," xv, 1882), by Dr. Drewitt (*Clin. Trans.*," xvii, 1884), and others. In the first of these Dr. C. Charles found excess of mucin in the œdematous tissues after death, whence the name "myxœdema" has been proposed for this remarkable cretinoid condition in adults.

Two important experimental facts have added to those recognized and described by Sir William Gull in 1873. Prof. Kocher, of Berne, has found that after removal of the thyroid for goitre (so common in Switzerland) a condition gradually supervenes which much resembles adult cretinism. Prof. Horsley, of the Brown Institution, University of London, has produced, by the removal of the thyroid in monkeys, a state which in other symptoms resembles "myxœdema," and particularly in the large increase of mucin in the tissues. See his lectures reported in the "*Brit. Med. Journ.*," January, 1885.]

AFFECTIONS OF THE NERVOUS SYSTEM PRODUCED BY POISONS AND BY HEAT.

Plumbism—Mercurial Tremors.

Alcoholic Poisoning—CHRONIC: TREMORS, PAINS, ETC.—TREATMENT.—ACUTE: DELIRIUM A POTU—TREATMENT BY NARCOTICS—BY PURGES—URINE AND PULSE.

Heat-stroke—CARDIAC AND CEREBRO-SPINAL FORMS—MORTALITY—SEQUELÆ—DIAGNOSIS—TREATMENT.

MERCURIAL TREMOR.—Besides lead, of which the effects have been described above (p. 463), mercury is another poison whose continued action upon the nervous centres is capable of giving rise to certain peculiar and well-marked effects, which are commonly included under the name of Mercurial Tremor. The work people exposed to this affection have called it "the trembles." It is at present so rarely seen in England that a brief description of it will suffice.

The first thing which a man notices when he is beginning to suffer from mercurial tremor, is that he is no longer sure of his arms and hands. So soon as he attempts to take hold of anything they shake. After a time every effort leads to jerking movements. When his arm is bent it is not by a continuous motion, but by fits and starts. His hand cannot be directed with precision to any object, but is thrown to one or the other side of it; when he has even grasped a thing he is often unable to let it go. Presently the lower limbs are affected; he may have to be led, and when he walks his limbs may tremble and dance about, so that (as Sir Thomas Watson says) they look as if hung upon wires. His tongue may become tremulous, his speech may be hurried, jerking (or *staccato*) and at length unintelligible; this last symptom constitutes what has been termed *psellismus mercurialis*. When the tremor has once been excited by exertion or emotion, the man himself is unable for a time to stop it, but if he remains quiet for a little while it passes off, and does not return until he has to make some fresh effort. Getting some other person to grasp the affected limbs sometimes controls their movements.

The commencement of mercurial tremor is generally gradual, but sometimes it is sudden, and a fit of passion has seemed to act as an immediate exciting cause of it. It is said to be more apt to occur in the cold and damp weather of winter than at other seasons.

As might be expected, salivation often accompanies the more severe forms of mercurial tremor. The teeth become loose, the gums are ulcerated, the breath is fetid. The patient grows weak, anæmic and thin. His teeth are said to become black, and his skin has been described as acquiring a remarkable brown hue. After a time he may begin to suffer from headache and loss of memory, he may become delirious and at length comatose, and in this way the disease may terminate fatally.

The disease, in fact, tends to advance so long as the patient remains exposed to the influence of the poison, but when he is withdrawn from it the tremor commonly subsides in two or three months. Sometimes, however, the recovery is incomplete, the upper limbs being those which are most apt to remain shaky. Relapses are frequent, and each successive attack is commonly more severe than the previous ones.

The workers in quicksilver mines—in which the ore is exposed to heat—are said to suffer severely from mercurial tremor. Formerly it was common enough in this country among "water-gilders," who employ for gilding an amalgam of mercury and gold, from which the volatile metal is expelled by the action of a charcoal fire. But water-gilding is now almost superseded by electroplating. Another occupation in which mercury is used extensively is that of "silvering" mirrors; this, however, comparatively seldom gives rise to the tremor, probably because the quantity of metal volatilized is but small, since heat is not employed. But mercury does give off vapor to some extent, even at 68° or 70°, so that it is not impossible for the disease to show itself in those who have to deal with the metal at ordinary temperatures and under conditions which would seem to expose them very little to its action. In 1864 a man suffering from an extreme form of mercurial tremor was admitted into Guy's Hospital, under Dr. Rees. He had merely been engaged in *packing* up skins which had been previously washed with an acid solution of mercury. He said that he had at one time been salivated, but some doubt would, perhaps, have remained as to the cause of his symptoms, which terminated fatally, had not Dr. A. S. Taylor succeeded in obtaining mercury from the kidneys, and (in smaller quantities) from the brain and liver. This man had worked at his occupation for three years before he began to suffer to any considerable extent. It is generally said that a time varying from two to twenty-five years from the date of first exposure to the action of the poison is required for the production of mercurial tremor. Sir Thomas Watson relates the case of a man who had been a water-gilder for seventeen years before he was attacked.

There is reason to believe that the disease is sometimes due not to the inhalation of mercury, but to its absorption by the skin. Dr. Mapother states that the anatomy porter of the Irish College of Surgeons, who at one time rubbed enormous quantities of mercury into the bodies of persons affected with syphilis, was for thirty years subject to mercurial stammering. Even the internal administration of this remedy has been known to cause tremor in those who have taken it for a very long time.

The diagnosis of mercurial tremor is very easy, except in cases such as that which occurred in 1864 in Guy's Hospital, in which the patient might be unaware that he was exposed to the action of the metal. In its earlier stages it is liable to be mistaken for paralysis agitans; in its later periods for chorea. Anstie ("*Lancet*," 1872, i, p. 734) mentions the case of a man who was supposed to have died of delirium tremens, until it was afterward discovered that in his trade he had been inhaling the fumes of the acid nitrate of mercury.

Much has been done to prevent the occurrence of this disease in those whose occupations would otherwise expose them to it. The workshops are freely ventilated and provided with flues or chimneys. In some places it has been found practicable to interpose a glass screen between the upper part of the workman's body and the fire by which the mercurial vapor is set free. Personal cleanliness, frequent ablutions, and avoiding taking food in the room where the work is carried on, are regarded as very important; and it is said that abstemiousness in the use of alcoholic stimulants exerts a beneficial influence, though there may be a doubt whether the effects of intemperance have been isolated from those of other careless habits, which would be apt to prevail in conjunction with it.

Like other forms of tremor, that which is caused by mercury is often diminished for the time by a glass of wine or of some other stimulant. Sir Thomas Watson's patient said that when the disease was first coming on he found himself unable to get up stairs to his work until he had taken half a quartern of gin.

Among medicines iodide of potassium appears to be the most useful. It was suggested by Melseus on the same theory as for chronic lead poisoning, and mercury has been said to have been detected in the urine while it was being taken. Sudorific remedies have been recommended, but Sir Thomas Watson says that he found tonics, and particularly iron, more useful than any other drugs in the case which he relates. Anstie found cod-liver oil very useful in several cases. Sir William Gull some years ago recorded an instance in which electricity appeared to be beneficial, sparks being taken from the spine.

Alcoholism.—Until recently the action of alcohol upon the nervous centres was commonly supposed to consist mainly in the production of an acute disease which, from two of its principal symptoms, was called delirium tremens. But it is now known that a long-continued state of ill health is of far more frequent occurrence, and for this the name of chronic alcoholism has come into general use.

Chronic Alcoholic Poisoning.—Among the *symptoms* the most important is muscular tremor. This is more commonly noticed in the hands, which are unsteady and shake; but Anstie found that in a majority of cases the lower limbs were in reality affected before the upper. The man is often able, at first, to control the movements by an effort of the will. They are generally more troublesome in the morning than at any other part of the day, and they may render him unable to do any work requiring nicety of manual adjustment, until he has taken a dram, or eaten some food, by which for a time they are removed. Even before marked tremor occurs, the action of alcohol is sometimes manifested by a peculiar restlessness; the limbs are apt to start involuntarily and cannot be kept quiet except by an effort of attention. At the same time there is an irritable condition of the mind; the patient, even if he feels drowsy when he goes to bed, cannot sleep, but keeps turning from side to side.

At a somewhat later stage, other cerebral symptoms develop themselves. Anstie speaks of a buzzing or rushing sound in the ears as very common; and with it there is often a dull, diffused headache. Muscæ volitantes are often noticed and complained of. Flashes of light may seem to pass before the eyes, especially at night, just when the patient is dropping off to sleep. Momentary attacks of vertigo may often occur.

The intellectual and moral powers afterward become impaired. All certainty of purpose is lost; as Anstie says, there is a mental inquietude which makes it impossible for the patient to settle down to any ordinary occupation or to complete the tasks he may begin. He often has a vague feeling of dread for which he cannot account; or he may become the subject of some delusion, such as that an enemy is lying in wait to inflict an injury upon him. Anstie mentions, as another symptom, that the patient often has a vivid apprehension that he is in danger of falling down a precipice, even when walking on firm ground in broad daylight, and he says that this is of unfavorable augury.

Another occasional effect of chronic alcoholism on which Anstie insists in his work on neuralgia, consists in pains in the limbs, especially around the wrists and ankles, as well as in the shoulders and down the spine. They are somewhat paroxysmal in their character, returning each day at about the same hour, most commonly toward night; and they are greatly aggravated by fatigue, whether of mind or of body.

Impairment of sensation is also apt to occur, especially in the upper limbs; and the power of muscular co-ordination may be lost, so that the state of the patient is like that of a man affected with locomotor ataxy. Epileptiform convulsions sometimes show themselves; they are of the gravest augury.

The mental state may pass into one of mania or of melancholia, and ultimately complete dementia may develop itself.

Indications of gastric disorder are present at an early period. A common complaint is of nausea in the early morning, or even of actual vomiting, which may recur day after day as regularly as during early pregnancy. There is generally a failure of appetite, particularly at breakfast time. The tongue is foul, with a thick, yellow fur; or it may be red and glazed. The breath commonly presents a peculiar fetor, which is not that of any alcoholic drink, and which can hardly be described, although (as Anstie says) when once it has been smelled it cannot afterward be mistaken. The eyes are red and watery; the conjunctivæ are often jaundiced; the features look flabby and expressionless; the nose, cheeks, and forehead are often reddened, with crimson points and lines, corresponding with minute veins which have undergone dilatation. Sometimes the same parts of the face present a papular or pustular eruption, such as is commonly known by the name of "acne rosacea," or the nose may be enormously enlarged, with pendulous outgrowths from its alæ. However, it must not be understood that the appearances last mentioned are seen only in those who have indulged in alcoholic excesses; they are sometimes seen in persons who have been perfectly temperate; acne rosacea in particular, in women whose menstrual functions are disordered.

The limbs of a person suffering from chronic alcoholism are commonly wasted, especially the legs. The abdomen, on the other hand, is generally enlarged or pendulous, and its parietes, as well as the contained viscera, may be loaded with unhealthy adipose tissue. In those whose chief beverage has been beer, even the extremities may be covered with fat.

With regard to the chronic action of alcohol upon the nervous centres Anstie remarks that, contrary to what is the case with the digestive organs, the effects depend almost solely upon the quantity imbibed and not at all upon the form in which it is taken. There are, however, immense differences in the liability of different individuals to suffer from this poison. Some can drink spirits very freely for years and seem to be none the worse for it; others break down in health under comparatively small amounts of drink. Women, in particular, are apt to show a great susceptibility to its influence; perhaps because those of them who are intemperate commonly pass the whole of their time indoors.

Among the *antecedents* of intemperance must be mentioned, in the first place, those occupations in which persons are brought continually into contact with intoxicating liquors. A large proportion of the cases which present symptoms such as I have been describing is presented by men employed in breweries or distilleries, by public-house keepers and their wives, by travelers for wine and spirit merchants. Cab drivers and hawkers, and others who are exposed to rough weather, are also very apt to be intemperate; while for some individuals it is admitted as an excuse that their occupation is monotonous, deprives them of proper exercise, and keeps them confined in close, ill-ventilated rooms. Depressing mental influences drive others to drink; poverty, despair, and recklessness, from the failure of cherished hopes and anticipations, make them eager for the oblivion which intoxication brings in its train. Again, there are cases in which stimulants were at first taken for the relief of pain, but in which the habit of indulgence grows upon the patient until it becomes a disease. Anstie has rightly laid great stress on the responsibility which falls on medical men who allow women to take wine or brandy with the object of rendering them less susceptible to neuralgia or to the sufferings which are so apt to attend on the menstrual period or on lactation.

Lastly, a peculiar condition of the nervous system—capable of hereditary

transmission—is among the causes that lead to intemperance in drink. To a certain extent this view may be regarded as itself a neurosis. There is, indeed, a particular form of it which writers, following Roesch, have termed "Oinomania" [more correctly "Œnomania"]. The patient is seized every few months with a craving for drink, and for days together he behaves like a madman, taking long journeys without any purpose, or perhaps behaving indecently. But when the affection passes off—at the end of a month or so—he regains his usual health. He then lives soberly and chastely, and manages his affairs with activity and intelligence.

The *morbid changes* that are found in the nervous centres of drunkards are essentially atrophic. The convolutions are rounded and wasted, and the cerebro-spinal fluid between them is in excess. But the arachnoid is often opaque and thickened, and even the bones of the cranium may be dense and their diploë may have disappeared.

The *diagnosis* of chronic alcoholism may present every degree of difficulty, or it may be perfectly easy. Commencing general paralysis, locomotor ataxy, hysteria, and the nervous malaise which may result from dyspepsia, are, perhaps, the conditions most likely to be confounded with it. Unfortunately, one cannot place any confidence in the statements of the patient himself, however guardedly our questions to him may be put, for persons who indulge in secret drinking are almost always the victims of a moral cowardice which makes them untruthful even to their medical advisers. Anstie recommends that one should rather abruptly, but not uncourteously, hazard the observation that the diet must be strictly unstimulating; this, he says, will often cause the patient to betray the truth by his manner, or his air of surprised unconcern may prove that the suspicion is groundless.

In the *treatment* of a case of chronic alcoholism, the most important point is that the patient should henceforth abstain entirely from all intoxicating beverages. It is in such cases, above all others, that health resorts, like Ben Rhydding, are of service, where regular habits and exercise in the open air of the moors are enforced, while an unstimulating diet is provided and all facilities for the procuring of drink are withdrawn.

The administration of certain medicines may also be of considerable service. Anstie recommends especially quinine, which (if there should be sickness) may be given in a state of effervescence. Dr. Marcet's favorite remedy was the oxide of zinc; he says that in doses of from two grains upward, thrice daily, it has a powerful effect in inducing sleep. I have sometimes prescribed it with decided advantage, but Anstie does not speak very highly of it. Bromide of potassium often does very great good in cases of this kind. According to Anstie, half-drachm doses of ether with the same quantity of tincture of sumbul may do much to restore tranquillity to the nervous system and so to make sleep possible. As a more direct hypnotic, he recommends from a quarter to half a grain of the extract of Indian hemp. One should always employ the hypodermic injection of morphia (from one-tenth to a quarter of a grain) rather than give any preparation of opium by the mouth. Even in advanced stages, Anstie obtained striking benefit from the administration of cod-liver oil; and he says that when there is much tremor, strychnia is often very useful in a dose of $\frac{1}{48}$ to $\frac{1}{32}$ of a grain three times daily.

Some persons suffer for years from the symptoms of chronic alcoholism; even the hardest drinkers may reach old age without any other ill effects arising. They are, indeed, apt to become prematurely old, their hair turning gray and their arteries becoming rigid. Gout, chronic disease of the kidneys or liver, or fatty degeneration of the heart, may be developed in them, and cerebral apoplexy often cuts them suddenly off. Such diseases as pneumonia or fever are ill borne, and are very likely to prove rapidly fatal.

Acute Alcoholism.—But in many cases (as I have already mentioned) the nervous symptoms of alcoholism themselves take an acute form, and one that may quickly endanger life. They then constitute a disease which has for many years been known under the name of Delirium tremens or "delirium potatorum." The earliest account of it, I believe, was published in 1813 by Dr. Sutton, of Greenwich, but the first writer to describe its course in detail, when not disturbed by the administration of medicine, was Dr. John Ware, of Boston, in the United States; and his work, based on the observation of nearly a hundred cases, seems to me superior to any that has since appeared.

The patient has, perhaps, for two or three nights been more disturbed by unpleasant dreams than usual, and has by day been more restless and tremulous. At length he becomes altogether unable to sleep, but as he lies awake his dreams still haunt him. Next morning he may again be rational, but his mind almost always wanders occasionally. Toward evening the delirium returns, and the second night is worse than the first. During the second day there is again a slight amelioration as compared with the night that preceded it, but the mental disorder is now fully established and persistent.

The delirium in this disease is in many respects peculiar. The patient is not fierce nor violent, nor, on the other hand, is he depressed in spirits and dull in manner. He is loquacious and restlessly anxious to follow his accustomed vocation, but he sets about his business in a blundering manner, and his mind quickly wanders away to something else. If spoken to, he is sufficiently intelligent to answer, and for a minute or two he may converse rationally, but before long he starts off on some fresh topic. His friends probably endeavor to keep him in bed, but he is always wanting to get up and dress himself. Yet, if firmly opposed, he forgets his intention, at least for the time. He commonly has hallucinations of vision. Very often he fancies that rats and mice, or snakes, or cockroaches, are running or crawling over his bed, and he tells those about him that he sees them. Or he may address remarks to persons who are really absent, but whom he supposes to be in the room. He often looks suspiciously behind the curtains or under his pillow, or he stretches himself out of bed to see if some one is not concealed beneath it.

His hands are in constant motion. He picks at the bed clothes, or grasps at imaginary objects. If one asks him to put his tongue out, it is very tremulous, and is quickly withdrawn; it is commonly moist, and more or less thickly coated with a white fur. The pulse is quick, soft, and feeble. The skin is moist and often in a state of profuse perspiration. There is an entire absence of appetite and even of desire for drink. It was pointed out by Dr. Bence Jones that the amount of phosphates in the urine is very greatly diminished.

The duration of these symptoms is not absolutely constant, but it is far more regular than most medical men suppose. The disease almost always goes on for two and a half days without showing any tendency to subside, and then between the sixtieth and the seventy-second hours it comes to an end. Toward this period the patient is very apt to show signs of exhaustion. His pulse becomes more and more rapid and feeble. His face, at first flushed, is now pale and haggard; his pupils are widely dilated; his tongue sometimes, but rarely, becomes dry and brown.

It is, however, precisely at this time that a favorable change is to be hoped for. Toward the end of the third night the patient commonly falls asleep. At first he is still uneasy and restless, his breathing is irregular, and after an hour or two he may wake up for a little while. He soon goes soundly to sleep again; his breathing is now slow and deep; a profuse

sweat breaks out over his body. After six or eight hours he awakes and he is tolerably relieved. In the next twenty-four or forty-eight hours he sleeps almost continuously, and after this his restoration to health appears complete.

In some cases, particularly in those who had been generally in good health, and in whom the attack has been the direct result of a debauch, it terminates earlier—perhaps at the end of twenty-four hours. In other cases it lasts beyond the specified time. Dr. Ware speaks of having once known it to extend to nearly six entire days. This is most apt to occur in those who have for a long time been habitually intemperate.

Delirium tremens has a marked tendency to attack the same patient over and over again—unless, indeed, he should make an entire change in his habits.

But the end of the disease is by no means always so favorable. Sometimes a sudden attack of convulsions occurs, by which the patient is carried off. Sometimes he becomes comatose, sometimes he falls back into his chair and dies unexpectedly, in a state of syncope.

So far as I know, delirium tremens never occurs except as the result of alcoholic intemperance. Sir Thomas Watson, indeed, says that it is met with in gamblers and speculators who are sober in their habits. But such a statement must be received with extreme caution, on account of the proneness to falsehood and deception which almost always become part of the character of those who give themselves to indulgence in stimulants. It is often exceedingly difficult to discover how intoxicating liquors are obtained. Women, in particular, will suborn their servants or attendants, or procure tincture of lavender or tincture of cardamoms from the chemist, or drink eau de Cologne.

At one time it was generally believed that, instead of the immediate exciting cause of delirium tremens being the alcoholic stimulus itself, the disease was most apt to occur in persons who, from whatever cause, had been suddenly deprived of the drink to which they had become accustomed. Now, there can be no question that before the attack the patient has sometimes left off drinking for a few days; and, again, that it does commonly show itself in those who have been kept for a time without stimulants, in consequence of their having broken a limb or received some other injury. But the conclusion is nevertheless incorrect, for many patients are attacked by delirium tremens as the direct result of a bout of hard drinking. And the experience of those who have the management of prisons and other institutions where abstinence is enforced, has demonstrated that this does not in itself bring on an attack, even in the most intemperate subjects. The facts which have been supposed to establish the contrary opinion are otherwise explicable. On the one hand, a dislike for stimulants is sometimes an early symptom, so that the reason why the patient leaves off drinking may be that he is already beginning to suffer from the disease. On the other hand, a sudden shock to the system is exceedingly apt to act as a direct exciting cause of delirium tremens on those who are predisposed to it by alchoholic intemperance. It is this which renders the disease so common in the accident wards of hospitals. Indeed, it often comes on within the first few hours after the patient receives an injury, when there could have been no time for the withdrawal of stimulants to be concerned in its causation. I have known of two instances in which delirium tremens followed railway accidents of so trifling a character that they were quite unlikely to have led to any modification in the accustomed habits of the patients. And it is also frequently observed as a complication of acute pneumonia, erysipelas, or some other febrile disease.

I have already mentioned that delirium tremens has been formally

recognized by physicians only during the present century. Dr. Sutton, however, mentions that Dr. William Saunders, a former lecturer on medicine at Guy's Hospital, had for forty years described it as distinct from phrenitis. Dr. Sutton had become acquainted with it when practicing in East Kent, where spirits brought in by smugglers could thus be had in great abundance at a cheap rate. Certain of the practitioners in that district, he says, had learned to treat such cases with opium; and this practice was attended with very marked success in comparison with that of others (including at first Dr. Sutton himself) who regarded the disease as an inflammation of the brain, to be combated by venesection, blisters, purging, etc. Even Sir Thomas Watson, in his classical work on medicine, approaches the subject entirely from this point of view. He speaks of once being summoned to a man who was supposed to be mad or to have brain fever, and of the necessity that one should be alive to the distinctive symptoms of delirium tremens. But at the present day the opposite mistake is the more likely to be committed, a case being set down as one of this disease when it is really one of acute mania or acute melancholia, which (it must not be forgotten) may themselves result from alcoholic intemperance. There is also reason to believe that acute meningitis is sometimes due to the same cause.

Dr. Wilks speaks of having seen general paralysis of the insane mistaken for delirium tremens on two different occasions by the same physician. And in the surgical wards of a hospital there is often great difficulty in distinguishing the latter disease from the effects of injury to the head. Again, fever or acute pneumonia may be overlooked, and a case wrongly set down as one of delirium tremens, particularly if the patient should have been intemperate. But in such instances it is, perhaps, really present as a complication. Moreover, we must remember that pneumonia may be altogether latent in drunkards. Dr. Wilks mentions rheumatic fever, in that form which is attended with cerebral symptoms, as particularly like delirium tremens in its superficial characters.

Treatment.—The observers who first distinguished delirium tremens from acute encephalitis obtained what seemed to them wonderfully successful results by treating it with opium, and until recently most writers have advocated this practice. Sir Thomas Watson, for example, recommends that three grains of solid opium should be administered as soon as the bowels have been cleared out by a moderate purgative; and that if at the end of two or three hours the patient should show no inclination to sleep, one grain should be given every hour afterward until the result is attained. Or, he adds, corresponding quantities of one of the salts of morphia may be prescribed, or of laudanum, or of Battley's liquor opii.

But this advice was based upon the supposition that the disease, instead of having a tendency to subside spontaneously, would run on and, perhaps, prove fatal unless the patient were made to sleep. Sir Thomas Watson expressly remarks that delirium tremens is not likely to be "healed with a *placebo* or by waiting upon nature." And it is clear that the physicians who laid such great stress upon the value of opium measured their success by comparison with cases of encephalitis healed by venesection, leeches, blisters, etc. Most moden writers endorse Dr. Ware's statements as to the natural course of the disease. I may mention especially Dr. Wilks and the late Dr. Anstie.

It is still, however, a question whether the course of the disease may not be abbreviated by hypnotic medicines. Anstie thought that this might be effected by chloral hydrate. He gave thirty grains for the first dose and repeated it in an hour if the patient did not sleep. He found that the patient almost always got two or three hours of sound repose, and some-

times much more. He was of opinion that in delirium tremens there is a tolerance for chloral, so that as much as ninety grains, or even two drachms, may be safely given in divided doses within twenty-four hours, at least for a day or two.

But Dr. Wilks is disposed to endorse Dr. Ware's opinion that the duration of the disease cannot be shortened by the administration of medicines to send the patient to sleep. He thinks that a certain lapse of time is required for the subsidence of the commotion, and that a severe attack must last at least three days.

If this view is correct, it would seem to follow that at the commencement of delirium tremens neither opium nor morphia should be given at all, or at least that their administration should not be repeated so as to cause contraction of the pupils. For, when hypnotic remedies are actively pushed, one is apt to find one's self, after two or three days, in a difficult position. The patient, instead of sleeping, may be as excited as ever. His pupils are perhaps reduced to the size of pins' points, and he has had as much opium as would kill two or three healthy persons. Under such circumstances—which are precisely those in which a consulting opinion is most likely to be sought —there can be no question that the proper course is to wait. The further administration of hypnotics is dangerous. Dr. Wilks says that he has many times seen persons suffering from delirium tremens sent to their last sleep by opium, and I fear that the same result followed the subcutaneous injection of morphia in one case that I saw some years ago. The fatal symptoms produced by these drugs in persons suffering from delirium tremens seem to differ from those to which they give rise in healthy individuals. At least the patient whose case I have just referred to became collapsed rather than comatose, and I have heard of other similar instances. I must, indeed, repeat what has already been mentioned, that the disease sometimes terminates by sudden collapse, even when no hypnotics have been given.

Another question, which has sometimes been raised when a patient has died after the administration of a large dose of opium, is whether one can infer that this could not have been the cause of death from the fact that a certain interval of time had elapsed. Thus, in a case that occurred to Sir Thomas Watson, that physician decided that the medicine could not have been concerned in bringing about the fatal result, because nine hours passed after the last dose (one of three grains) was given before the patient became comatose. This conclusion would be perfectly valid if the case were one of suspected poisoning in a healthy man, but I doubt whether it is so in delirium tremens, in which there is known to be a great tolerance for hypnotic medicines. Dr. Christison long ago pointed out that in persons actually intoxicated with alcohol the effects of opium are much retarded.

But whatever doubts may fairly be entertained as to the part played by opium in bringing about a fatal result in cases of this kind, there can be none as to the importance of treating delirium tremens so that no such question can be raised. We have seen that most patients do well even if no opium at all is given, and therefore the fact that many recover after having taken heroic doses is no justification for administering them.

This, however, does not settle the problem whether or not it is advisable to give safe and moderate doses of opium in the early period of delirium tremens, at a time when there is no reason to suppose that the case will be one of so grave a kind. To determine that point one requires to know whether the medicine, if it should fail to give sleep, can do harm in any other way; and, again, whether it is ever needed at a later period, so that one may be then debarred from using it by having employed it at the commencement.

But before we can answer these questions, we must know what is the class of subjects in whom delirium tremens is most apt to prove fatal. Now, Anstie speaks of old age as especially unfavorable as regards prognosis, and one has been always accustomed to think that for young subjects the disease ought to be unattended with danger in a first attack, and even in two or three subsequent ones; but Dr. Aitken quotes some statistical facts published by Dr. Macpherson, and based upon observations made in the General Hospital at Calcutta, and in them it is curious that the highest average mortality was in persons between twenty-five and thirty-five years old, being from 23 to 24 per cent. of those attacked, whereas the mean mortality at all ages was about 15 per cent. I find that among fifty-nine cases of fatal delirium tremens which have occurred within the last few years at Guy's Hospital, there were three in persons between twenty and twenty-nine years of age, eight in those between thirty and thirty-nine, ten in those between forty and forty-nine, eight in those between fifty and sixty, but what ratios existed between these numbers and the numbers of patients attacked by the disease during the corresponding periods of life I have not been able to ascertain.

The state of the *kidneys* affects the prognosis in delirium tremens very materially, uræmic coma being very apt to occur as a complication. The urine must be repeatedly examined for albumen and for casts; and the quantity passed each day must be noted. If the secretion should begin to fail, Anstie recommends hot fomentations and dry cupping to the loins, hot foot-baths, and the administration of half-ounce doses of infusion of digitalis every three hours. I may take this opportunity of mentioning that digitalis was recommended as a remedy for delirium tremens itself by the late Mr. Jones, of Jersey, who used to give enormous doses of the tincture, and clearly showed that digitalis is one of the poisonous agents for which there is a special tolerance in this disease. But I do not think that the success which he attained was measured against what is now known to be the natural course of delirium tremens.

Of even greater importance in reference to the prognosis of delirium tremens is the state of the *heart*. Anstie pointed out that besides feeling the pulse, one should carefully watch the first sound of the heart with the stethoscope. And he laid still more stress upon the use of the sphygmograph. An "irregularly undulating" character of the pulse wave—such as occurs in the typhoid condition—was found by him to be of the most unfavorable augury. In Anstie's article in the "System of Medicine" a tracing is given which was taken from a man, aged forty, who, after being delirious for nearly a week, fell into a sound sleep of six or seven hours' duration, and when he woke appeared to be so much better that a confident opinion was expressed that he would recover. Anstie's sphygmographic observation, however, led him to augur the worst possible issue, and about twenty-four hours later the case terminated fatally.

The symptoms which indicate approaching failure of the heart's action are chiefly faintness and lividity of the countenance. Free stimulation appears to be the only resource when they manifest themselves. If the stomach will retain ether this may be given in half-drachm doses. But, if not, Anstie recommended port wine, especially such as is old and contains volatile ethereal compounds in abundance. He speaks of giving an ounce and a half of such wine every hour. He also says that hot mustard plasters to the chest may help somewhat.

The liability for the heart to fail in delirium tremens renders it necessary that the patient should be well supported by nourishment from the very commencement of the disease. He may have milk if he will take it; but, if not, he must be compelled to swallow strong beef tea, beef tea jelly,

or soup. He may even have small pieces of under-done chop or steak, if he can be induced to eat them.

The administration of alcohol in moderate doses was formerly recommended in cases of delirium tremens as a routine practice; it was thought to aid in inducing sleep. But all those who have studied the natural course of the disease are now agreed that there is no proof of its acting in this way; and they point out that there are great objections to its use, since the patient's future depends entirely upon the effect which his illness may have in leading him to give up stimulants for the rest of his life.

Another matter of the highest importance is that the room should be kept cool and dark and quiet. The patient's friends, who commonly collect around him, must be sent away, one or two active men being alone left to watch him. The gas-lights are to be turned down, and in the daytime a dark cloth is to be hung before the window, unless the patient should be terrified by the hallucinations which visit him in the dark. If he should be very troublesome in wanting to get out of bed, the question must be entertained whether he should not be tied down. If an attendant can, by persuasion or by employing a little force from time to time, induce him to lie quietly in his bed, that is, no doubt, to be preferred. But it is far better that he should be kept in a recumbent position by a sheet folded across his chest and tucked well in, than that he should be violently held down and restrained for any length of time together by the hands and arms of those about him.

HEAT-STROKE.—That exposure to intense heat is sometimes followed by alarming or fatal cerebral symptoms is now well known. The affection is not uncommon in the hotter parts of India, and it now and then occurs during the summer in England. Technically it is called *insolatio*, but the more usual name for it is "sunstroke" or *coup de soleil*. All of these terms, however, are unfortunate in one respect, namely, in suggesting that the direct rays of the sun are required to give rise to it; whereas, the truth is that it often comes on at night, when the temperature is very high, and when a number of persons are crowded together, as among soldiers in barracks, or sailors and others on board ship. Sir Joseph Fayrer mentions it as occurring to the stokers in the engine rooms of the Red Sea steamers. I therefore prefer to term it *heat-stroke*.

As might be expected, this disease often attacks several men simultaneously or in rapid succession. Dr. Maclean speaks of having seen a great many soldiers of the 98th Regiment struck down, of whom about fifteen died on the spot, in taking possession of a steep hill in China, in 1842. But I do not find it stated that those who suffer from heat-stroke are ever more than a minority of those who are exposed at the same time to the sun's rays. This, of course, depends upon the fact that the disease is due to the failure of those natural processes which should enable the human body to resist the action of excessive heat. Dr. Maclean remarks that the closely-shaven heads of the Chinese bear the hottest sun without ill effects, although it is true that these strange people generally make vigorous use of their fans to keep up a free current of air about their faces. Sportsmen in India expose themselves to very high temperatures with an impunity that would be surprising, were it not for the care which they take to wear light, loose clothes, to protect the head and the spine, and to abstain from stimulants. Thus it is of the highest importance that soldiers and others who have to bear fatigue in very hot climates should be suitably dressed, and should have no accoutrements which can interfere with the play of the lungs. More than one observer has noticed that large and fat men are especially apt to be attacked. Dr. Maclean thinks that exhaustion from prolonged exertion is a

predisposing cause of heat-stroke. Other factors which are believed to be often concerned in its development in those who are not directly exposed to the sun are overcrowding and defective ventilation. It would seem to be more likely to occur when the air is loaded with moisture, since this must interfere with evaporation from the skin; but there was extreme dryness at the time of an outbreak which Mr. Longmore saw at Barrackpore. Europeans have been supposed to be more likely to suffer from the disease when they have been only a short time in India, but there is good evidence that natives are by no means free from liability to it.

Symptoms.—The phenomena of heat-stroke vary in different cases.

(1) There is a "cardiac" form of it. In this death may be almost instantaneous by syncope, the man falling down suddenly insensible and making only a few hurried gasping respirations. Dr. Morehead also describes milder cases, in which a sense of prostration and faintness are experienced, with vertigo, dimness of vision, dilated pupils and drowsiness. The patient can be roused by pinching him, speaking to him, or sprinkling his face with cold water. There is constriction of the chest, with sighing respiration, a sense of weight or sinking at the epigastrium, nausea, and sometimes vomiting. The face and lips are pale. The skin is generally cold and clammy, with the exception of the head, which is somewhat hot. The pulse is feeble, and it is generally slow. Under judicious management such cases often recover; but sometimes the pulse sinks, the breathing becomes more sighing and irregular, and death occurs, being, perhaps, preceded by convulsions. "Cardiac" cases seem to be met with only among those who are attacked in consequence of direct exposure to the sun's rays. When recovery takes place, it is complete, leaving no sequelæ.

(2) There is a "cerebro-spinal" form, of which coma is the principal feature. This often comes on gradually. It may be preceded by nausea and loathing of food, giddiness, congestion of the eyes, extreme debility, and (as more than one observer has noticed) an unusual frequency of micturition. "I cannot hold my water" is said to have been in a large number of cases the first thing complained of. Dr. Maclean speaks of the patients beginning with a wild shout of laughter, or with a delirious attempt to escape from an imaginary enemy; headache, he says, is seldom present. When heat-stroke occurs in a man sleeping in barracks, what draws the attention of his comrades to him is often his stertorous breathing. He is then found already insensible, with contracted pupils, deeply congested conjunctivæ, and a quick, sharp pulse. As far back as 1860 Dr. Morehead pointed out that the skin continued pungently hot to the close of these cases, and even for some time after death. More recently, since the introduction of the thermometer into practice, it has been shown that the condition is really one of hyperpyrexia; and, in consequence, the name of "thermic fever" has been proposed for them. Three instances of this variety of the disease occurred in 1876, at Bristol, and were fully recorded in the "*Lancet*" by Dr. Shingleton Smith and Dr. Fox. One of them was in a man, aged forty-five, who had been turning a winch on board a steamship, exposed to the full glare of the sun, from 8 A. M. The temperature in the shade was from 92.3° to 96°. At one o'clock he felt unwell, and began to talk incoherently, and to throw his arms and legs about. He had been drinking water freely. He was admitted into the hospital half an hour later, and he was then comatose, with pin-point pupils; the temperature was 107°; the pulse 160, weak and intermitting; the breathing labored; the face not flushed; the skin sweating. At twelve minutes past two the thermometer registered 109°. Under energetic treatment the temperature gradually fell to 100.4°; the pupils became normal and afterward dilated; but the pulse failed more and more until

it could not be counted, and at 7.40 he died. On the very same day, another man, aged thirty-five, was taken into the same hospital in a state of only partial consciousness, with general muscular tremor, soon followed by convulsions and opisthotonos. His temperature was then 110.2°; his pulse was too rapid and too feeble to be counted. He died twenty minutes later, the thermometer at that time registering 111°. Two days afterward, a third case was admitted at 2.30 P. M., in the person of a man, aged fifty-five, who had been driving a hearse, when he fell backward, and the reins dropped from his hands. He was comatose; there was tonic spasm of the muscles of the legs; the pupils were contracted, but sensible to light. The temperature at 2.40 was 106.4°; at 2.50 it was 107°. The pulse was 141; the breathing was stertorous and at the rate of 27 in the minute. Under treatment the temperature quickly fell; at 3.25 it was 102°, at 3.40 it was 100°. In this instance recovery took place. These three cases appear to have presented all the chief symptoms of "thermic fever," as it is described by those who are familiar with sunstroke in India.

(3) Dr. Morehead and others speak of a "mixed form" of heat-stroke, in which the symptoms are a variable combination of those of the other two forms.

When heat-stroke ends in the patient's death this generally occurs within nine hours, but sometimes not until toward the end of the second day. The average mortality of the disease is estimated at from 45 to 50 per cent. Fatal relapses, after recovery from incomplete coma, are not infrequent; cases which seem to be doing well require to be carefully watched with the thermometer until the skin becomes moist and cool. The only post-mortem appearances are those of congestion of the viscera generally. The blood is said always to remain fluid. At p. 620 I have spoken of meningitis as being occasionally set up by the sun's heat; but the cases to which I was then referring seem to be distinguished by there being an interval between the time of exposure and the onset of symptoms. The recovery from heat-stroke is often slow. In 1856 there was in Guy's Hospital a sailor who mentioned that four years previously, when crossing the equator, he had been attacked with the disease in company with another man, who died. He himself came to in a few hours, but he could not speak for a month afterward, and for a time he lost the use of his right arm and leg. From all these symptoms, however, he had perfectly recovered; and he was now suffering from renal dropsy, which at length proved fatal. At the autopsy Dr. Wilks found the arachnoid opaque and marked with white spots, the ependyma granular, and an excess of fluid in the brain. It is to be noted, however, that the vessels at the base were much diseased, so that the other morbid appearances may have been unconnected with heat-stroke.

It is well known that sequelæ of a very serious kind are not infrequent. Those mentioned by Dr. Maclean are "persisting headache, either fixed or shifting; a chorea-like affection of the muscles, generally those of the forearms and hands; epilepsy, particularly in those who have inherited a tendency to that disease, or have had fits in youth; mental weakness."

The *diagnosis* of heat-stroke is no doubt easy in most cases; but I think that this often is because the patient is known to have been exposed to a high temperature, rather than because the symptoms are in themselves distinctive. At least, Dr. Maclean seems to me to place far too much reliance on certain characters of the pulse and of the respiration, and on the state of the skin and of the pupils, in apoplexy, as serving to exclude that disease from consideration. Probably there need be no doubt as to the real nature of these cases in which there is hyperpyrexia from the very commencement. But this is not always present; and I should imagine that a person attacked on a very hot day, in India, with cerebral hemorrhage, or

embolism, would be exceedingly likely to have his case set down as one of "insolatio," even by good observers, especially if there should happen to be no obvious paralytic symptoms. It now often happens that patients tell one that their complaints all result from a "sunstroke" which they had in England during the previous summer; but such statements must, I think, be always received with great caution.

In the *treatment* of the disease the most important thing is the employment of cold. Ice may be applied to the head; a stream of iced water may be passed through coils of elastic tubing (which are now made for the purpose) in contact with the back and the chest; a douche may be directed upon the head and neck and chest for a few minutes at a time. If the patient can swallow, he should be allowed to drink freely. In the one successful case at Bristol (which, however, was less severe than the others) Dr. Fox, besides applying cold, injected a grain of quinine under the skin in each of five different places; and at the end of half an hour the patient took ten grains of quinine by mouth. Indian practitioners are agreed that venesection is likely to be injurious rather than useful.

In many cases, however, in which the circulation fails, blankets and hot-water bottles have to be applied instead of, or in succession to, the ice. And brandy or liquor ammoniæ (℥viij to ℥iss of water) may then be injected subcutaneously or into a vein. Dr. Maclean says that the application of a blister to the nape of the neck or to the shaven head may be of service.

When convulsions set in, the inhalation of chloroform is recommended.

For sequelæ of heat-stroke, occurring in a person living in a hot country, removal to a temperate climate appears to be the best treatment. Dr. Maclean states that at Netley there are always some cases of this kind, and that they are often very obstinate. He has seen long-continued counter-irritation to the nape of the neck, and a course of iodide of potassium relieve permanently some patients troubled with severe fixed pain in the head; but in other instances these measures have altogether failed. He gives a favorable prognosis to those who come home with epilepsy under such circumstances, as he has usually found the fits subside.

DISEASES OF THE RESPIRATORY SYSTEM.

AFFECTIONS OF THE LARYNX.

THE LARYNGOSCOPE AND ITS USE.

Laryngeal Paralysis—OF ONE OR BOTH RECURRENT NERVES—OF THE ABDUCTOR MUSCLES—OF THE ADDUCTORS—APHONIA—LARYNGISMUS STRIDULUS AND OTHER SPASMODIC AFFECTIONS—LARYNGEAL MALFORMATIONS.

Laryngitis—CROUP—ITS RELATION TO DIPHTHERIA AND TO SPURIOUS CROUP—ITS CAUSES, DIAGNOSIS, AND TREATMENT—ACUTE AND CHRONIC CATARRH—TUBERCLE—LUPUS—SYPHILIS—ŒDEMATOUS LARYNGITIS—PERICHONDRITIS.

Tumors—PAPILLOMA—POLYPUS, ETC.—SARCOMA AND CARCINOMA—FOREIGN BODIES IN THE LARYNX.

The diseases of the larynx, like those of some other parts of the body, have within recent times been made accessible, as they never were before, to accurate clinical study, as the result of the invention of a special instrument by means of which they can be almost completely investigated during life.

The *laryngoscope* was first introduced into medical practice in Vienna by Türck and by Czermak, between whom there arose a lively contest for priority in its discovery. The question has happily now become almost forgotten, but the main facts are, perhaps, still worth recalling. Türck, in the summer of 1857, began to examine his hospital patients with a laryngeal mirror, such as had been used in physiological researches a few years before by Manuel Garcia, a singing master, who read a paper on the voice before the Royal Society of London. Even this was not the first starting point of efforts to see the interior of the larynx in the living subject, for it had been attempted, though with but little success, by several other observers, including the younger Dr. Babington in 1829, and Mr. Avery in 1844. Türck himself suspended his operations in the winter of 1857–58 for want of sunlight, and he lent his mirrors to Czermak, who, setting to work with artificial illumination, became rapidly convinced of the extreme value of laryngoscopy for clinical purposes, and after publishing a paper on the subject in March, 1858, in the "*Wiener Medicinische Wochenschrift*," traveled over Germany, France and England, to make it more widely known.

I need not describe the laryngoscope, for it is now in the hands of every practitioner. In using it, the first thing is to secure a bright light, whether from an Argand burner, an oil lamp, the lime-light apparatus, or the sun itself. The patient is placed with his back to the source of illumination, the rays from which pass over one of his shoulders. The observer seats himself opposite, on a chair slightly more raised, and throws the light upon the lower part of the patient's face by means of a flat or slightly concave mirror. The patient is next made to open his mouth and to protrude his tongue, and this

is gently grasped by the observer with his thumb and forefinger in the folds of a small napkin, or given to the patient himself to hold should the observer require to keep both hands free. The light is thrown into the back of the fauces and kept steadily fixed there. In the meantime a stalked laryngeal mirror is warmed over a flame, so that its surface may not become dimmed by the moisture of the breath. To be sure that it is not too hot, the observer tests it by laying it for an instant upon the back of his hand. The patient is now instructed to go on breathing quietly and regularly, and to sound an "a" (as in fate) on rather a high note. This brings the fauces into a position advantageous for the introduction of the laryngeal mirror, which is held like a pen between the finger and thumb, and gently but rapidly passed through the patient's mouth until it reaches the uvula, while the stem lies at the angle of the mouth so as not to interfere with the entrance of light. The observer should learn to use the laryngeal mirror with the left hand as freely as with the right, because it is often necessary to have the right hand disengaged for some other purpose while the mirror is held in position. In traversing the mouth the instrument must have its face turned downward, and it must take a curved course, being kept close to the palate, and as far as possible from the tongue, contact with which would, of course, soil its surface. As it reaches the uvula it has to be tilted so that its face looks forward as well as downward. It has also to be gently pushed upward and backward, lifting the uvula and the velum on its back. While this is being done, the epiglottis and the interior of the larynx commonly become visible, being reflected from the surface of the mirror. If this is not the case, slight changes in its position or in its inclination generally bring them into view. It must not be moved about in the fauces, for such a proceeding is almost certain to irritate the mucous membrane and to set up retching or cough. As a rule, it should not be carried so far back as to touch the posterior wall of the pharynx, which in many patients is far more sensitive than the velum, but some persons bear the mirror perfectly well, even when it is made to rest on the pharyngeal surface. There is seldom any necessity, so far as purposes of diagnosis are concerned, for continuing a single observation for more than a very short time. Should it be found that the interior of the larynx is not completely visible, it is better to withdraw the instrument and to reintroduce it a minute or two later, when the patient has had a little rest.

It may well be supposed that the practical use of the laryngoscope is in some cases attended with difficulties. In this matter, as in all others, habit goes a long way. The student finds himself baffled, and fails altogether to obtain a view of the larynx, whereas the trained observer succeeds at the first attempt; and, on the other hand, the patient, who, when the mirror is first introduced in his mouth, thinks he cannot tolerate its presence, finds after a few trials that it is scarcely even uncomfortable. One trouble is with the tongue, which in some persons becomes arched upward, so as to interfere not only with the passage of the mirror, but also with the admission of light to the back of the throat. They must then be directed to practice before a looking glass until they can "make a wide throat." Or the tongue may be held down by a spatula, which, however, is very apt to cause retching. Another obstacle is sometimes the presence of enlarged tonsils narrowing the faucial space. This is best overcome by using a mirror small enough to be slipped in between them. In some patients, again, the uvula and the velum are so irritable that the slightest contact of the mirror causes choking, or retching, or cough. Dr. Morell Mackenzie then recommends that small pieces of ice should be sucked for fifteen or twenty minutes before the laryngoscopic examination is begun; this, he says, rarely if

ever fails to blunt for a time the sensitiveness of the mucous membrane. In yet other patients, again, who have affections of the throat or lungs, the introduction of the mirror seems at once to be followed by the entrance of a quantity of muco-purulent fluid into the fauces from below, notwithstanding the repeated use of a gargle. Or the uvula may be so long and pendulous that it curls round the under edge of the mirror, interfering with the view of the larynx, or soiling the reflecting surface. The way to correct this is to use a large mirror, so as to lift up the whole of the uvula. But the most serious difficulty of all is that which is sometimes caused by a large epiglottis, which hangs backward over the entrance of the larynx in such a way as to prevent anything else being seen beyond itself. In many cases this obstacle is easily removed by making the patient sound, or attempt to sound, the vowel *e* (as in *feet*) on a high note. The sound itself cannot actually be produced while the tongue is protruded, but Störk says that the effort to produce it is often sufficient to raise the epiglottis. Sometimes the interior of the larynx can be seen, in spite of a pendent epiglottis, if the mirror is placed rather lower in the fauces than usual and with a more vertical inclination of its surface, the patient's head being at the same time thrown far backward. But it may happen that all these plans fail. An attempt may then be made to raise the epiglottis by a curved sound brought into contact with its under surface. Fränkel, in Ziemmsen's "Handbuch," says that when the epiglottis has been raised by such an instrument it falls slowly back again, so that even after removal of the sound, one can obtain a glimpse of the interior of the larynx. But in most persons the under surface of the epiglottis is so sensitive that a choking sensation is produced as soon as it is touched.

The parts reflected in a laryngeal mirror retain their proper positions so far as concerns the side of the body on which they seem to lie: the left vocal cord is visible upon the left side of the mirror, the right one upon the right side. But in an antero-posterior direction they appear to be inverted in such a way that one might suppose one's self to be looking at the larynx from behind instead of through the mouth, as is actually the case. In other words, the base of the tongue and the epiglottis form the top of the laryngoscopic image, the arytenoid cartilages and the entrance of the œsophagus are at the bottom of it. It is to be observed that the appearance of the epiglottis varies widely in different persons. Sometimes little more than the edge of it is seen, sometimes a large part of its posterior surface, which has normally a bright red color, apt to be taken for morbid congestion. The rest of the laryngeal mucous membrane is of a paler tint, the vocal cords themselves being white and glistening.

Most of the affections of the larynx interfere with the natural performance of both of its two principal functions, the formation of the voice and the passage of air into and out of the trachea; and many of them are also attended with other symptoms, such as pain, tenderness, a peculiar cough, and dysphagia, as well as with varied and complicated laryngoscopic appearances. It therefore seems advisable to begin the description of laryngeal diseases with certain affections, of which some give rise only to an impairment or loss of the voice, others only to interference with the breathing, the results of examination with the mirror being correspondingly simple and definite. These are the paralytic and the spasmodic affections of the muscles of the larynx. Although, being of secondary importance to the specialist—they are usually consigned to the end of systematic treatises—they possess peculiar interest for the general physician, on account of their bearing on diseases of distant structures. Their proper place in this work should, indeed, be among the affections of the nerves or of the nervous centres, but,

for reasons of practical convenience, it is desirable to take them with other affections of the larynx.

Different writers classify laryngeal paralyses in different ways. Störk first discusses affections of the several muscles one by one, as they may theoretically be imagined to occur; afterward he passes to more complex forms, in which many muscles are involved together. Dr. Mackenzie arranges them according to their supposed seat in the bulb, in the trunks of the vagi, or in the various laryngeal branches of those nerves. For my present purpose it will be sufficient if I describe such forms of paralysis as are actually recognizable in clinical practice, pointing out for each of them as far as is possible the nervous lesions that give rise to them and the muscle or the muscles involved.

Afterward I shall give brief accounts of the spasmodic affections to which the laryngeal muscles are liable.

Paralysis of all the Muscles supplied by the Recurrent Laryngeal Nerve or Nerves.—Among the most frequent paralytic affections of the larynx, as might naturally be anticipated, is one which involves all the muscles supplied by the recurrent nerve, either on one side or on both. This, when unilateral, is sometimes spoken of as "hemiplegia of the larynx," but I think it is clearly unadvisable, as it is altogether without precedent, to employ the term hemiplegia for a local form of paralysis. If a special name is wanted, it would be far better to follow the analogy of the word ophthalmoplegia, invented by Mr. Hutchinson for a general paralysis of all the muscles of the eyeball, and to speak of "laryngoplegia" when the muscles of both sides of the larynx are affected, while the name of "hemilaryngoplegia" might be assigned to cases in which the paralysis is one-sided. It is true that the paralysis is not quite universal, since the crico-thyroid muscles must be supposed to escape. But it does not appear that any appreciable physiological action results from their contraction when the other laryngeal muscles are powerless. Moreover, Türck is said to have observed fatty degeneration and atrophy of the crico-thyroid muscle in a case in which the recurrent laryngeal nerve was alone affected, while the superior laryngeal nerve entirely escaped.

It will be necessary to describe separately the effects of unilateral and of bilateral paralysis of the recurrent nerve.

1. *Unilateral Recurrent Paralysis* is characterized laryngoscopically by a complete immobility of the corresponding vocal cord, whether the patient only continues to breathe or utters a vocal sound. The position occupied by the cord is usually what is termed the "cadaveric position"—the same as that assumed by the vocal cords in the dead body, when no muscular force any longer acts upon them—intermediate between those which may be usually observed during phonation and during inspiration. But sometimes the cord stands nearer the middle line, being drawn inward by the action of the arytænoideus muscle. The cord itself looks concave. The summit of the arytenoid cartilage is placed a little further forward and inward than that of the opposite cartilage, so that it looks larger in consequence of more of its hinder surface being exposed to view. When a sound is uttered, the opposite arytenoid cartilage moves further than usual, and the unaffected vocal cord is drawn up to and even across the middle line, until it may come close to the affected cord, so that the chink of the glottis is placed obliquely. At the same time the summit of the mobile arytenoid cartilage crosses in front of the cartilage on the paralyzed side. When paralysis has lasted a long time, the affected cord may be seen to be obviously atrophied, and may oscillate backward and forward as the stream of air passes over it.

The voice of a patient with unilateral recurrent paralysis is often much less altered than might have been expected. It is apt to be weak and more or less hoarse, and to break into a falsetto as soon as an attempt is made to speak forcibly. This last peculiarity was originally pointed out by Frank, but recent writers say that it is less constantly observed than was at one time supposed. A point to which Gerhardt has drawn attention is that when two fingers are placed, one on each side of the thyroid cartilage, while the patient is speaking, a more distinct vibration can be felt with one finger than with the other. There is not the slightest dyspnœa.

2. *Bilateral paralysis* of all the muscles supplied by the two recurrent nerves is characterized, laryngoscopically, by immobility of both vocal cords in the cadaveric position. It is to be noted, however, that the paralysis is not seldom incomplete on one side or on the other, in which case partial movements of one cord may, of course, be detected.

There is complete aphonia, the voice being reduced to a whisper. The patient is unable to cough or to expectorate at all forcibly. There is no dyspnœa, at least in adults. This is a point about which there was at one time some divergence of opinion, but it seems now to have been finally settled. Scheck has recorded a case in a boy of seven, whose breathing was in no way interfered with. Ziemssen, however, still says, in his "Handbuch," that in deep inspiration the cords may be drawn a little further inward than before, and that a stridulous sound may be produced by their oscillations or by those of the other laryngeal structures in the stream of air.

Diagnosis.—It must be borne in mind that immobility of the vocal cord, whether on one side or on both sides, is not in itself proof of the presence of a paralytic affection. As Dr. Semon pointed out in the "*Medical Times and Gazette*" for 1880, precisely the same laryngoscopic appearances may be the result of anchylosis of the crico-arytenoid joints. This fact had, indeed, been recognized to some extent by previous writers, especially in Germany. But it seems to have been generally supposed that a perichondritis, whether of the cricoid or of one of the arytenoid cartilages, before leading to fixation of the corresponding joint, must, almost of necessity, be attended with suppuration and with swelling of the overlying submucous and mucous tissues. Dr. Semon has insisted that all such changes may be absent, and that the affection may produce only a development of fibrous tissue which obliterates the synovial cavity and unites the cartilages firmly to one another. The causes of anchylosis of the crico-arytenoid joints will be discussed under the head of laryngeal perichondritis. It is, of course, only when the position of the affected arytenoid cartilages is such as to bring the cords into the cadaveric position that the case can be taken for one of recurrent paralysis.

Pathology.—The causes of paralysis of the muscles supplied by the recurrent nerve or nerves fall into two groups. On the one hand there may be *central* disease of the nuclei of the nerves of the eighth pair, on one side or on both sides, in the pons; the affection then commonly forms part of a bulbar paralysis or of a multiple sclerosis, or of some chronic lesion of the cerebro-spinal centres ascending from the cord. The paralysis is then usually bilateral. Or, on the other hand, the disease may be *peripheral*, causing interference with the roots or trunks of the spinal accessory and pneumogastric nerves near the base of the skull, or with the trunk of the pneumogastric after its separation from the spinal accessory or with the recurrent laryngeal nerve itself below its origin from the pneumogastric. As a rule, the paralysis is in such cases unilateral. Indeed, by far the most frequent cause of paralysis of the muscles of one-half of the larynx is aneurism of the aorta, in which case the affection is most often on the

left side. Aneurism extending to the innominate artery may, however, compress the right recurrent nerve. And, again, mediastinal growths of various kinds may interfere with the left nerve, while either the left one or the right may be pressed upon by goitrous or other enlargements of the thyroid body, or by cancerous tumors starting from the œsophagus. It is a further peculiarity of these last-mentioned peripheral causes that they not infrequently affect in succession both recurrent nerves, and produce a bilateral paralysis. Dr. Mackenzie has published a case in which there were two aneurisms of different parts of the aorta, one of which compressed the right and the other the left recurrent nerve. And in 1866 Dr. Bäumler recorded a very interesting example of a bilateral paralysis of the recurrent nerves, apparently due to the pressure of a large pericardial exudation. It is, however, a more remarkable fact that peripheral interference with a single vagus sometimes causes precisely the same result. This, I believe, was first pointed out by Dr. Bäumler in the "*Path. Trans.*" for 1872; two years later, Dr. George Johnson made it the subject of a very interesting paper, which may be found in vol. lviii of the "*Med.-Chir. Trans.*" The only possible explanation seems to be that an irritant action is conveyed upward to the bulb by the centripetal fibres of the affected vagus. Consequently, it is obvious that a lesion involving only the recurrent nerve and not the vagus itself must be incapable of producing the same effect, as was well shown by Dr. Semon in the "*Berl. Klin. Wochenschrift*" for 1883. The circumstance that the paralysis set up by such an irritant action is bilateral may be explained by the existence of a close connection between the nuclei of the two sides, as is postulated by Dr. Broadbent's hypothesis (see p. 501). But Lockhart Clarke further showed that some of the fibres of origin of the spinal accessory nerve (which, in reality, include the root fibres of the recurrent laryngeal) actually pass across the middle line, being derived from the opposite nucleus; perhaps, therefore, it is not inconceivable that disturbance of a single nucleus should cause a bilateral paralysis. Whether any organic change develops itself in the nucleus or nuclei is as yet uncertain; it seems as yet not to have been carefully looked for. The muscles on both sides become atrophied, as was clearly shown in Dr. Bäumler's case. I may, indeed, observe that there is no form of paralysis in which the resulting muscular atrophy is more obvious on dissection than in paralysis of the muscles supplied by the recurrent nerve. In unilateral cases, in particular, the contrast between the whitish-yellow, shrunken *crico-arytænoideus posticus* on the affected side and the red, fleshy muscle opposite to it is more striking than any similar condition that I have ever seen in other parts of the body. The recurrent nerve, also, below the point at which it is compressed, is greatly wasted and of a dull gray color.

The *prognosis* of recurrent paralysis depends upon its cause. In Bäumler's case, in which it was dependent upon exudation into the pericardium, the patient, whose voice had been reduced to a whisper, recovered quickly as the effusion underwent absorption; within four or five weeks from the time when he first became hoarse he could speak nearly as well as ever. As a rule, the disease which causes the paralysis is incurable, and the paralysis itself persists until death. A goitre, if this is present, should, of course, be actively treated. It is generally useless to prescribe strychnia or to apply galvanic or faradic currents.

PARALYSIS OF THE ABDUCTORS OF THE CORDS (THE CRICO-ARYTÆNOIDEI POSTICI).—Since recurrent paralysis is commonly the result of a morbid process gradually destroying the fibres of the nerve or the nucleus from

which they arise, it is natural enough that cases should be met with in which some only of the muscles supplied by it suffer, while others escape. But what is a very remarkable fact is that such incomplete forms of paralysis should invariably (so far as is at present known) affect one particular muscle, the *crico-arytænoideus posticus*, of which the function is to keep the cords apart during respiration, and especially to widen the space between them on deep inspiration. Indeed, within the last twenty years a good many cases have been recorded in which there has been paralysis on both sides of the larynx, and yet no muscle has been attacked except this one on either side. Gerhardt published the first example of such an occurrence in 1863, and an admirable lecture on the subject by Riegel may be found in the second volume of "German Clinical Lectures," edited for the New Sydenham Society, in 1877. But it is especially to Dr. Semon that we are indebted for an adequate appreciation of the special proclivity of the abductor muscles to become paralyzed. The reason for this proclivity is still obscure. When there is a nuclear lesion in the bulb, it may, of course, be accounted for on the supposition that the nucleus for the fibres to the abductors is really distinct from that for the other laryngeal muscles, just as we explain how in ordinary cases of bulbar paralysis the lower part of the face generally becomes affected while the upper part escapes. But the result is just the same when the disease is one which involves either the upper part of the vagus or the recurrent nerve. The only possible explanation then seems to be that the fibres to the abductors undergo complete destruction earlier than those to the adductors. Riegel, indeed, actually found in one of his cases, in which both the recurrent nerves were embedded in dense connective tissue, that although the majority of the fibres had undergone fatty degeneration, some still retained their normal structure. May we suppose that, on account of their relatively simple function, the crico-arytænodei postici have a smaller nerve supply than the rest of the laryngeal muscles, so that when the recurrent nerve is pressed upon there is in the case of the muscles in question no possibility for undamaged fibres to take up effectively the work of those which have been destroyed, whereas for other muscles having more varied functions and a richer nerve supply, such substitution readily takes place? During the meeting of the International Congress in 1881, Rosenbach pointed out that in paralytic affections of the limbs there is an analogous fact in the greater liability of the extensors and abductors than of the adductors to suffer. Perhaps, too, a further corroboration of such a view may be found in the circumstance, to which I shall shortly have to allude, that when the laryngeal muscles are affected with spasm, the abductors are invariably overpowered by the adductors. This is certainly altogether consistent with the hypothesis that the nerve supply to the latter group of muscles may be more abundant than that to the former.

The laryngoscopic appearances which characterize paralysis of a single crico-arytænoideus posticus are that the corresponding vocal cord lies more or less near the median line, and does not move outward as it normally should when the patient takes a deep breath. On the other hand, during vocalization it moves freely inward. Hence one must bear in mind that unless the state of the larynx is carefully inspected while the patient is not, as well as while he is, attempting to utter a sound, this affection will necessarily be overlooked. When both abductor muscles are paralyzed, the two cords lie nearer one another than they do in health. The degree to which they are approximated varies with the duration of the paralysis. In cases of long standing, they may lie so close together that during inspiration it is scarcely possible to perceive the slightest chink between them; during expiration they then recede slightly from one another. Riegel insists on

the fact that this extreme narrowing of the space between the cords is the result of a gradual contraction of the antagonists of the paralyzed muscles, exactly analogous to that which occurs in paralytic affections of certain muscles of the eyeball, of the face, or of the limbs. But in a case recorded by Feith it seems to have come on within a few days from the commencement of the paralysis.

Another factor in the production of such a very marked stenosis of the glottis during inspiration is, however, in all probability, a sucking in of the cords toward one another in consequence of the diminution of atmospheric pressure upon their lower as compared with that upon their upper surfaces.

In two cases seen by Dr. Semon, there was a modification of the usual laryngoscopic appearances; the cords were close together only along their anterior two-thirds, and diverged posteriorly so as to leave a triangular opening with its base at the inter-arytenoid fold. It is suggested that this depends upon a limitation of the paralysis to the outer fibres of the crico-arytænoidei postici, their inner fibres escaping. Rühlmann has, in fact, endeavored to show that each of these muscles consists of two portions, having different functions; the inner portions, he says, draw the arytenoid cartilages downward and outward upon the cricoid; the outer portions rotate the arytenoid cartilages upon their vertical axis.

Apart from the results of a laryngoscopic examination, *subjective symptoms* of paralysis of a single crico-arytænoideus posticus are altogether wanting. The patient's voice is, of course, perfect, and as there is plenty of room for the entrance of air, he experiences not the slightest dyspnœa, even on exertion. It is this fact which gives its special importance to Semon's observations as to the invariable occurrence of paralysis of this muscle as the result of a partial destruction of the recurrent laryngeal, or of the vagus nerve trunk, or root fibres, or nucleus. He has shown that there are a great many cases of aneurism, of mediastinal growth, of carcinoma of the œsophagus, and even of disease at the base of the brain, upon which a routine use of the laryngsocope throws as much light as does the routine use of the ophthalmoscope in some cases of cerebral tumor or of chronic Bright's disease.

On the other hand, when both abductors of the vocal cords are paralyzed, the symptoms may be of the most urgent and dangerous character. The voice is still unimpaired unless there is a coincident inflammatory affection of the larynx. But there may be the most distressing inspiratory dyspnœa, the air being slowly drawn into the chest with loud stridor, and the patient becoming livid, with cold extremities, and ultimately dying of suffocation. In such cases the laryngoscope is needed, not so much to distinguish the affection from other diseases of the larynx as from stenosis of the trachea or of the main bronchi. It is to be borne in mind that the mere fact that during inspiration the cords are drawn close together near the middle line is not of itself proof that there is any paralysis of the abductors; there may be merely a spasmodic affection of the adductors. What generally at once marks the difference is the way in which the dyspnœa begins. A primary spasm is sudden and rapid in its development; the contraction of antagonists which obstructs the entrance of air in cases of paralysis of the abductors comes on slowly. At first there is difficulty of breathing only when the patient makes some effort or exertion; gradually it becomes persistent and increases in severity. Another morbid condition from which paralysis of the crico-arytænoidei postici muscles has to be distinguished is an anchylosis between the cricoid and the arytenoid cartilages, in which the latter happen to be placed in such a position that the cords lie close together. One point which ought to serve as a criterion between these two affections is that in anchylosis the immobility is, or may be, absolute,

which should not be the case in paralysis. But Dr. Semon speaks of the diagnosis as sometimes impossible.

With regard to the *causes* of paralysis of one or both of the abductor muscles of the cords, we have already seen it may be either central, or due to some affection involving the upper parts of the vagi, or the recurrent nerves in their course. In the "*Pathological Transactions*" for 1882 a case is recorded by Dr. Whipham, in which a bilateral paralysis of the abductors was dependent upon implication of the left pneumogastric and recurrent nerves in the walls of a thoracic aneurism. In some cases this form of paralysis has followed diphtheria, and once it has been a sequela of facial erysipelas. Sometimes it has been ascribed to exposure to cold; and Dr. Mackenzie thinks it is not infrequently due to direct pressure upon, or irritation of, the fibres of the crico-arytænoidei postici through the anterior wall of the pharynx in swallowing. Indeed, a case by Ott is cited, in which it was the result of the impaction of a piece of meat for twenty-four hours, at the orifice of the œsophagus.

Prognosis and Treatment.—In some cases, such as those to which I have just been referring, as well as in others in which the ætiology remains altogether obscure, recovery takes place after weeks or months, without its being obvious that treatment has been directly concerned in bringing about this result. Much more often the affection remains incurable. Sometimes a large amount of relief to the subjective symptoms, with temporary disappearance of the attacks of dyspnœa, may be attained by the subcutaneous injection of strychnia ($\frac{1}{30}$ grain of the sulphate, gradually increased to $\frac{1}{10}$ grain, daily) or by systematic use of faradization. The proper method of applying the current is by a laryngeal electrode (which will be described at p. 800), with a flat, spade-shaped extremity that can be laid upon the pharyngeal mucous membrane, over the paralyzed crico-arytenoid muscles. It is, however, very important that one should not be content with a partial success from this or any other plan of treatment. So long as the objective signs of stenosis of the glottis continue, there is always the risk of the sudden supervention of a fatal attack of dyspnœa, as in a case recorded by Dr. Semon in vols. xi and xii of the "*Clinical Society's Transactions*," in which the patient's life was just saved at the last moment by tracheotomy, and by artificial respiration, continued for three and a half hours. The rule is, therefore, laid down by him, that the trachea should be opened without delay, as a measure of precaution, in every case of paralysis of the abductors, attended with considerable stenosis of the glottis, and with marked dyspnœa, unless by some other method one succeeds within a short time in bringing about not merely subjective relief, but also an actual enlargement of the opening.

Paralysis of the Adductors of the Cords.—There is, in every respect, the most marked possible contrast between paralytic affections of the abductors and of the adductors of the cords, respectively. They differ in their causes, in their symptoms, and in their course. A paralysis limited to the adductors is never due to organic lesions affecting either the fibres of the vagi or of the recurrent nerves, or their nuclei of origin. On the other hand, such an affection is not at all infrequent as the result of other causes, which I shall presently give, but which themselves seldom, or perhaps never, give rise to paralysis of the abductors.

The laryngoscopic appearances which accompany paralysis of the adductors vary somewhat according to the precise seat of the affection; for it is to be borne in mind that, instead of being (like the abductors) a single pair of muscles, the adductors consist of a group of muscles on each side of the larynx, any one of which may probably be paralyzed separately. These

muscles are classed together by Henle and Luschka under the name of the *sphincter sive constrictor rimæ glottidis;* and as in one important point their descriptions, both anatomical and physiological, differ widely from those which have generally been taught in English text-books, it is necessary for me briefly to allude to their views. The point to which I am referring concerns the pair of muscles which in this country are known as the thyro-arytænoidei, and which are commonly supposed to have the function of relaxing the vocal cords, thus acting as antagonists of the crico-thyroidei. Now, the German anatomists divide the muscles in question into two on either side, naming them the *thyro-arytænoidei externi* and the *thyro-arytænoidei interni*, respectively. The thyro-arytænoidei interni are described as a pair of prism-shaped muscles, each of which has one of it edges projecting into the substance of the corresponding vocal cord. Their function is to straighten and to approximate the cords in the act of vocalization. In other words, they co-operate with, instead of being opponents of, the crico-thyroidei; and they are sometimes spoken of as being the "internal tensors," the crico-thyroidei as the "external tensors" of the cords.

Now, if the thyro-arytænoidei interni are paralyzed, the effect is that when the patient attempts to speak, the cords, instead of being straight, are both concave, and enclose between them a narrow oval space. If the affection is unilateral, the space appears bounded by a straight and a curved line. The width of the space between the cords depends partly upon the pitch of the sound which the patient is trying to utter, being greater when the pitch is low than when it is high. For one must remember that every degree of loss of power of the vocalizing muscles may occur, from the slight possible paresis up to the most complete paralysis.

On the other hand, an isolated paralysis of the *arytænoideus* muscle causes the rima glottidis to gape posteriorly, between the two arytenoid cartilages, while the cords themselves meet perfectly. The laryngoscopic appearance is, then, that of a triangle behind, with its apex prolonged into the normal narrow chink. Pieniazck, however, writing for Störk in his work, says that in some cases the ligamentous and the cartilaginous parts of the rima form two continuous straight lines, one on each side, there being a triangular interval between, which has its apex in front at the thyroid cartilage, its base behind, beneath the posterior commissure.

Another laryngoscopic appearance sometimes observed is that the processus vocalis of each arytenoid cartilage forms an angle inward; it is believed to represent a combination of paralysis of the *thyro-arytænoidei interni* with that of the *arytænoideus*.

An isolated paralysis of the two *crico-arytænoidei laterales* is said by Pieniazck to produce in the laryngeal mirror a figure with an angle outward, corresponding with the processus vocalis on each side, the rima being quadrilateral and lozenge-shaped. Ziemssen, however, doubts whether such an affection of these muscles ever occurs.

If all the muscles forming the *sphincter rimæ glottidis* are paralyzed at the same time, the rima glottidis, when the patient attempts to speak, forms an oval space; but this is not bounded behind by the point of contact between the two processus vocales, as when the thyro-arytænoidei interni alone are affected; it extends backward between the arytenoid cartilages to beneath the posterior commissure.

Lastly, according to Dr. Mackenzie, it is possible to recognize laryngoscopically a paralysis limited to the *crico-thyroidei* muscles which, of course, differ from all the rest in receiving their supply from the superior laryngeal nerves. The appearance which indicates such an affection is said to be that the rima glottidis presents a wavy outline, the form of which is described in one case as having been subject to frequent variation.

It has still to be remarked that paralytic affections of some of the muscles forming the *sphincter rimæ glottidis* may be associated with spasm of others, and also that the exact seat of a paralytic affection may be different on the two sides of the larynx. One can easily imagine that it may sometimes be difficult, or even impossible, to determine the precise character of such cases.

Aphonia.—The main symptom in all cases of paralysis of the adductors of the cord is impairment of voice, extending from hoarseness up to the most complete aphonia, so that the patient may be utterly unable to speak except in a whisper. As to whether such a total loss of voice is ever the result of the isolated affections of individual muscles there seems, however, to be some doubt. Ziemssen says that this effect can hardly be produced even by paralysis of the *thyro-arytænoidei interni*, so long as the *crico-arytænoidei laterales* and the *arytænoideus* remain in action, and bring together the processus vocales of the two arytenoid cartilages. Paretic states of the various muscles give rise not only to hoarseness, but to an undue sense of fatigue in speaking or in singing, to an inability to maintain the voice for as long a time as usual, and to a want of power to call out loud or to shout.

On the other hand, it is a striking fact that many patients who have complete aphonia nevertheless continue to be able to cough, and also to sneeze, and that these reflex acts are accompanied with a laryngeal sound, which clearly proves that for this performance the cords can be perfectly well brought into contact. This is especially apt to be the case with hysterical women, who, in fact, are the most frequent subjects of paralysis of the adductor muscles. Dr. Mackenzie says that has met with such an affection in girls only eight or ten years old, but hysteria is by no means unknown at such an age. Phthisis is another disease in which a paralytic aphasia is of rather frequent occurrence. Dr. Mackenzie speaks of having in 1865 examined at the Brompton Hospital thirty-seven consumptive patients in whom the voice was affected, and of having found that in eleven of them "the affection was purely functional." Sometimes a paretic, or even a completely paralytic, state of the thyro-arytænoidei and of the transverse muscles follows an attack of laryngeal catarrh; it may then continue long after the mucous membrane ceases to show congestion. Dr. Mackenzie says that this frequently occurs in public speakers, and especially in clergymen. In other cases paralysis of the adductors of the vocal cords is regarded as "rheumatic," in the German sense of the word, *i. e.* directly dependent upon the action of cold upon the affected nerve twigs or muscles. Sometimes it is the result of over exertion of the voice, as in singers, and such cases are usually slight and transitory. But Dr. Mackenzie speaks of paralysis of the thyro-arytænoidei interni as being occasionally the result of an actual "sprain" of the muscular tissues in some undue effort at vocalization, and as then proving exceedingly intractable. Paralysis of the *transversus* (or of some of the other muscles) seems to be sometimes dependent upon gummatous or other lesions directly destroying the substance of the muscle. Dr. Mackenzie also mentions poisoning by lead or by arsenic as possible causes of a paralytic affection limited to one or more laryngeal muscles; a case which he cites, and which occurred in a painter, is described as one of complete loss of power of "the adductor of the right vocal cord."

It is a peculiarity of hysterical aphonia, which (as we have seen) is dependent upon paralysis of some or all of the adductors of the cords, that the patient is apt to suddenly regain the voice under the influence of violent emotion or shock. The recovery in such cases may be either transitory or permanent. Since the introduction of the laryngoscope it has become the

usual practice to treat such cases by the application of a powerful induced current to the interior of the larynx, and this leads to brilliant success. The method is as follows: One electrode is connected with a metal plate fastened upon a necklet which is put round the patient's neck so that the metal plate rests on the front of the larynx. The other electrode consists of a small metal ball or sponge fixed to the end of a long curved stem, which can be passed down into the space between the vocal cords. The stem of this "laryngeal electrode" is so constructed that it transmits no current until the end of it has entered the larynx; at that moment the operator with his finger presses down a key by which the circuit is completed. All observers seem to be agreed that the only way by which it is possible to count upon a successful result from this procedure is that of using on the very first occasion a current of great power, which is, of course, exquisitely painful, but which makes the patient almost involuntarily utter a loud articulate cry, whereupon the electrode is instantly withdrawn. If less than this be attempted at first, Dr. Semon tells me that electricity often fails altogether. Dr. Mackenzie speaks of having cured cases of six, seven, eight, and even ten years' standing. Sometimes it is necessary to repeat the application of the induced current several times and during many weeks before a permanent result is attained. In some cases, however, the introduction of an electrode within the larynx is not required; it is sufficient to apply a current across the neck from one side to the other, the electrodes being placed one over each thyroid cartilage. Dr. Mackenzie speaks, also, of having occasionally succeeded with stimulating steam inhalations made with oil of Calamus aromaticus, or with creasote; or he has used a strong solution of nitrate of silver (ʒj ad ℥j) or of perchloride of iron (ʒij ad ℥j), applying it with a brush to the larynx or introducing it in the form of spray.

Laryngismus Stridulus.—This appears to me to be, in spite of its clumsiness, the best name for an affection which by many recent writers is described as *spasmus glottidis*. The latter term is, in fact, at once too wide and too narrow for the purpose—too wide, because obstruction of the glottis by spasm is really present in many diseases and not in one only; too narrow, because in the disease now to be considered there is, or may be, spasm of many other muscles besides those of the larynx. Laryngismus stridulus in its extreme forms exhibits an organized series of phenomena, comparable only with those which characterize an epileptic seizure. As we shall presently see, there have been doubts as to whether it is dependent on a primary disturbance of the central nervous system, or is reflex and due to peripheral irritation. But however this may be, its proper place in the nosology is certainly among the neuroses (*cf.* p. 631), and the only reason for discussing it in this place is that clinically its symptoms have to be studied in relation with those of laryngeal diseases. The name of laryngismus stridulus was invented by Dr. Mason Good, and has since been very generally adopted. Another name for it is that of "child-crowing," proposed by Dr. Gooch. At one time it was called "thymic asthma," under the idea that it was caused by enlargement of the thymus, an idea which, originally promulgated early in the last century, was strongly maintained by Kopp in 1830, but has since been finally rejected by Bednar in 1852, and by Friedleben in 1858. As a matter of fact, it is now certain that there is no relation whatever between laryngismus and the state of the thymus.

In its simplest form laryngismus stridulus consists of a disturbance of the natural rhythm of the respiration, such that the child (for the disease is one of childhood) first holds its breath and then makes a more or less

noisy inspiration. This occurs again and again at varying intervals—perhaps especially on the first waking from sleep—but neither parents nor nurses may attach much importance to it, thinking that it is merely a "catching in the breath." Gradually, however, the paroxysms may assume a more serious character, or this may belong to them from the very commencement. The child then shows signs of great distress and alarm. Its neck and back become arched, its chest and abdomen fixed and rigid, its eyes turned upward, its limbs tonically contracted, the thumbs being bent inward, the fingers extended, the wrists flexed, whereas the legs are thrust out, the soles turned inward, the toes stretched wide apart. Its face, at first pale, may turn purple, or of a ghastly, leaden color. Sometimes the fæces and the urine are discharged involuntarily, sometimes there is a noisy expulsion of flatus. After a few seconds, or a minute or two at the longest, the spasm yields. In all probability, while it lasts, the glottis is completely closed. As it passes off a chink is formed, through which the air can slowly enter, making a loud crowing sound. This usually ends the seizure, but sometimes two or more paroxysms occur in rapid succession. Or, according to Steffen, in Ziemssen's "Handbuch," a few unrhythmical and noisy expirations, and one or more whistling or crowing inspirations, may precede the stoppage of the breath, which constitutes the central feature of the attack. After the paroxysm is over the child often frets or cries for a little while; or it may fall asleep; or, again, it may at once seem to be as well as ever, returning with as much zest as before to its toys, or to any game in which it may have been engaged.

It is clear, from this description, that, as I have already remarked, many other muscles besides those of the larynx are concerned in the production of attacks of laryngismus stridulus. The preliminary stoppage of breathing may, in mild cases, be perhaps attributed to a mere inhibition of the respiratory centre, rather than to spasm. Steffen, indeed, says that with few exceptions the diaphragm and the chest walls are "in the inspiratory position" when the pause occurs, but I think he can hardly mean that they are in the position which they normally occupy when inspiration is completed, for if so, how is the prolonged inspiratory movement that immediately follows to be satisfactorily accounted for? Even as to the part played by the intrinsic muscles of the larynx at the time when the crowing inspiration takes place there has been some difference of opinion among writers. Some have doubted whether there is simply a spasm of the sphincter of the glottis (as is generally supposed), and have thought that there must be also paralysis of the crico-arytænoidei postici, of which the function, of course, is to keep the cords apart. Now, in experiments on rabbits it is found that if the superior laryngeal nerve on one side is divided, and its central end is then excited by a faradic current, the result is a strong bilateral adduction of the vocal cords. But if stimulation of the centres in the bulb thus normally tends to evoke such a movement, the only question that remains is whether, when it occurs, the nerve fibres to the abductors are left out of the reflex circle, or whether these muscles are overpowered by their antagonists as a result either of their own intrinsic weakness or of a less powerful degree of innervation. For my own part I think that the preponderant action of the sphincter is sufficiently explained by the latter hypothesis, supported, as it is, by facts already adduced at p. 795 with regard to the relative liability of the two groups of muscles to paralysis.

But it does not always happen that a seizure of laryngismus stridulus ends favorably. Sometimes, instead of relaxing, the spasm persists until life is extinct. There is then, of course, no crowing sound, and there may be nothing whatever to indicate the cause of a death for which the parents

are utterly unprepared. Some years ago I made an autopsy on the body of an infant aged sixteen months, all that I was told about it being that it had died suddenly and had been brought to the hospital by its mother. Finding nothing to account for such an occurrence, I presently thought of laryngismus stridulus, and on inquiry next day I learned that the child had previously had attacks of "child crowing." Steffen relates the case of an infant six months old, who was one night taking the breast when it was attacked with slight spasm of the glottis, after which it went on sucking. However, the attack returned more severely, and the child fell backward. Within a quarter of an hour Steffen was at the spot. The child had been laid in its cot, under the idea that it was sleeping. He found it livid and dead, without any signs of spasm of the limbs or of any other part than the larynx. Sometimes, however, death is preceded by tremulous twitchings of muscles, or even by a more or less complete epileptiform convulsion, exactly as is the case in other forms of asphyxia. As may well be supposed, after death from laryngismus stridulus the brain and its membranes are found gorged with blood, but there is no reason whatever for imagining that the congestion is antecedent to the paroxysms or concerned in producing them. When the disease has been of some standing the lungs may, according to Steffen, be found emphysematous; if this is really the case, in uncomplicated instances of an affection in which there is only inspiratory dyspnœa, it has an important bearing on the theory of emphysema in general.

Ætiology.—The most obvious fact in the ætiology of laryngismus stridulus is its relation to rickets. According to Steffen, at least nine-tenths of all cases are in rachitic children. By Elsässer it was thought that craniotabes was in a special way associated with the development of laryngismus, but this appears not to be the case. It is doubtless in consequence of their having all in turn been sufferers from rickets that laryngismus has been sometimes noticed in several successive offspring of the same parents. Dr. Reid mentions a family of thirteen, of whom only one escaped laryngismus and four of them died of it. Children affected with laryngismus are not infrequently fat, so that their parents fondly imagine them to be hearty and strong; but this, as is well known, is quite compatible with their being highly rachitic. A curious point, which I believe to have been originally noticed by Henoch, is that laryngismus stridulus is of far more common occurrence in the early part of the year than later on. Dr. Gee, among sixty-three cases, observed no fewer than fifty-five between the months of February and June inclusive. His explanation is that the extent to which children are kept indoors during the winter increases the irritability of their nervous centres.

Age and Sex.—The *age* at which laryngismus stridulus begins is generally from four months to two years. But Dr. Reid relates cases in infants only a few hours after birth; and some have been recorded in children of various ages up to nine years. There is, however, one remarkable fact which shows that rickets can only be looked upon as a predisposing cause of laryngismus, namely, that the latter affection is greatly more frequent in boys than in girls. Steffen cites figures from different observers, showing that the proportion of males to females is higher than that of two to one. In relation to it he alludes to the circumstance that membranous croup also is far more commonly seen in male children than in female; and this is still more markedly the case with false croup. Such an indication of a relationship between laryngismus and other diseases of the main air passages no doubt tends to make one hesitate in assigning a principal place in its pathogenesis to an over-excitability of nerve centres in the bulb. Otherwise it is almost impossible, in meditating upon the subject, to avoid arriving at that

conclusion. What are the facts as regards the occurrence of tetany and carpo-pedal contractions in children of the two sexes? On making cursory reference to some of the recently recorded cases of tetany, I find that the patients have been chiefly boys. And although chorea is well known to be more common in girls than in boys, one must remember that its seat is probably not in the bulb, but in the large basal ganglia; and, moreover, that it occurs at a later age, when the course of development may probably have altered the relations between the two sexes as regards the susceptibility of the nervous centres. In this connection it is, perhaps, worth noting that many more males than females, whether children or adults, succumb to tubercular meningitis.

Whatever may be the real bearing of these facts upon the ætiology of laryngismus, I think that we may, without hesitation, reject another hypothesis which a few years ago had a great many supporters, namely, that it is necessarily of reflex origin and dependent upon some irritation conveyed upward to the nervous centres from the periphery. This was declared by Dr. Marshall Hall to be the "only true mode of viewing" the disease, and he maintained that the cause of it was almost always to be found either in a morbid state of the teeth, in disorder of the stomach, or of the bowels. A few years before Dr. Hugh Ley had endeavored to prove that laryngismus was the result of mechanical irritation of the vagi nerves by enlarged bronchial or cervical glands. He did, in fact, show that glandular enlargement was often present. But it certainly may be absent, as I have myself once observed; and I think that no morbid anatomist, knowing how frequently in children who had been entirely free from laryngismus the vagi nerves are found surrounded by caseating glands, will assign to them any important share in the ætiology of that affection. I am not at all concerned to deny that when the nervous centres are in a morbid state, their successive explosions, due mainly to a gradually increasing irritability of their cells, may be directly brought about by stimuli conveyed to them from internal organs, just as in severe cases it often happens that they can be at once excited by merely touching the surface of the child's body. The analogy of epilepsy and of many other neuroses is entirely in favor of such an opinion. Even then, however, the fact that what must be a constant stimulation produces only paroxysmal effects shows how preponderant is the influence of the varying state of the nervous centres—unless, indeed, one accepts Steffen's idea that the pressure caused by enlarged glands alters with the degree of hyperæmia in which they happen to be.

As regards *diagnosis*, I think it may almost be said that there is no other affection with which laryngismus stridulus can be confounded by those who know its symptoms. At the moment of attack, if there had been none previously, or if the child's history were unknown, one might doubt whether the obstruction of the larynx was not due to a foreign body. It would be quite right to pass one's finger to the back of the throat to settle the question; but the subsidence of the spasm (unless it proved fatal) would very quickly show the real nature of the disease.

Prognosis.—The natural course of laryngismus stridulus, when undisturbed by treatment, varies greatly in different cases. Sometimes the attacks continue to be slight, and occur at wide intervals; sometimes they increase in severity and in number, until there may be thirty or forty of them in the twenty-four hours. In either case they may after a few weeks gradually become less frequent, until at length they cease entirely. Different writers have expressed different opinions with regard to the proportion of deaths to recoveries, some saying that one case in every two or three proves fatal. I should rather agree with Steffen, who says that if mild and severe cases be reckoned up together, the prognosis is generally favorable. It is, of course,

better when the child is not very young; and according to Steffen it is better in girls than in boys.

Treatment.—The treatment of laryngismus stridulus is, in the first place, that of rickets; sunlight, fresh air, good food, cod-liver oil. A change into the country or to the seaside is often quickly followed by the subsidence of the attacks. Dr. Ringer strongly advocates sponging with cold water twice or thrice daily. As a medicine, I have always been well satisfied with the bromide of ammonium, of which from three to five grains may be given as a dose, even to a young child. Steffen, however, says that musk is more useful than any other drug; he also gives castoreum.

It is quite right to be on the lookout for any morbid affections of distant parts that may possibly be concerned in irritating the nervous centres. If the gums were hot and tense, I should certainly advise their being lanced. Should the bowels be loaded, or should they contain worms, a few aperient or vermifuge doses ought to be given. But I do not think that it will commonly be found that by such measures any great results are attained, in the way either of diminishing the severity or of reducing the frequency of the seizures.

When a paroxysm is so prolonged as to require treatment, cold water may be dashed over the face and head, the body being, perhaps, immersed in a warm bath. A bottle containing ammonia may be held to the nostrils. The inhalation of chloroform is recommended by some writers. Faradization of the phrenic nerves seems likely to be useful. Even if life should have apparently become extinct, it may sometimes be restored by artificial respiration, as was pointed out many years ago by Mr. Johnson, in the fifth volume of the "*Dublin Hosp. Reports.*" In all severe cases the parents or the nurse should be taught beforehand how to act should the emergency arise.

Functional Disorders of the Voice.—To complete my description of the laryngeal affections due to disordered action of the muscles of the glottis, I must briefly mention certain cases in which there is an alteration in the pitch of the voice. Störk relates instances of children with an unduly low voice, and others of young men, after the "breaking" of the voice at puberty, with an excessively high voice. The remedy, in the former case, is to practice speaking with a falsetto voice; in the latter, with a bass voice. This is often perfectly successful, if sufficient perseverance be shown. Sometimes the desired change in the pitch of the voice is brought about very rapidly; for instance, a young man, aged eighteen, who for about a year had spoken in a falsetto voice which contrasted ridiculously with his broad, well-built appearance, was told to utter the vowel *u* for an hour daily, in as deep a voice as possible, at the same time holding the head fixed, this last direction being merely for the purpose of keeping up his attention; on the fourth day his voice became normal, and from that time it remained so.

Another curious affection is that which Dr. Mackenzie has named "spasm of the tensors of the vocal cords." This is characterized by a state of the voice so peculiar as to be at once recognized by those who are familiar with it. The following is Dr. Mackenzie's description: "The patient is often able to produce some notes, either in his own natural voice, or in a slightly muffled tone; but, while he is speaking in this way, the current of the voice seems to be partially interrupted, and the sound conveys the idea of an arrested action of the respiratory muscles. In fact, it is very much like the straining and rather suppressed voice of a person engaged in some act requiring the prolonged and steady action of the expiratory muscles (parturition, defecation). The patients often complain that they cannot

get their voice out. After speaking a word or two, or even several sentences, in this peculiar tone, the patient may again utter a few words in a comparatively healthy voice, and then may immediately relapse into the diagnostic intonation." Or there may be a complete absence of sound, the lips moving in the usual way for the utterance of words and phrases, which nevertheless are lost in silence. A clergyman is described as having been greatly distressed by the fact that while he kept on reading the service some of the words dropped soundless from him. Dr. Mackenzie, in 1880, had seen only thirteen cases of this kind; eleven of them were in men, ten being clergymen, and the eleventh a barrister; two were in women, both of whom had had constantly to speak to deaf relatives. Doubtless, therefore, it is the result of over-use of the voice. The onset of the affection was sometimes gradual, sometimes sudden; in the latter case it was attributed by the patients themselves to "catching cold." No treatment was permanently successful in any instance.

Malformations.—This will be the most convenient place for me to mention certain congenital abnormalities of the larynx that occasionally give rise to clinical symptoms. One such appears to be almost confined to female infants, and causes the act of inspiration to be attended with a loud crowing noise, which is nearly constant, continuing even during sleep and after the administration of chloroform, though it is louder during the day, and may sometimes be increased by exposure of the body to cold or in other ways. The noisy state of the breathing is present from the time of birth, but disappears entirely at the end of about a year. Dr. Lees has had an opportunity of making an autopsy in a case of this kind, and in which death was due to diphtheria; and he found ("*Path. Trans.*," 1882) that the epiglottis was folded on itself, like a leaf on its midrib, the aryteno-epiglottic folds being almost in contact. A similar state of things had been seen in the laryngeal mirror during life. The affection is probably not uncommon, for it was the fourth case that came under Dr. Lees' notice, and others have been observed by Dr. Gee and by Dr. Barlow.

Another, and a much more serious malformation, described by Dr. Mackenzie, consists in a longitudinal bifurcation of the epiglottis, forming two flaps which (in a case that he saw) fell into the larynx, causing constant symptoms of laryngismus from the first week, and death at the end of four months.

Lastly, a congenital band of mucous membrane sometimes connects together the anterior parts of the cords. Dr. Mackenzie has recorded such an instance in vol. xxv of the "*Pathological Transactions.*" The patient was a young lady, aged twenty-three, who had had complete aphonia from birth, never having cried, even as an infant. There seems to have been no dyspnœa. Laryngoscopically the web was seen as a flat membrane during inspiration, but on attempted vocalization it became folded up, and protruded so as to resemble a tumor, of red color, and of about the size of a haricot bean. Dr. Mackenzie excised it, and the patient immediately afterward spoke, and soon acquired a perfectly natural voice. In a case of Dr. Poore's, exhibited at the International Congress in 1881, the patient, a girl of thirteen, could speak, but with a peculiar falsetto tone of voice; she had been liable to attacks of dyspnœa from infancy.

We now pass on to the *inflammatory affections of the larynx.*

They differ in different cases very widely as regards their exact seat, their symptoms, and, above all, the course which they run. It is, therefore, necessary to classify them in some way, and one very obvious distinction between them lies in the fact that some of them mainly affect the mucous

membrane, whereas others start in the deeper structures. At present I shall speak only of the former, and these, again, have to be further subdivided.

In clinical practice, inflammations of the laryngeal mucous membrane fall naturally, and indeed inevitably, into two groups. On the one hand, there are cases attended with dyspnœa so severe that it threatens and often actually destroys life; these are commonly known as cases of *croup*. On the other hand, there are cases in which the chief symptom is an impairment of the voice; these commonly pass under the name of *catarrhal* laryngitis, which again may either be *acute* or *chronic*.

Croup.—In 1765 Dr. Francis Home, of Edinburgh, His Majesty's Physician, published, in a short tract of sixty pages, an "Inquiry into the Nature, Causes, and Cure of the Croup," a disease which he declared to be entirely unrecognized by medical writers, although it was known to the common people of Scotland by several distinct names. In each of the post-mortem examinations which he made he found the trachea lined by a more or less complete membranous layer. Thirty-six years later, in 1801, another Scotch physician, Dr. John Cheyne, wrote on the same subject a work which has become classic; his views upon the pathology of croup were the same as those of Home. In the meantime the existence of such a complaint had become matter of common knowledge in England as well as on the Continent. And there were also known certain other affections of the throat, the relations of which toward Home's disease are discussed at some length by his successor, so that in that work one may discover the rudiments of a controversy which has of late years attracted much notice, and which even now is not finally settled.

This consists of two questions, separate and yet closely connected with one another; the first, whether the membranous "croup" of the Scottish writers is distinct from the disease which has since been called *diphtheria;* the second, whether it is distinct from a milder affection of the air passages, unattended with the formation of any false membranes, and which is known by several different names, including *false croup, stridulous laryngitis, spasmodic laryngitis,* inflammatory croup.*

To each of these questions an answer must be given before we can pass on to consider the clinical history of croup. I shall therefore proceed to discuss them in turn, and, first, I will take that which concerns the relation of the disease to diphtheria.

I. The controversy in regard to this point was definitely commenced by Bretonneau, of Tours. In his earlier "Memoirs on Diphtheria," 1821–26, he made it his principal object to prove that that disease and croup are identical. His view was in due course adopted by his pupil, Trousseau, and by Guersent, Barthez, and almost all the other leading French physicians. In England it was for a long time repudiated by every medical writer, but within the last few years it has met with a much more favorable reception. The late Dr. Hillier advocated it in 1862, and since then Dr. Johnson, Dr. Semple, and Sir John Cormack have maintained it, and Sir William Jenner has withdrawn his previously expressed opinion that the two complaints are distinct.

Now, in the first place, I must point out that both Home and Cheyne were perfectly acquainted with the fact that the disease which they described

* By an unfortunate confusion some writers have designated as "spasmodic croup" the complaint which is commonly known as "*laryngismus stridulus*," or "*child crowing*." This complaint, the symptoms of which are altogether unlike those of croup, is described at p. 800. Let me point out, once for all, that in the present chapter no reference whatever is made to it.

was liable to be confounded with one which affected the larynx secondarily, having its original seat in the fauces. The first of these writers, quoting Dr. Hare's graphic account of the "morbus strangulatarius" in Cornwall (which was epidemic diphtheria in its most typical form), says that that complaint "appears more nearly allied to the malignant sore throat, although it sometimes attacked the trachea." And the second of them commences his section on diagnosis by remarking that he had seen several children whom he would have supposed to be suffering under the second stage of croup had he not discovered sloughs upon the tonsils and uvula. Probably each of these observers had better opportunities of studying the relations of the two complaints than any London physician at the present day, and I think it is worthy of notice that if they should prove to have been wrong in regarding them as distinct, the progress in medical science will, in this instance, lead to a result directly opposite to that which it is bringing about in all other cases; for in regard to every other group of diseases, the more our knowledge advances the more are distinctions and subdivisions multiplied.

One argument in favor of the identity of membranous croup with diphtheria may, I think, be very easily disposed of, although stress is laid on it by the authority of Sir William Jenner. I refer to the supposed fact that mucous membranes are not, like serous membranes, prone to pour out lymph upon their surface when inflamed by simple irritants, so that, it is urged, an affection of the larynx, attended with the formation of false membranes, must be a specific inflammation. But, both for the fauces and the air passages, it is certain that this inability is far from being absolute. In the "*Guy's Hospital Reports*" for 1877 I have recorded seven cases in which those parts presented appearances indistinguishable from diphtheria as the results of scalds of the throat by boiling water, which the patients (always children) had sucked from the spout of a teapot or kettle; one case of a boy who got a bean into his right bronchus, and who had his larynx and trachea coated with lymph; two cases (already referred to at p. 296) of children who had their fauces irritated by the ingestion of a piece of hot potato (which stuck in the throat) and by a burning stick respectively, and in whom the morbid action took the same form; one case of a man, aged twenty-four, who was admitted for a cut throat, and who died of a plastic inflammation of the larynx, trachea, and bronchial tubes; three cases in adults, in which a membranous laryngitis was secondary to cancer of the pharynx, tubercular ulceration of the vocal cords, or syphilitic disease, for which tracheotomy had been performed; and, lastly, two cases, both in adults, in which a similar affection was associated with an acute or a chronic pneumonia—sixteen in all. Many of the patients, indeed, had had tracheotomy performed some days before death, and it might be plausibly argued that a badly-cleaned tube, if it had before been used for a case of diphtheria, might conceivably have inoculated the parts with the specific disease. This, however, would not apply to some of the cases at all, and it is also inapplicable to one recorded by Mr. Parker ("*Clin. Trans.*," 1875) of a child who had scalded its throat with hot water, and from whose trachea false membranes were drawn up by means of a feather almost immediately after the operation. Moreover, both Rietz and Oertel have found it easy to set up a plastic inflammation of the trachea in dogs and rabbits by dropping a few minims of Liquor Ammoniæ into it through an external wound. Oertel says that he performed this experiment on seventeen animals, and succeeded in every instance in generating an artificial croup.

On the other hand, some arguments have been adduced on the opposite side which have been shown by recent researches to be untenable. Thus, it was long believed that the false membranes presented constant differences of microscopical structure and of chemical constitution in croup and

in diphtheria respectively, but we now know that in diphtheria itself they vary in their appearance, in the relation which they bear to subjacent parts, and even in their histological characters, according to the part of the mucous tract upon which they are developed. This fact necessarily involves the overthrow of all the histological distinctions that had been drawn as between the two diseases. A single point of difference is still declared to exist by Oertel. He believes the presence of micrococci to be essential to diphtheria, but in the false membranes which he set up artificially in animals by dropping Liquor Ammoniæ into the trachea, he found micrococci in small numbers and only in the more superficial layers. He therefore argues that the criterion of a simple non-specific croup is the absence of these organisms in the inflammatory products. Now, I have (at p. 292) given reasons for believing that the micrococci which are generally present in diphtheritic membranes are far from possessing the importance which Oertel attributes to them, but it might still be true that their non-occurrence in croup should be distinctive of that disease. As a matter of fact, however, other observers have failed in cases of diphtheria to detect the parasites in false membranes below the glottis. It seems impossible to avoid the conclusion that their presence, so far from being a secondary part of the diphtheritic process, is rather an indication that it is of an unhealthy character, or that the false membranes themselves are undergoing putrescence. The laryngeal form of diphtheria, perhaps, kills too quickly for such indications to be manifested.

This brings me to another distinction between diphtheria and croup, on which some observers have laid great stress, but which seems to be capable of ready explanation. I refer to the clinical fact that marked symptoms of depression of the vital powers—a dry, brown tongue, sordes on the lips, petechiæ on the skin, hemorrhages upon the internal serous surfaces—are present in the former, but absent in the latter affection. In many cases of diphtheria, however, no such symptoms show themselves until several days have elapsed, and since croup destroys life rapidly by the mere effects of the presence of false membranes in the air passages, one could not reasonably expect that it should be attended with indications of depression and of septicæmia, even if it were a modification of diphtheria.

Again, the fact that a definite exposure to cold has immediately preceded and apparently excited an attack of membranous laryngitis, seems to be no proof that the case is not one of diphtheria. At least, Sir William Jenner says that he has seen cases which arose in this way, and which he believed to have been diphtheria because albumen showed itself in the urine; and I have at p. 295 quoted Dr. Yeats's observations (which seem to refer to faucial diphtheria occurring in adults) as to the frequency with which, during an epidemic, those persons were attacked who had immediately before been exposed to the night air. What, then, should one suppose to have been the probable nature of the disease in Dr. Gregory's twin children, who (as Sir Thomas Watson relates) were both seized with croup on the same night, having been walking together in the sunshine, during the evening, in a cold wind?

Thus I am prepared to allow that no criteria, based either upon morbid anatomy or upon clinical symptoms, avail to distinguish laryngeal diphtheria from croup; that is to say, that the cases referred to by Sir William Jenner and others, in which diphtheria is limited to the air passages, are really undistinguishable from croup. But I am, nevertheless, far from admitting the identity of the two diseases. For it is certain (see p. 301) that, whereas in *epidemics* laryngeal diphtheria is rare, laryngitis and tracheitis are of common *sporadic* occurrence. Now, this is in itself difficult of comprehension if we suppose the latter affection to be a form of diphtheria. One

could perhaps understand that a laryngeal diphtheria might be less contagious than one affecting the fauces, as being further from the surface of the body, and so one might account for its being less apt to be the starting point of an epidemic. But this would by no means cover the fact that while isolated cases of membranous laryngitis are frequent, a laryngeal diphtheria is rarely set up by the direct action of the specific contagion of the disease. In Guy's Hospital it has never happened, when a person admitted for diphtheria has communicated the disease to other patients in the same ward, that the morbid process in those patients has limited itself to the air passages.

The question at issue, however, is not to be so easily disposed of. At p. 294, I have brought forward some facts which prove that cases in which there is an affection of the fauces, such as would be commonly called diphtheria, are much less often traceable to contagion when the air passages are affected than when they escape. There seems to be a regular descending scale of contagiousness, according as the morbid process falls with less intensity upon the tonsils and palate, and with more intensity upon the larynx and trachea. Moreover, whereas diphtheria attacking the fauces is common in adults, all the cases at Guy's Hospital in which these parts are but slightly affected, and the brunt of the disease has fallen upon the air passages, have been in children below five years of age. Now, I think it is very improbable that such differences should exist, either as regards the contagiousness of the disease, or as regards its occurrence at a special period of life; but one sees at once that the recognition of these differences is exceedingly favorable toward the inclusion within the domain of diphtheria of cases in which the larynx and trachea are alone attacked, the tonsils and palate remaining free. For the latter cases are likewise peculiar to children, and their non-contagiousness is the very point on which the whole discussion turns.

To me, however, it seems preferable to adopt another solution of the difficulty which involves no such improbabilities. I think that the greater number of the cases referred to at p. 806 (and of which I have given details in the "*Guy's Hospital Reports*" for 1877 at p. 384, *et seq.*) were really not examples of diphtheria at all, although the fauces were affected. After all, it seems an absurdity to draw a fixed line at the edge of the epiglottis, and to say that so long as an inflammatory process is limited to parts below it, the case may be one of simple membranous croup, but that if it spreads above this line it must be due to the specific poison of diphtheria. I am aware that the great difference in histological structure between the mucous membranes of the larynx and pharynx, respectively, has been supposed favorable to such a view. But we know that, whether in diphtheria or after scald of the throat, no obstacle to the *descent* of an inflammatory process is offered by this difference in structure. And I fail to see why it should be a bar to its *ascent*, when the air passages are the first to be attacked.

II. I must next pass on to consider what relation toward the membranous croup of Home and Cheyne is borne by those cases—of not infrequent occurrence—in which the air passages are less severely affected, so that no false membranes are found. Now, all writers of the present day describe certain cases of this kind as entirely distinct, and give to them a special name. Bretonneau is generally supposed to have been the first observer who indicated clearly the points of difference, and his name for the cases in question was "stridulous angina." In reality, however, the English writers of the end of the last century were well acquainted with the clinical history of the affection, which was known to them by the designation of "spasmodic croup" or "spurious croup." I have not been able to discover who originally pointed out its peculiar characters; but they are fully set

forth in a paper which Mr. Field, Secretary to the Medical Society of London, read before that body in 1796.

The most distinctive features of spasmodic or spurious croup are the suddenness of its onset and the alarming nature of the symptoms which it presents from the very commencement. A child who is apparently in perfect health, or who may have had a slight cold for a day or two, goes to bed without any sign of laryngeal affection, and falls asleep as quietly as usual. About eleven o'clock, or at midnight or a little later, he suddenly starts up in a state of extreme excitement and terror. He coughs incessantly, making a hard, hoarse, barking noise. He pants for breath, and each inspiration is attended with a loud, crowing sound. His voice is hoarse and it may be very feeble, but it is not whispering, as in true croup. His face, at first flushed, afterward becomes pale and covered with a cold sweat. The nurse and parents are horror-stricken, and send at once for the nearest medical man. But, instead of the child getting worse, each paroxysm of coughing is rather less severe than the preceding one. And after half an hour, or in two or three hours, he becomes calm and sleeps. In the morning, when he wakes up, his cough is still hoarse and barking, but it is not so hard; his respiration is attended with little or none of the whistling sound; his voice has nearly regained its natural tone. During the day the child is as cheerful as before, and has but little cough; his pulse is not accelerated; he is scarcely, if at all, feverish. On the following evening, however, the symptoms often return, sometimes as severely as at first, but generally less so. They may even repeat themselves for several nights in succession, with gradually diminished intensity.

It is doubtful whether attacks of this kind ever prove fatal. Trousseau speaks of having seen three cases in which death occurred. But the only one of which he gives details is that of a school-boy, thirteen years old, who was suddenly seized with dyspnœa on waking in the morning, and who seems to have died at the end of about four hours. On post-mortem examination the laryngeal mucous membrane was found to be reddened, and the aryteno-epiglottidean folds were a little swollen; the vocal cords were a good deal swollen, and on one of them "there was a slight membranous concretion, possessing, however, none of the characters of diphtheritic false membrane." It seems to me that in this case a severe inflammation of the larynx would probably have developed itself if the patient had lived a little longer. It certainly was not a typical case of spurious croup as regards the time at which the attack began, and the boy was much beyond the age at which that affection is most apt to occur.

At present, therefore, the pathology of spurious croup is matter of inference only. But I think there can be no doubt that it depends upon a slight catarrhal inflammation of the laryngeal mucous membrane, complicated with spasm of the muscles of the glottis.

Another feature of this complaint is its tendency to recur again and again in the same individual. A child who has once had it is always likely to be attacked a second time if he is exposed to cold or wet; and up to the age of fourteen or fifteen every slight catarrhal affection is apt to be accompanied with the peculiar hard, barking cough. When one hears that a person suffered from croup repeatedly during childhood, I believe that one may safely conclude that the disease was "spurious."

Now, Cheyne, in the second edition of his great work, discusses at considerable length the relation between the disease of Home and this "spurious" or "spasmodic" affection, with the description of which he was familiar through the writings of Field and of Ferrier, a physician of Manchester, who had published an essay on the subject in 1810. And he comes to the conclusion that there are no just grounds for admitting two kinds of

croup. The affection in question "occurs," he says, "in those families which are subject to genuine croup; it arises from the same exciting cause (exposure to cold); it prevails during the same weather." And he goes on to point out that in many cases in which the breathing afterward becomes permanently affected, the symptoms are for the first few days most marked during the early part of the night, the patient in the daytime seeming to have nothing the matter with him with the exception of a cough. But I think it must be admitted that there are two criteria which point very strongly to the existence of spurious croup as a separate member of the nosology. One is the sudden onset of the complaint with all its symptoms in full force, whereas the affection attended with the presence of false membranes comes on more or less gradually. The other is its liability to return again and again in the same individual, whereas membranous croup seems never to attack a child more than once. If spurious croup were merely an undeveloped or milder variety of the disease, one would expect that persons who were subject to it would be very apt, on some occasion when the exciting cause happened to be powerfully in operation, to have it in its severe form, and to die with membranous exudation into their air passages. I cannot find a single recorded instance in which this has occurred.

There can be no doubt, therefore, that Cheyne did include in his description of croup cases which were really distinct from those in which false membranes were found in the larynx and trachea. And, indeed, it would seem that every case of which he gives the details, and in which a fatal termination did not occur, happened to be an example of the spurious form of the disease.

But it is quite another question whether all, or even most, of the non-fatal cases of croup that one meets with in practice are to be placed in the same category. Trousseau says that although "stridulous laryngitis" (as he terms the affection) is very common, he had had only one case in his wards at the Hôtel Dieu, a principal reason for this being the sudden way in which it declares itself, and the rapidity with which it yields, so that children attacked by it are very seldom brought to hospitals. But in Guy's Hospital, between the years 1867 and 1876, there were admitted ten cases of croup, at least (and probably several more), in which recovery took place, and in which there was no proof of the formation of false membranes. In many of these cases the symptoms were continuous for some days in succession; and the clinical reports very seldom say anything about previous attacks of a similar kind. Perhaps it may be that in London a form of non-membranous croup is of frequent occurrence which in Paris is not met with. At any rate it is a striking fact that Dr. George Johnson—who is anxious to draw a sharp line of distinction between the cases which present false membranes (and which he regards as examples of diphtheria) and those in which no such membranes are found (which cases he terms "infantile laryngitis" or "inflammatory croup")—is obliged to extend very greatly the definition of the latter affection beyond the narrow limits which had been set for it by the earlier English and by the French writers. In fact, in the last edition of Sir Thomas Watson's "Lectures" (in which Dr. Johnson's views are adopted) the whole description of "croup" is transferred bodily to the new "infantile laryngitis." This is no longer the harmless affection of Bretonneau and Trousseau, but a disease which "proves fatal sometimes within twenty-four hours and often within forty-eight hours," and which "may continue for five or six days before it terminates." Now, so far as I can ascertain, there has at Guy's Hospital, in the course of many years past, been only one case which has ended fatally, and in which on post-mortem examination false membranes have

been absent. But, as I have already remarked, it often happens that there is no evidence of their presence during life. I, therefore, think it probable that the systematic performance of autopsies would lead any believer in the view that all membranous croup is diphtheria to transfer to the latter disease almost all of his fatal cases. But surely it is without precedent in pathology to draw what is in fact an arbitrary line of distinction between those cases of a disease in which recovery takes place and those which prove fatal. I cannot doubt that in many cases of croup which recover, false membranes are really present in the air passages, even though none may at any time be expectorated; probably they may become disintegrated in the more fluid products of the inflammatory process and disappear; or they may be hawked up into the mouth and swallowed.

To sum up: I think that no fixed line can be drawn between membranous croup and the milder forms of the disease in which no false membrane is developed, except that those cases in which it occurs over and over again—with sudden and alarming but very transitory symptoms—may be separated under a distinct designation, for which purpose the name of *spurious croup* seems to be the most appropriate. As regards the relation of diphtheria to membranous croup, I have shown that it is impossible to continue to occupy the position which some writers formerly took up, and according to which the presence of false membranes upon the fauces proves a case to belong to the former, even if their absence is not a proof that it is an example of the latter disease. I admit that in very rare cases a diphtheria may begin in, and remain limited to, the air passages, but I think it very much more often happens that a non-specific membranous croup extends to the tonsils and palate, leading to the formation of milk-white patches of greater or less size upon the surface of these parts. It is an instructive fact that in two out of eleven cases of "idiopathic croup" which came under Dr. West's care between 1839 and 1849, when diphtheria (at least in an epidemic form) was not prevailing in this country, there was "a scanty formation of false membrane upon the velum and tonsils." I am perfectly ready to allow that in an individual case the possibility that the disease may be diphtheria can never be absolutely negatived. The points in which I should lay most stress would be the absence of a history of contagion, the circumstance that no other person in the house or in the neighborhood had had anything that could possibly be set down as diphtheria, and (with due allowance for the facts stated at p. 295) there having been a direct exposure to weather or to some considerable change of temperature immediately before the commencement of the attack.

I regret to have to add that the opinions which I have been expressing are antagonistic not only to those of French and of some English writers, but also to those of the German physicians who have published the most recent articles on the subject. These do, indeed, all theoretically admit the existence of a simple laryngitis and tracheitis attended with the formation of false membrane. But in practice they seem to assign to diphtheria almost all the cases which come under their observation. It is an important question whether this may not depend upon the fact that on the Continent non-specific membranous croup is really a much more rare disease than in England. Cheyne makes the assertion that it is far less known in the temperate than in the northern regions of Europe; but one can hardly tell on what facts such a statement is based. I shall, however, advert to this question again further on. Even in Great Britain, the disease appears to be irregular in its distribution. Home states that in his time it was far more frequent in Leith and Middleburgh, which are near to the seashore, than in Edinburgh, and in that city Dr. Alison found it most prevalent in those parts which are lowest in situation. Wet and marshy spots are said

to be favorable to its occurrence. I have been assured by some of the medical men practicing in Norwich that it is unknown in that city, which has a very dry and bracing air, although it is much exposed to cold easterly winds.

Symptoms.—Croup commonly begins as a catarrh of but slight severity. The child is noticed to be a little feverish, refusing its food, but asking frequently for water. Its voice is hoarse; it sneezes frequently; it may have rather a shrill cough. It complains of no pain in swallowing. After some hours, or not until the lapse of four or five days, symptoms characteristic of the disease first begin to declare themselves. Their onset is generally gradual, but it may be sudden. And sometimes they appear in a child who had up to that moment seemed to be perfectly well; usually then coming on in the nighttime, just as in the case of spurious croup.

Of all these symptoms the most important is dyspnœa. The breathing is not only hurried, but noisy. Each inspiration is attended with a peculiar whistling sound, which may be audible at a considerable distance; and a similar sound, or one of a more snoring character, may accompany the expiration. On uncovering the child's chest, one sees that the structures above the sternum and the clavicles, and the lower intercostal spaces, are all drawn inward each time the child breathes. And as the disease advances, the epigastrium, the false ribs, and even the lower portion of the breast-bone itself, form part of a deep hollow, produced by the action of the diaphragm; this muscle, being arched upward into the chest by the atmospheric pressure, can only drag its attachments backward toward the spinal column, where it contracts.

The cough, at first harsh and clanging, gradually grows husky and at length is inaudible. The voice, from being hoarse, becomes whispering, and is finally extinguished. When the child attempts to speak, its lips can be seen to move, but not the slightest sound is heard. The nostrils dilate with each effort to breathe. The head is thrown backward as far as possible, and the spine is curved in the same direction. It is not very obvious why this peculiar attitude should be adopted, but the supposition is that the trachea is stretched, so that air can better pass by the side of the false membrane. Ferrier speaks of having seen the corpse of a child who had died of croup resting on its head and heels, exactly as if it had had tetanus.

Besides its persistent difficulty of breathing, a child laboring under this disease is also liable to frequent exacerbations, attended with the most extreme suffocative distress. An extraordinary restlessness is a principal symptom of such attacks. If lying or sitting in bed, the little patient starts up and throws itself into the arm of its mother or nurse; in an instant it begs to be put back into its crib. It clutches at anything that may be within reach, or even at its own throat, as if to tear away the obstacle to the free entrance of air. After a few minutes, or a quarter of an hour, it sinks back exhausted and may fall asleep. There is still a doubt whether such paroxysms depend upon muscular spasm, or upon the impaction of portions of false membrane, or of inspissated secretion in the chink of the glottis. And, indeed, different opinions are held as to the cause of the other symptoms of croup. Some observers think that they are the mechanical result of a swollen state of the laryngeal mucous membrane, and of the presence of a membranous layer, or of muco-purulent matter upon its surface; some refer them to spasm of the laryngeal muscles; and some (including Niemeyer) attribute them to a paralyzed state of the same muscles, which are supposed to be involved in the inflammatory process. Niemeyer, in fact, says that after death their substance is found to be watery, pale, and

soft. Of course, it is only during the act of inspiration that a whistling sound could be produced by paralysis; and, accordingly, this writer draws the distinction that when such a sound accompanies the expiration it is invariably caused by the presence of false membranes, obstructing the glottis.

It is a question whether the urine is ever albuminous. On theoretical grounds one would think it very likely that a disease which is attended with such extreme dyspnœa should be attended with albuminuria. But in Mr. Lamb's series of cases, recorded in the "*Guy's Hospital Reports*" for 1877, there was only one in which this symptom was detected, and in which there were not some other grounds for believing that the disease might really have been diphtheria.

Event.—When croup is to terminate favorably, the little patient's breathing becomes easier, its cough softer and more loose. It sometimes begins to expectorate muco-purulent matter in considerable quantity; and in this one may detect flakes and shreds of membrane, by floating it out in a saucer containing water, or even without doing so. In some cases large pieces are spat up, forming complete casts of the trachea. The symptoms are then greatly relieved; but the improvement is sometimes of very short duration. Sir Thomas Watson relates the case of a child who was on the brink of suffocation when tracheotomy was performed; a tubular portion of membrane of the size and shape of the trachea was presently forced through the opening; and the patient fell asleep; but within seven hours the dyspnœa returned and was soon fatal; at the post-mortem examination the windpipe was found to contain a new tube of lymph.

Indeed, remissions in the symptoms are of not infrequent occurrence, especially in the morning hours, even when no false membranes are expectorated. The child breathes better; its cough is less distressing; it partially regains its voice; it may ask for food, or get a little quiet sleep. One must not be deceived by such a change. Too often it masks a steady progress of the disease toward death. Thus Dr. West relates the case of a little girl who seemed to have been freed from all her alarming symptoms when she was admitted into the hospital on the fifth day of the disease. But nine hours afterward she died, without any great distress or violent struggle; and extensive false membranes were found in the trachea and bronchi.

In most cases the approach of a fatal termination is indicated by symptoms of asphyxia. The cheeks and lips become pale and bluish; the forehead is covered with a clammy sweat; the child ceases to take notice, and lies with half-closed, drowsy eyes; the breathing becomes shallow; the pulse is rapid and intermittent; the extremities are cold. Death is often ushered in by convulsions.

Ætiology.—The chief exciting cause of croup is by all writers said to be exposure to cold. But as scarcely any of them seem to have been careful to exclude cases of spurious croup from their accounts of the disease, one hardly knows what degree of significance to attach to their statements. Dr. Alison is quoted by Sir Thomas Watson as having noticed that it was often produced in a child by its sitting or sleeping in a room newly washed, and consequently that in Edinburgh cases of this disease occurred with especial frequency on Saturday night. Cheyne said that in all but three of the cases of croup which he saw there had been exposure to the weather; and of the exceptions one occurred in a child who had got up out of bed on the previous night, and stood for some time in a cold passage, snow being on the ground at the time; while another was the case of an infant, thirteen months old, who had been confined to the house on account of the weather for above a week, and was seized with the disease after a very cold, damp day. This accords with our experience in Guy's Hospital, where there has

not in the course of many years been an instance of a child being attacked with croup while an inmate of any of the wards. And Dr. Langdon Down, writing to the Committee of the Royal Medical and Chirurgical Society in 1876, mentioned that croup was unknown among the children at the Earlswood Asylum, whereas there had been many mild epidemics of diphtheria.

It is stated that croup is more frequent in the winter than in the summer months. One cannot draw any conclusions from so small a number of cases as that which I recorded in the "*Guy's Hospital Reports*" for 1877, but I may say that no such difference is observable among them; except, indeed, among those instances in which no false membranes were proved to be present. Perhaps, like acute pneumonia, the disease attacks those who are exposed to chills in the evening hours of a hot day, or during the cold weather, which in our climate may occur at any period of the year.

The *age* at which a child is most apt to be attacked with croup is between two and seven years. The complaint is rarely seen in infants at the breast; indeed, both Home and Cheyne thought that children weaned early were especially liable to it. In adults it is unknown. All writers agree in stating that boys are more often affected by it than girls. This is a point of some importance, because it may aid in fixing the boundary line between croup and diphtheria, a disease which (as might be expected) is equally frequent in both sexes. Indeed, I am not sure that the figures given by different observers, if taken in conjunction with their opinions about the two affections, may not suggest an inference with regard to the prevalence of croup and diphtheria, respectively, in different parts of Europe. Thus, Sanné says that out of 1575 cases of diphtheria admitted into the wards of M. Barthez (where the number of beds for males and females is equal), 813 occurred in boys, 762 in girls. The difference might fairly be attributed to the inclusion of a few cases of membranous croup, which is not regarded by French physicians as distinct from diphtheria; but does not the fact that the difference is not larger indicate that in Paris membranous croup is rare? On the other hand, in 101 cases of croup occurring at Prague, Steiner found that seventy-seven occurred in males, twenty-four only in females. He defines croup as an inflammation of the air passages, attended with the formation of false membranes, and seems to make no attempt to exclude cases due to the contagion of diphtheria. Do not his figures show that the latter disease must be relatively infrequent in the Bohemian capital? The experience of a single institution like Guy's Hospital is too limited to afford a secure basis for a comparison of the numbers of the two sexes, but I am bound to state that they show no considerable preponderance of males among the cases of membranous laryngitis, while they do show a marked preponderance of males among those cases in which it is probable that no false membranes were found. This fact, taken with the difference in the frequency of the two forms at certain periods of the year, is undoubtedly an argument against the view which I have maintained as regards the connection between membranous and non-membranous croup.

Histology.—I have already said almost everything about the pathological anatomy of croup, in speaking of its relations toward diphtheria. The false membrane itself possesses a beautifully laminated structure, being formed of layers of a homogeneous fibrillated substance which alternates with other layers consisting chiefly of leucocytes. It adheres very firmly to the epiglottis and the vocal cords; and Dr. West says that when it is detached the mucous membrane of these parts is often found to be slightly eroded or ulcerated. But lower down it lies

loose in the channel of the trachea—if it extends so far. Sometimes it is only in very small quantity, being represented by a few little shreds or fibres, embedded in muco-purulent matter, which then lines the larynx. In any case, such a muco-purulent matter is present wherever the membrane ceases in the parts of the air passages immediately adjacent. Thus, transitions between the two kinds of inflammatory product exist in every instance of croup; and we have every reason for expecting that unless the disease reaches a certain pitch of intensity, no false membranes should be found. I believe, however, that this is very seldom the case when it is severe enough to prove fatal.

The color of the membranes in croup is commonly whitish, or whitish yellow; but sometimes they are gray or brownish, from admixture of blood. Home relates, at second hand, a case in which the patient expectorated a piece "which had a mortified appearance, like black, shaggy silk."

All observers now believe that the larynx is ordinarily the starting point of the morbid process in croup. The earlier writers supposed that it began in the trachea, their reason for this opinion being the absence of soreness during deglutition. Cullen accordingly called the disease Cynanche Trachealis. Steiner, however, says that he has seen four well-marked cases of "ascending" croup. In each instance the disease began with slight febrile symptoms, a rather painful cough and dyspnœa. After four to six days, while the voice was still sonorous, membranes were expectorated. A week or more passed before the child became hoarse, and signs of laryngeal obstruction showed themselves still later, which in three cases proved fatal.

Extension of the false membrane into the lower air passages is of very frequent occurrence. Steiner says that out of fifty-five autopsies of children (among which, however, many were doubtless cases of laryngeal diphtheria) there were thirty-one in which it reached the bronchi, with casts even in the smaller tubes; in nineteen it affected the trachea but did not pass lower, and in five it was present only in the throat and larynx. He remarks that "in England, during recent epidemics, implication of the bronchi has been strikingly rare," but I do not know on what evidence this statement rests. Among seventeen cases occurring in Guy's Hospital, in which the fauces were unaffected, there were seven in which the bronchial tubes were lined with casts. Some observers have thought it possible to detect clinically such an extension of the morbid process, the symptoms supposed to indicate it being feebleness of the vesicular murmur, urgent dyspnœa, and marked inspiratory depression of the epigastrium. But I agree with Steiner that these signs are not to be relied on, at any rate before tracheotomy has been performed. The sounds produced in the larynx in cases of croup are so loud that auscultation of the lungs is very unsatisfactory in its results.

According to Steiner, pneumonia is of much less frequent occurrence than has generally been supposed. In seventy-two autopsies he found it in the lobular form eight times, in the lobar form only six times. Its recognition during life is difficult. Steiner and Peter have each met with cases in which there was dullness on percussion, with great weakness of the breath sounds over one lung, before an opening was made into the windpipe, but in which after the operation these signs quickly disappeared. Probably they depended upon atelectasis of the pulmonary tissue, which is a frequent result of obstruction to the entrance of air, the base of the lung, or even the greater part of its posterior surface, becoming purplish-red, dry, and airless.

The principal points in the *diagnosis* of croup have already been mentioned when I was discussing its relations toward the "spurious" affection and toward diphtheria. One must never forget that in reality none of the

special symptoms do more than indicate the existence of laryngeal obstruction. The distinction between croup and other diseases of the larynx is based upon the acute character of the attack and upon the age of the patient rather than upon anything in the symptoms themselves; but even children are liable to different affections in which the breathing may be no less stridulous. A post-pharyngeal abscess, for example, sometimes gives rise to "croupy" dyspnœa and cough, although it is not obvious why this should be the case. One day, when I was visiting my patients at the Infirmary for Children, the house surgeon told me that he had just been called to see a case of supposed croup, in which he had felt an abscess at the back of the fauces with his finger, and that relief was afforded as soon as the matter was let out. In a little child, eighteen months old, whom I saw a year or two since with Dr. Hudson, of Waltham Abbey, the respiration was rather of a snoring character than croupy, but there was a brassy cough. The abscess seemed to have commenced in a suppurative affection of the cervical glands, which had made its way inward, instead of pointing externally. According to Steiner, post-pharyngeal abscess occurs chiefly in infants at the breast, except when it is dependent upon disease of the spine. He also says that it develops itself more insidiously than croup. Sir William Jenner speaks of abscess at the side of the larynx in the connective tissue as another affection which may cause great distress in breathing by compressing the tube and pushing it aside, and as not being always quite easy of recognition. The possible presence of a foreign body in the upper air passages must never be left out of mind. Laryngeal papillomata are, perhaps, always too slow in their clinical course to be mistaken for croup.

The *prognosis* of croup is always grave. The mortality, under the most favorable circumstances, amounts to 60 or 70 per cent. of the children who are attacked by it, and the younger the patient the less is the chance of recovery. It is only when the case is one of the spurious affections that we can speak confidently of a happy issue.

Treatment of Spurious Croup.—The latter requires no active treatment, but it is usual to give an emetic of two to five grains of powdered ipecacuanha, with or without one-sixth of a grain of tartarized antimony, repeated at intervals of ten minutes until free vomiting takes place. Jenner says that besides removing from the stomach any source of reflex irritation, and relaxing spasm by the nausea and faintness to which they give rise, these medicines also promote secretion from the laryngeal and bronchial mucous membrane and so relieve the catarrh. He has observed that cases left to themselves last much longer, going on for two or three days, whereas as soon as an emetic has acted, the child generally falls asleep at once. However, he follows it up with a dose of calomel and jalap. Another plan of treating spurious croup is that recommended by Graves, of squeezing a sponge out of hot water—as hot as the hand can bear—and applying it beneath the chin, changing it as often as it gets cool, for ten or twenty minutes, until the skin becomes vividly reddened. One must always warn the relatives of a child who has had one attack of spurious croup that it is likely to have others if it is exposed to cold or to wet weather and allowed to get chilled. Such children must, therefore, have special care taken of them, particularly as regards the warmth of their clothing, but they should be accustomed to have the neck and chest sponged over every day with cold water, and Steiner suggests that they should gargle the throat with it several times a day. A child who has repeatedly suffered from this affection is sometimes left with a permanent hoarseness of voice.

Treatment of Membranous Croup. — When a child is attacked with "spurious" croup, the parents commonly send for a medical man at once.

On the other hand, in a case of "genuine" croup (except in that rare form which begins quite suddenly and proves very quickly fatal) advice is very seldom sought until the disease has already existed for some hours, and often not for two or three days. The consequence is that one is very likely to deceive one's self as to the results of treatment if one is not well acquainted with the distinctive features of the two complaints. Cheyne seems to have fallen into this error when he formed the opinion that "if medical assistance were procured early enough, croup would scarcely ever be a fatal disease."

For, when the affection is really of a grave or dangerous character, it seems to be very doubtful whether the morbid process can ever be cut short by emetics or by the administration of tartarized antimony in nauseant doses, as has been recommended by so many writers. I have repeatedly followed this practice, but with no marked success, and it is obvious that one can draw no conclusion from the occasional subsidence of the disease under whatever plan of treatment, since the same result might have occurred spontaneously.

Whether any benefit is derivable from the administration of calomel in croup is admitted on all hands to be very doubtful. Niemeyer, however, was in the habit of prescribing it in doses of a quarter to half a grain every two hours, and he speaks of it as being, at any rate, more useful than the tartar emetic. This writer also advocates in decided terms the application to the outside of the throat of cold compresses frequently changed.

In very young children one cannot attempt in any way to reach the interior of the larynx with topical remedies. It seems to me absurd to apply nitrate of silver to the epiglottis. Older boys and girls may be made to breathe the steam of boiling water, or to employ a spray apparatus; lime water is said by Steiner to be preferable to any other liquid for inhalation, on account of its power of dissolving false membranes.

Even if emetics are useless at the commencement of the disease, they often do good service later on by bringing about the expulsion of pieces of the membrane. Sulphate of copper should then be preferred to antimony or ipecacuanha; from two to five grains are dissolved in an ounce of water, and a teaspoonful is given every ten minutes until the stomach rejects its contents. If this should be followed by a decided increase in the dyspnœa, the emetic may be repeated a few hours later, when a fresh aggravation of the symptoms occurs. But not infrequently such medicines fail in their action at this stage, in consequence (as is supposed) of the carbonic acid poisoning, which renders the pneumogastric nerve insensitive.* Niemeyer recommends that the little patient should then be placed in a warm bath, and have some cold water poured over its head and shoulders. This, he says, almost always brings it round and makes it cough more strongly, and may even lead to the ejection of bits of false membrane. If no such result should follow, there can be no doubt of the necessity for the immediate performance of tracheotomy; and after the operation the stomach often rejects the emetics which had been retained.

But of late years it has become more and more the practice to open the trachea early. If it is certain that the case is not one of spurious croup, I myself think that the operation should very seldom be postponed beyond the time when the chest walls begin to be much sucked in during the act of breathing. Very often, however, a great deal of time is lost from

* The statement in the text is based upon the authority of other writers. I think I have seen cases of croup in which vomiting could not be excited by emetics, although there were no indications of asphyxia, the disease being at an early period of its course; and Dr. Yeats mentions an instance of this kind in which (although the disease was diphtheria) the strength was well maintained, and the child, after two weeks' illness, recovered.

the parents refusing their consent until it becomes apparent that there is no other hope of recovery. And it must be admitted that no period is too late for the possibility of success from tracheotomy: children, actually moribund, have sometimes been saved by it. There is, however, a much better chance of a favorable issue when it is performed at an early stage, before the lungs become congested, with some parts of their tissue entirely collapsed and the rest intensely emphysematous. Moreover, the child may thus be saved from the terrible distress and suffering which accompany the gradual development of asphyxia.

The method of performing tracheotomy is a surgical question upon which I do not presume to touch. But it may be expected that I should say something about the after-treatment. At Guy's Hospital we place the child as near a fire as possible; we make a kind of tent, within which its crib is placed; and we direct a long metal tube from a kettle into the space so shut off from the rest of the ward. Of late years, however, many writers, including Sir William Jenner, have expressed the opinion that such precautions are not only unnecessary but injurious; that physician goes so far as to say that he is sure he has seen cases terminate fatally that would have recovered had they not been thus over-nursed and over-cared for. The plan, introduced by Trousseau, of covering the neck with a large folded piece of muslin, appears to afford sufficient protection against cold; moreover, it keeps the air which enters the trachea moist, giving up to it the aqueous vapor which had been deposited from the breath.

If the operation should not be followed by subsidence of the dyspnœa, it is well to introduce the feathery part of a quill pen into the trachea through the wound or through the cannula, and to twist it round a few times, in the hope that it may entangle and bring away with it some of the false membrane. And when this procedure has succeeded once it may be repeated again and again. A moderately abundant discharge of mucous fluid from the tube is said to be a favorable sign; its remaining perfectly dry is of evil omen.

When a case goes on well after tracheotomy, one can often finally remove the tube on the sixth, seventh, or eighth day. In 87 out of 134 cases collected by Sanné, it was withdrawn between the fourth and the tenth days. Cases in which it has to be retained after the end of the third week are quite exceptional, but it does sometimes happen that the larynx becomes thickened and narrowed by a process of chronic inflammation, and Steiner mentions instances in which its channel was completely closed by adhesions. A practical point of much importance is that when there is partial stenosis the child may appear free from dyspnœa until it goes to sleep. I once made an autopsy in a case of this kind, in which the tube was removed one afternoon, four months after tracheotomy for a scald of the throat, and the child seemed to be perfectly comfortable all day, but died unexpectedly in the course of the following night.

Catarrhal Laryngitis.—From what has been said in the previous section it will be evident that the affection now to be discussed does not altogether correspond in scope with the name under which it is known; for it would be certainly difficult to prove that in cases of "spasmodic" or "spurious croup" (as described at p. 809) catarrh of the larynx is entirely absent. But it is in clinical practice impossible to avoid drawing a broad line of distinction between cases of inflammation of the laryngeal mucous membrane attended with dangerous dyspnœa, and those of which the chief symptom is impairment of the voice. The latter are commonly known by the name of catarrhal laryngitis; and this, again, may be either acute or chronic.

1. *Acute Catarrhal Laryngitis* is a very common affection, but comparatively seldom comes under medical advice. Some persons are very subject to it; whenever they get a cold they become hoarse or lose their voice, and this condition sometimes lasts for several days or even for weeks. It is particularly apt to occur in those who are intemperate, and in those who habitually use the voice a good deal; the reason being that in such individuals the laryngeal mucous membrane is constantly more or less congested. It may also arise as a complication of some other acute disease, especially measles. The chief symptom, besides the impairment of the voice, is the expectoration of a little tough mucus, which comes away with a short hawking effort, hardly amounting to cough. With the laryngoscope, according to Ziemssen, one may find that there is reddening and slight swelling of certain parts of the interior of the larynx, especially the hinder ends of the vocal cords, the inter-arytenoid space, the false cords, etc. During attempted phonation the cords may leave an oval space between them, exactly as though the internal tensors were paralyzed. In somewhat more severe cases Störk speaks of the cords as looking red, dry, and lustreless, or even as being covered with yellowish-green crusts formed of dried-up exudation. They may also become ecchymosed, and sometimes their surface shows superficial excoriations.

The prognosis in acute catarrhal laryngitis is generally favorable. It must not be forgotten, however, that cases in which the early symptoms were those of mere catarrh sometimes run on into œdematous laryngitis; and, on the other hand, the affection, if neglected, may become chronic, and may then be exceedingly intractable. In the treatment, one of the most important points is that the patient should entirely abstain from using the voice. He should remain in a room of which the temperature is kept uniform. Steam inhalations should be frequently employed, the best apparatus for the purpose being (according to Dr. Mackenzie) either the "eclectic" or "Martindale's" or "Bullock's" inhaler. Dr. Mackenzie advises the addition of *tinct. benzoin. comp.*, or of *succus conii* (℥ij with gr. xx of *sodæ carb. exsiccat.*), or of *lupulin* (℥ss) to the hot water used for inhalation, the temperature of which should be from 140° to 160°. The patient should be encouraged to drink freely of demulcent liquids, such as barley water, linseed tea, and the decoction of cetraria or of althæa. Dr. Mackenzie recommends warm milk and seltzer water as a useful beverage. If there is great irritability of the larynx, evidenced by cough and tickling or pricking sensations in the throat, it should be kept in check by opium or morphia, or (according to Störk) by chloral or by lactucarium. It seems to be doubtful whether the application of either hot poultices or cold compresses over the front of the larynx is advisable. Störk and others recommend, at the commencement of the disease, that a strong solution of nitrate of silver should be brushed over the cords, but Dr. Mackenzie thinks that this is better left undone.

The best way to overcome the liability to acute laryngeal catarrh, in those who are subject to frequent attacks of it, is to make the patient gradually accustom himself to daily sponging with cold water, and to exposing the throat in the open air without wraps, even in the winter. No one can avoid allowing a draught to play upon the neck from time to time indoors, or in a corridor, or in passing from house to carriage; and the more a person endeavors to keep the part protected the more surely will he suffer when the occasion arises. It is also important to live, especially during the night, in airy, cool, well-ventilated rooms. Warm, light clothing should be worn. A long stay by the seaside in the autumn, or in the bracing air of Scotland or Switzerland, often does a great deal toward diminishing the susceptibility to catarrh in the following winter.

2. *Chronic catarrhal laryngitis* often arises out of the acute affection, especially in persons who, in spite of hoarseness of voice, persist in attempting to continue duties requiring loud speech. Such cases are common in clergymen, schoolmasters, costermongers. Another frequent cause is extension downward from a "granular pharyngitis." Störk believes that a liability to this form of chronic faucial irritation often passes by inheritance from parent to child, having been originally set up in the former by a course of mercury or of iodine. He also thinks that in other cases inoculation of the nasal cavity with gonorrhœal or even leucorrhœal discharge is the starting point of a catarrh that may last for years. As a rule, chronic catarrhal laryngitis occurs during the middle period of life; it is more common in males than females.

The principal *symptom* of this affection is hoarseness of voice, which may even pass on to complete aphonia. The degree to which the voice is impaired may vary very much at different periods of the day. Mackenzie remarks that it is often greater when the patient first begins to talk after an interval of silence than it is a few minutes later, after he has gone on speaking for a time. The attempt to use the vocal cords often gives rise to a painful sense of fatigue, and there frequently are also complaints of a feeling of dryness or soreness in the throat, and of a tickling sensation, leading to a constant desire to hawk or to cough. The expectoration is scanty, consisting generally of a viscid, gray mucus, but sometimes yellow and puriform.

The laryngoscope shows all gradations, from a slight localized injection and swelling of some part of the mucous membrane of the larynx, up to the most extensive and diffused redness of the whole interior of the organ. Dr. Mackenzie remarks that one vocal cord may be of a bright red color while the other is white; he also says that the congestion may be limited to a small portion of one cord, this being always on the outer or attached side of it. Small pieces of mucus are often seen adhering to the mucous membrane at different points; in the form of whitish threads they may pass across from one cord to the other; in cases of long standing the whole surface of the larynx may be covered with secretion. During vocalization the cords in many cases fail to meet one another. This may be due to swelling of the inter-arytenoid mucous membrane, which is sometimes so extensive as to form a convex projection even when the arytenoid cartilages are as far apart from one another as possible. But in addition to this a paretic state of the muscles is not seldom present. Ziemssen says that this is most frequently unilateral, in which case the opposite cord may pass across the middle line to meet the affected one, after the manner already described at pp. 791–792.

Sometimes certain parts of the larynx are greatly thickened. Ziemssen says that this is especially apt to be the case with the epiglottis, the aryteno-epiglottidean folds, or the false cords. Lewin maintained that thickening of these folds is a particularly marked feature of "clergyman's sore throat," but this is disputed by Mackenzie. Störk relates in detail a case in which the whole of the interior of the larynx was affected with an extreme degree of hypertrophy of its mucous membrane, so that there was great dyspnœa, rendering tracheotomy inevitable. The disease had been of fifteen years' duration. From the right false cord there grew a hard, solid, fibrous tumor, of the size of a hazel nut; every part of the interior of the larynx was thrown into enormous folds and ridges. Polypoid excrescences are, indeed, not uncommon results of a chronic laryngeal catarrh. Another affection which has sometimes been observed under similar circumstances is a thickening of the mucous membrane below the glottis, reducing the channel for the passage of air to a narrow chink or

ring; in almost every instance it has been necessary to open the trachea. Lastly, the vocal cords themselves sometimes become granular on the surface, a condition which has by Türck been designated "chorditis tuberosa" or "trachoma." It has been supposed to depend upon a partial dermoid change in the epithelium; but in one case Wedl found microscopically only a connective tissue and nuclear overgrowth.

Enlargement of the mucous glands is not infrequent in chronic laryngeal catarrh. Dr. Mackenzie speaks of seeing their enlarged orifices upon the epiglottis and upon the posterior parts of the cords in some cases, either as pale specks on a congested surface or as small red circles on a pale surface. Another morbid appearance which Ziemssen regards as an accidental complication of catarrh, is a dilatation of the veins of the mucous membrane, especially upon the epiglottis or upon the cords.

There has been some difference of opinion among writers as to whether chronic catarrhal laryngitis is or is not apt to cause erosion or ulceration of the laryngeal mucous membrane. Störk says that such a result is not infrequent, and that when an ulcer forms over the *processus vocalis*, it may even give rise to the expectoration of blood in sufficient quantity to suggest the presence of tubercular disease of the lungs. This observer also lays special stress upon the occurrence of a vertical fissure in the centre of the inter-arytenoid mucous membrane. This, indeed, is not peculiar to cases of chronic catarrh. Störk speaks of it as being extremely frequent, occurring in as many as 50 per cent. of those who attend his out-patient practice. The fissure, as the result of the traction upon its sides, assumes a rhombic form; the upper part of it, which is alone visible in the laryngeal mirror, appears triangular. Its detection is often very difficult; the patient must be placed in the position which I shall describe further on as required for inspection of the trachea with the laryngeal mirror. The symptoms are not always very marked. Störk had seen a singer of reputation, whose voice remained perfect after such a fissure had existed for many years. Generally, however, there are symptoms which appear to be identical with those of chronic laryngeal catarrh. And sometimes the subjacent arytænoideus muscle becomes paralyzed, in which case the patient's life may be made almost unendurable by the running down of fluid into the larynx whenever he attempts to swallow or even to lie down.

The course of chronic laryngeal catarrh is generally very tedious and protracted, one reason for this being the fact that patients will seldom carry out the necessary treatment with sufficient perseverance, imagining that they ought to be well in two or three weeks, and neglecting all precautions as soon as they begin to improve a little. There are, however, among schoolmasters and clergymen many who go to the opposite extreme, being so nervous and fidgety about their throats that they may almost be classed with hypochondriacs.

A good many cases recover perfectly under careful management. A very important point is that rest should be given to the voice, the patient either using a slate to express all his wants, or at least speaking only in a whisper. He must also abstain from smoking, be very moderate in taking alcohol, and avoid all highly-seasoned foods. If there be evidence of portal congestion, I need hardly say that this must be carefully attended to. Locally, if the larynx is irritable, the patient should make use of a spray containing bromide of potassium or carbonate of soda with a little morphia. But the chief therapeutic results are to be expected from the use of astringent sprays containing tannic acid (gr. j–v ad ℥j) or alum (gr. j–x ad ℥j), or from the systematic application of astringent solutions to the interior of the larynx by means of a brush. Dr. Mackenzie says that what he generally employs is a solution of chloride of zinc (gr. xv ad ℥j); he

applies this daily during the first week, on alternate days during the second and third weeks, and afterward less frequently. Ziemssen lays great stress on the value of the topical use of the solid nitrate of silver fused upon the end of a laryngeal probe; this he repeats at intervals of a week or a fortnight; it causes violent spasm, which, however, is at once relieved by filling the throat with cold water. When there is an inter-arytenoid mucous fissure, the application of solid caustic is the best treatment; this must, however, be done with great exactitude, for if the surrounding healthy mucous membrane is touched instead of the sore, the patient's sufferings will be made worse rather than better.

In many cases of chronic catarrh of long standing, electricity applied locally aids in restoring the voice; and some patients find benefit from a course of the waters at Aix-les-Bains or at Ems.

Tubercular Disease of the Larynx and Trachea.—It is in the medical literature of the end of the last century that the earliest allusions are to be found to what was then, and commonly is now, termed *laryngeal phthisis*. This name, however, is not a good one, because it is apt to suggest the idea that an affection of the larynx may give rise to wasting and to other symptoms resembling those of ordinary pulmonary phthisis, without there being any mischief in the lungs. That such is sometimes the case has, indeed, been asserted by Trousseau and by some other writers. But I believe that no pathological proof of it has ever been brought forward. The experience of all pathologists is that in every instance in which a tubercular affection of the larynx is found after death the lungs are invariably found to contain tubercles, and present destructive changes which have evidently been of long standing, even though there may have been little or no auscultatory evidence of the existence of disease in them during life. So far as I am aware there is no recorded instance in which, when death has been due to tuberculosis of other organs, leaving the lungs healthy, the larynx has been found to be affected with tubercular lesions; nor, again, have such lesions ever been found in the bodies of those who have died of acute general tuberculosis, unless there was old as well as recent disease of the lungs. Consequently, in spite of the *à priori* probability that tubercles should sometimes form in the larynx earlier than in any other part of the body, the pathological evidence points at present to the conclusion that this is never the case. It may, indeed, be urged, on the other side, that the laryngeal affection almost always lasts for a considerable time before death occurs, and even that its capability of producing a fatal issue by itself is doubtful. But I certainly think that either by interfering with deglutition, or by setting up necrosis of cartilages, or œdematous laryngitis, it may sometimes greatly shorten life, and consequently it appears to me that if tubercular disease of the larynx ever arises before similar mischief in the lungs has begun, one ought, sometimes at least, to see in the post-mortem room cases in which, with an advanced laryngeal affection, the lungs are either quite healthy or in a very early stage of phthisis.

In speaking of "tubercular disease of the larynx" I have not forgotten that it has been doubted by many pathologists whether in "laryngeal phthisis" local tubercular lesions are really present. The view that the laryngeal affection which accompanies pulmonary phthisis is (like the lung affection) dependent upon the formation of tubercles was originally advanced by Laennec. It was, however, soon afterward disputed by Louis, and since that time pathologists have ranged themselves into two camps with regard to this question, some affirming, others as positively denying it. There can, in fact, be no doubt that in the larynx one very seldom sees conspicuous

gray or yellow tubercles, such as, for example, are observed with great frequency in the ileum in the very same cases. But I have myself always maintained a belief that the laryngeal affection was really tubercular, basing this opinion not merely on the very large proportion of cases of pulmonary phthisis, especially with tubercular ulceration of the intestine in which this affection occurs, but also on the fact that many laryngeal ulcers have thick caseating edges which appear to me to be characteristic of a tubercular process. Recently, however, the matter has been taken out of the range of speculation by the careful investigations of Heinze, of Leipzig, who published a monograph on the disease in 1879. The basis of his work was a microscopical examination of the tissues in fifty cases of phthisis, in each of which there was disease of the larynx or of the trachea, or of both together. For it is to be observed that in many instances the morbid process involves not merely the upper but also the lower air passages, extending even sometimes into the bronchial tubes; and, for the sake of convenience, I shall in this place discuss all such affections together, inasmuch as there are no special symptoms that characterize the tracheal or the bronchial lesions. Now, Heinze found that in forty among his fifty cases tubercles were plainly recognizable in the larynx; in thirty-nine of those forty there was ulceration; in one there was merely a tubercular infiltration of the mucous membrane without ulceration; in eleven of the forty there was in the trachea tubercular ulceration likewise; in eight the trachea contained ulcers which could not be shown to be tubercular. With regard to the remaining ten cases he found that in eight there were in the larynx ulcers of which the tubercular nature could not be demonstrated, but in five of these the trachea showed tubercular ulcers, in two the larynx was healthy, but in the trachea there were tubercular ulcers. In other words, there were only three out of the whole fifty cases in which tubercles were not detected either in the larynx or in the trachea, or in both. The tubercles themselves were plainly visible to the naked eye in hardened sections, but they could not be seen in the recent textures. They lay partly in the mucous membrane, partly in the submucous tissue, but always on a plane superficial to the laryngeal mucous glands. They had often undergone more or less complete caseation. With regard to the characters of the laryngeal ulcers which Heinze classifies as non-tubercular, he states that there was nothing in their appearance to distinguish them from those that were tubercular. In every instance, too, they were very superficial, in fact, little more than erosions, and they were generally confined to the vocal cords. It is, therefore, quite open to question whether they were not originally preceded by a formation of tubercles, which had softened and been cast off by ulceration, as was believed by Virchow to be very frequently the case when a laryngeal phthisis failed to show definitely tuberculous character. In the trachea, on the other hand, there seems to be no doubt that ulcers occur which are really non-tubercular; these appear as minute depressions of yellow color surrounding the mouths of the tracheal glands. In one point Heinze appears to me to go too far; this is when he asserts, on the strength of his observations as to the exceedingly small size of laryngeal tubercles in general, that those writers were in error who have maintained that in some exceptional instances they have recognized obvious tubercles in the larynx in making autopsies or even by the laryngoscope during life. Türck, for example, figures a larynx from the dead subject, in which besides tubercular ulcers there is what he describes as a miliary tuberculosis plainly visible to the naked eye. And I certainly think I have several times seen tubercles both gray and caseating in the laryngeal tissues. Considering how variable is the size of tubercles in other organs, I can see no reason why they should not in the larynx be much larger in some cases than in others.

The facts that tuberculosis of the larynx never occurs independently of pulmonary phthisis, and that it perhaps never even precedes the lung affection in order of development, cannot but suggest the idea that the upper air passages become infected as the result of the passage through them of tuberculous sputum. This idea was originally suggested by Louis, but he thought that the sputum acted merely as an irritant upon those parts with which it came into contact. At the present time one can form a far more definite conception of the way in which tubercle bacilli, settling upon the laryngeal or upon the tracheal mucous membrane, may germinate and invade the tissues. It does not seem to me that the possibility of such an occurrence is at all invalidated by the observations of Heinze as to the commencement of laryngeal tuberculosis beneath the intact epithelium. Ziemssen even maintains that a continuous tract of ulceration can sometimes be followed from a vomica in the upper lobe of one lung along the corresponding bronchial tube (which alone of all the bronchial tubes may be affected) through the trachea to the larynx. But, of course, it is quite conceivable that, without there being any vomica, tuberculous sputum from the lung may infect the larynx. And conversely—if laryngeal tuberculosis really does ever precede the pulmonary affection in point of time—one can easily imagine that secretion from the larynx may descend the air passages and infect the lungs.

That tubercular disease of the larynx and of the trachea should be more frequent in males than in females might have been anticipated from the fact that this is the case with pulmonary phthisis, but the preponderance of males over females is, in reality, far greater; according to Heinze 33.6 per cent. of male phthisical patients have ulceration of the larynx, but only 21.6 per cent. of female phthisical patients. The age at which tubercular laryngeal affections are absolutely most frequent is between twenty-one and thirty; but among fatal cases of phthisis the proportion in which the larynx is found diseased is relatively larger at a more advanced age, namely, between forty-one and fifty for males, between thirty-one and forty for females. During childhood, tubercular disease of the larynx is not common; among nearly 400 cases Heinze found only nine in patients under the age of fourteen; in none of these was there ulceration of the trachea; one was an infant of eleven months. The nature of a man's occupation seems to have no marked influence in modifying his liability to have his larynx affected, assuming him to become phthisical. Heinze found that the proportion of cases in which laryngeal complications occurred was high among consumptive butchers and tailors, as well as among open-air singers and others whose throats were exposed to local irritation in various ways.

The *symptoms* of tubercular disease of the larynx, of course, vary with its exact seat. When the epiglottis is affected, or any other part of the upper orifice of the larynx, there is often extreme dysphagia, the attempt to swallow even fluids causing very great pain and distress. But Heinze relates one case in which this symptom remained entirely absent, although the epiglottis was greatly swollen and thickened. The voice becomes hoarse and weak, and ultimately it is often reduced to a whisper. In some cases in which the true cords are entirely destroyed by ulceration, it is believed that the false cords sometimes vibrate so as to produce harsh, deep tones. The effort to speak is often painful, producing a feeling of soreness in the throat. The cough becomes weak and hoarse and toneless.

It must not be supposed, however, that marked subjective symptoms occur in every case of phthisis in which one subsequently in the post-mortem room finds that the larynx is affected with tubercular disease, even when it has advanced to ulceration. A few isolated ulcers are often found when no

laryngeal affection had been suspected during life; and in working at practical morbid anatomy I acquired a decided impression that an extensive superficial affection of the mucous membrane possessed clinical significance far more constantly than did the presence of localized ulcers, however deeply they might penetrate into the laryngeal structures. On the other hand, there are many cases of phthisis which are attended with hoarseness of voice, or even with aphonia, but in which no tubercular affection of the larynx can be detected, whether during life or in the dead body. In Virchow's "*Archiv*" for 1877, Fränkel, of Hamburg, endeavored to find an explanation of such cases in the occurrence of lesions in the laryngeal muscles, the fibres which he showed to have undergone a granular change ending in a complete absorption of their substance and emptying of the sarcolemma, while at the same time the nuclei of the *perimysium internum* underwent increase. It does not appear that the patients who furnished the material for his observations had had any definite paralysis, and, indeed, the lesions which he detected were always distributed equally over all the muscles. But paretic states of the *thyro-arytænoidus internus* are said to be not uncommonly present in phthisis, and Gerhardt has described the occurrence of paralysis in the right recurrent nerve as the result of its having become involved in a thickened mass formed by the pleura covering the apex of the right lung. In the laryngeal mirror what chiefly characterizes cases of phthisis attended with impairment of voice, when there is no local tubercular disease, is the extremely anæmic state of the mucous membrane of the interior of the larynx.

The laryngoscopic appearances which indicate tubercular disease of the larynx differ greatly in different cases. Heinze lays great stress upon the recognition of tubercular infiltration of the mucous membranes, which (he says) is quite peculiar to this disease, and which was present in twenty-one of his forty cases. Its most frequent seats were the false cords and the ary-epiglottidean folds. He speaks of it as forming in the dead body a smooth, tense swelling, of a grayish-white or grayish-yellow color, often presenting on its surface the appearance of fine, pale yellow granules, or spotted with points of hemorrhage. Over the summits of the arytenoid cartilages tubercular infiltration gives rise to swellings which by Heinze are compared to two rounded sugar loaves; Mackenzie describes them as "pyriform." The epiglottis, when it is affected, appears rounded and thickened, or (as Mackenzie says) "turban shaped." The false cords become greatly swollen, so that they lie in the same vertical plane with the true cords, or even overhang them, while the entrances into the ventricles of Morgagni are greatly narrowed, or seem to be entirely obliterated. Thickening of the inter-arytenoid mucous membrane gives rise to a local bulging or excrescence at the back of the glottis, to which Störk draws attention as characteristic of tubercular disease, it being all the more so because neither polypi nor other new growths are ever seen in that position. The cords themselves very rarely exhibit a true tubercular infiltration, but they may become swollen and rounded, a change which Heinze found to be dependent upon the presence of numbers of small round cells between their fibres. In the trachea, it is only in the posterior membranous part that tubercular infiltration occurs.

But in a great many cases of tubercular disease the diagnosis, so far as the laryngoscope is concerned, is based mainly upon the presence of more or less numerous ulcers. According to Heinze, their most frequent seat is upon the vocal cords; among his fifty cases ulceration of the cords was present in not fewer than forty; on both sides in twenty-seven, on one or the other side separately in ten, at the anterior commissure in three. Sometimes the ulceration was limited to a small part of the cord, sometimes

it affected the whole length; in eleven cases one or both of the cords was completely destroyed. In the laryngeal mirror an ulcer upon one of the cords may appear either as a narrow linear fissure, or as an actual excavation of its edge, situated upon a more or less reddened surface. When the process of ulceration is further advanced, it often happens that the cord looks as if it were split up into two or three separate longitudinal bands, with very irregular edges, arranged one above the other like a short flight of steps. The false cords are comparatively seldom ulcerated; among Heinze's cases only in fourteen, of which there were eight in which the affection was bilateral. Over the arytenoid cartilages ulcers were present in twenty-three of Heinze's cases, sometimes toward the bases of the cartilages, sometimes upon their summits or even toward the pharyngeal surface. Those which lie toward the bases of the cartilages are almost always bilateral. They are seldom, if ever, visible in the laryngeal mirror. They have a peculiar tendency to penetrate deeply into the tissues, so that they often reach the perichondrium and lead to necrosis of the cartilages. In the dead body they are seen to have their outline irregular, their edges smooth or fringed with papillary outgrowths, and their surface uneven, perhaps of a dirty-gray color. The epiglottis was ulcerated in twenty-six of Heinze's cases, generally upon its laryngeal surface, sometimes upon its border, never on its lingual surface. The appearance of epiglottic ulcers varies in different cases; often there are great numbers of small, round, shallow sores; sometimes they have run together into a large, irregular excavation. The surface upon which they lie is often but little reddened. In some cases the substance of the epiglottis itself becomes eaten away from its margins inward, so that it may present one or more deep fissures, with pointed processes between them; or it may even be reduced to a mere stump. In such cases the ulceration, of course, affects the lingual surface as well as the laryngeal; indeed, it often spreads for some distance upon the base of the tongue. In such cases, too (and, indeed, in many others), the whole surface of the larynx is often covered with ulcers varying in shape, in size, and in depth.

It must not be imagined that the detection of ulcers by means of the laryngeal mirror is always an easy matter, even when they come fully within the field of observation. Störk remarks that they are often recognized, not so much by the presence of an obvious depression in the mucous membrane, as by a change in its color and a deficiency in the lustre naturally belonging to its epithelium. Unskilled observers are often misled by patches of puriform mucus lying upon the surface of some part of the larynx, which they take for ulcers.

Even when the presence of ulceration of the larynx is established, it still remains to be considered whether the disease is tubercular. We have seen that in catarrhal inflammation ulcers are seldom found. And, practically, the diagnosis generally lies mainly between "laryngeal phthisis" and syphilis, the points of difference between which two affections I shall leave to be discussed further on. It must not, however, be supposed that, in the post-mortem room at any rate, ulceration of the larynx is of rare occurrence, apart altogether from the diseases with which it is commonly associated. In acute pneumonia ulcers over the arytenoid cartilages are, I think, tolerably frequent; and I have seen ulceration there, or upon the vocal cords, in two cases in which there was double pleurisy with pericarditis, in two cases of erysipelas, in one case (probably pyæmic) in which there were abscesses both in the liver and in the brain, and in two cases of Bright's disease. One of these last-mentioned cases occurred in a man, aged twenty-four, who had been hoarse for three months before his death; laryngoscopically, all that was observed was that his cords were at one time

reddened, but afterward pale; at the autopsy I found on each cord a narrow linear ulcer extending over a quarter of its length. Heinze gives details as to eight cases in which ulcers were found in the larynx after death from various diseases.

I have met with three cases of phthisis in which the larynx after death presented what appeared to be the cicatrices of ulcers that had healed; and both Ziemssen and Heinze have recorded similar instances, some of which occurred in patients who had been the subjects of repeated laryngoscopical examinations. One of Heinze's cases is that of a man whose right cord was much ulcerated; two or three years later this was found to have healed, and there were, also, cicatricial bands on the left cord, which must have become affected in the interval. Both this patient and another appeared to owe their recovery to residence in a southern health resort. Of course, however, it remains a question whether the ulcers in either case were actually tuberculous in a strict sense of the term. At the meeting of the International Congress in London, in 1881, Rossbach and some other observers spoke very positively with regard to the occasional "curability" of "laryngeal phthisis," while Krishaber virtually denied it. The former speaker attributed considerable value, at an early stage of the disease, to the inhalation of antiseptic agents; the latter declared that no such treatment was of the slightest use. Ziemssen lays stress upon the importance of giving absolute rest to the voice by maintaining perfect silence, or speaking only in a whisper, for months together. Dr. Mackenzie speaks of the local application of perchloride of iron (℥j ad ℥j) as sometimes greatly diminishing the irritability of the mucous membrane and so relieving the troublesome cough. Nothing, however, is so serviceable in this disease as the local insufflation of morphia as a powder, in a dose of ⅛–½ a grain mixed with half a grain of starch. A special instrument, called an "insufflator," is made for the purpose; it consists of a hollow vulcanite tube, one end of which is bent downward so that it can be directed toward the orifice of the larynx; in the middle of the tube is a hole, through which the powder is introduced, and which is then closed by a movable covering; the other end of the tube is connected with a piece of elastic tubing. In using this instrument the operator places the elastic tubing in his own mouth, and introduces the vulcanite tube into the mouth of the patient. He then blows the powder down into the larynx, just at the moment when the patient is drawing his breath. Dr. Mackenzie says that as the greatest diminution of the sensitiveness of the affected parts occurs in rather less than an hour, it is well, when there is much pain in swallowing, to introduce the morphia at about that interval before the time of taking food. It may be repeated twice daily. If distress is caused by liquids entering the air passages, they should be thickened with arrow-root or corn flour. Dr. Mackenzie also remarks that the patient is more likely to swallow well in taking off a good draught than in sipping. Sometimes it is necessary to administer food through an œsophageal tube for weeks together. Tracheotomy is now and then required when there is extreme dyspnœa; but this operation seems in no way to retard the further progress of the laryngeal affection.

The duration of life in cases of tubercular disease of the larynx is seldom long. Dr. Mackenzie gives a list of 100 cases, of which seventy-nine ended fatally in from six months to two and a half years after "throat symptoms had become troublesome;" in nine only did death occur within six months; twelve were prolonged over a period of from thirty to forty-nine months. Ziemssen, however, seems to maintain that the quickly fatal course of "laryngeal phthisis" depends rather upon the coexistent lung mischief developing itself with great rapidity, than upon any marked tendency on the part of the affection of the larynx to shorten the patient's

life. According to this writer, when the pulmonary disease assumes a chronic form, laryngeal ulcers may exist for years.

Lupus of the Larynx.—Within the last few years a small number of cases have been recorded, in which lupus of the skin has been associated with a like disease of the epiglottis, and even of the interior of the larynx. Such an affection is characterized by the presence of nodules which may be as large as peas, and of ulcers which may have undergone a partial cicatrization. It is mentioned that the ulcerated epiglottis often looks as though a heart-shaped piece were taken out of the middle of its free edge. Otherwise there is nothing in the laryngoscopic appearances which could distinguish lupus of the larynx from the effects of syphilis; nor are there any peculiarities in the symptoms, which consist of hoarseness, sore throat, dysphagia, and perhaps dyspnœa. Consequently, the diagnosis of a case which Ziemssen gives as one of laryngeal lupus, in a girl of twelve, whose skin was free, depends largely upon the fact that treatment with iodide of potassium proved a complete failure. What is recommended for this disease is the systematic administration of cod-liver oil for a length of time, with energetic cauterization by means of nitrate of silver. An arrest of its further progress may, perhaps, be hoped for, but hardly a cure.

Syphilitic Disease of the Larynx.—Laryngeal affections resulting from syphilis are by no means rare, whether relatively to other effects of the venereal poison, or relatively to other diseases of the larynx. It seems probable that their occurrence is to some extent determined by the existence of local causes of irritation. Thus Ziemssen speaks of them as being especially frequent in persons whose throats are exposed to cold, and who abuse the voice, as, for example, in costermongers. And Dr. Mackenzie found that whether in the earlier or in the later stages of syphilis the larynx was far more apt to suffer during the winter than during the summer. The majority of patients are, as might be expected, between the ages of twenty and forty, but among those who suffer from laryngeal affections as remote sequelæ of syphilis it is not uncommon to find persons up to the age of fifty or sixty, or even seventy. A few instances have been observed in which the larynx has become diseased as the result of inherited syphilis; and Fränkel has recorded an instance in which a syphilitic infant, less than three months old, became the victim of a fatal laryngeal stenosis, as the result of perichondritis with exfoliation of the cricoid and of the left arytenoid cartilages. Some of the earlier laryngeal manifestations of acquired syphilis may occur within two or three months after infection; its more remote effects may appear five, ten, twenty, or even thirty years afterward, when all other indications of the disease have long since disappeared, and when the patient, indeed, may have almost forgotten that he had ever taken it. However, a collection of some twenty cases that have at different times come under observation in the post-mortem room at Guy's Hospital gives me a decided impression that severe lesions of the larynx are much more often associated with cutaneous eruptions and other obvious signs of syphilis than are other visceral syphilitic affections, as (for example) those of the brain or of the liver.

In some cases, especially those of recent infection, the larynx shows nothing more than a mere superficial catarrh. In this there is nothing characteristic; Ziemssen warns his readers against supposing that a livid-red or dirty-brown injection of the mucous membrane justifies a diagnosis of syphilis. Nor does the protracted duration nor the obstinate recurrence of a laryngeal catarrh afford grounds for concluding that it is not simply inflammatory, whereas in the case of the pharynx such points sometimes

possess a marked significance. Störk speaks of syphilitic catarrh of the larynx as so transitory that patients seldom come under treatment for it.

Next in order of development come "mucous patches," or "flat condylomata." As to their frequency, widely different statements are made by different writers. Some almost deny that they ever occur in the larynx, others say that they are often to be seen laryngoscopically. Ziemssen speaks of their chief seats as being the cords, the posterior laryngeal wall, and the false cords. They may also appear on either surface of the epiglottis. According to Dr. Mackenzie they differ from pharyngeal condylomata in being yellow rather than white, and in being generally accompanied by less marked congestion of the surrounding mucous membrane; he also says that they are less apt to undergo superficial ulceration, and that they generally disappear quickly even without treatment.

At a later period of the disease gummata are of not infrequent occurrence in the laryngeal mucous membrane and submucous tissue. They are described as generally forming small rounded elevations, from the size of a pin's head to that of a small pea, of the same color with the rest of the laryngeal surface, isolated or collected together into masses of considerable size. They may be seated upon the epiglottis, the posterior wall of the larynx, the false cords, or even the surface below the glottis. In 1874 Mr. Norton showed to the Pathological Society a gumma larger than a pigeon's egg, which occupied the right aryteno-epiglottidean fold, and reduced the air passage to a mere chink, so that it caused suffocation. Laryngeal gummata often ulcerate, but sometimes they remain stationary for a long time, and finally disappear by absorption.

Syphilitic ulcers in the larynx are often observed. During the earlier stages of the disease they are generally superficial; at a more advanced stage they are apt to eat their way deeply into the tissues, causing great destruction. It has been much discussed whether syphilitic ulcers present any characters by which they can be distinguished from non-syphilitic ulcers.

Türck maintained that some of these cases can be recognized at the first glance by their more or less circular form, by their excavated surface coated with a whitish-yellow material, by their edges, which are sharp, sometimes much raised, and surrounded by an inflammatory areola. It is especially from tubercular ulcers that the diagnosis has to be made. A point of great importance is the comparatively rapid development of syphilitic ulcers. Störk remarks that a patient with extensive destruction of the epiglottis as the result of syphilis may still retain a fresh, healthy appearance, which is never the case where such disease is of tubercular origin. Tubercular ulcers are smaller than syphilitic, except when several have coalesced together; they are often numerous, affecting both sides of the larynx at once; they are generally seated upon a surface which is pale and anæmic instead of being reddened. But ulcers of the larynx sometimes occur in persons who, having had syphilis, are also affected with phthisis; it may then be quite impossible to declare positively what is the nature of the laryngeal affection.

In some difficult cases great help is afforded by the presence of ulceration of the pharynx, or of the base of the tongue, which in tubercular disease of the larynx is very infrequent. Indeed, Ziemssen remarks that in most cases destructive ulceration of the larynx is preceded by a like affection of the fauces, which passes by continuity of surface from the pharyngeal wall to the side of the epiglottis. At the seat of the earlier lesions cicatrices may very often be observed to have already developed themselves, and this is conclusive as to the syphilitic character of the affection, for in tubercular disease such partial cicatrization is never seen.

Cicatrices, indeed, frequently form within the larynx itself, and produce an extraordinary amount of deformity. Sometimes a web is formed between the cords, as in several cases recorded by Elsberg, of New York. In other cases the epiglottis is dragged down and fixed to the side of the pharynx, or the parts forming the entrance of the larynx may be puckered together so as to reduce it to a small round hole. Papillary outgrowths of considerable size are occasionally developed in the neighborhood of syphilitic cicatrices and may play an important part in increasing the obstruction to the passage of air.

The *symptoms* of syphilitic disease of the larynx generally include hoarseness of voice, which may pass on to complete aphonia. Pain is often entirely absent, but if the epiglottis or some other structure at the entrance of the larynx is affected swallowing may be exquisitely painful, although even in that case there may be an entire freedom from pain at all other times. It is wonderful how well some patients manage to swallow, even when they have lost a large part of the epiglottis; the base of the tongue is carried backward, and keeps even fluid from passing into the interior of the larynx. Cough is often troublesome, and if there is extensive ulceration, muco-purulent exudation and blood may be expectorated in considerable quantity. I have notes of one case at Guy's in which hæmoptysis occurred to such an extent that the patient was thought to have phthisis, and Türck has recorded an instance of fatal hemorrhage from an extensive ulcer of the left side of the interior of the larynx, exposing a necrosed piece of the cricoid cartilage. Otherwise it is chiefly by the supervention of an œdematous laryngitis, or perichondritis of the larger cartilages, that syphilitic disease of the larynx tends directly to destroy life. But Türck and Ziemssen point out that, in the case of the arytenoid cartilages, an incurable necrosis is far less apt to follow deep ulceration of the mucous membrane covering them when the disease is syphilitic than when it is tubercular. In some instances that have occurred at Guy's a fatal termination has resulted from pneumonia, which was probably set up by the entrance of purulent matter from the affected parts into the bronchial tubes.

The *treatment* of syphilitic disease of the larynx should generally consist mainly in the administration of mercury, and inunction is perhaps to be preferred to other methods, as it is often important to produce an effect quickly. Spray inhalations with a weak solution of bichloride of mercury are said to be sometimes very serviceable. Sometimes it is advisable to brush over the affected parts with dilute tincture of iodine, or with a mixture of two parts of iodine, two of iodide of potassium, and ten of glycerine. When mercury has already been used freely full doses of iodide of potassium may, of course, be prescribed internally. Tracheotomy is sometimes necessary, and cicatrices may have to be cut through with endo-laryngeal instruments.

Inflammation of the deeper Laryngeal Structures.—The inflammatory affections of the deeper laryngeal structures fall under two heads, those of the submucous tissue and those of the cartilages. The former may be described under the name of "Œdematous" or "Phlegmonous Laryngitis," the latter under that of Laryngeal Perichondritis.

Œdematous or Phlegmonous Laryngitis.—This is the affection which is commonly but inaccurately termed *œdema glottidis*, that name having been originally given to it early in the present century by Bayle. The inaccuracy is twofold; on the one hand, the part diseased is not the *glottis*, or space bounded by the vocal cords, for (except in very rare instances) they remain free from swelling, which really affects the entrance of the larnyx above

them; on the other hand, the morbid process is by no means merely an *œdema*, but assumes any gradation from an exudation of serum loaded with leucocytes into the submucous tissue up to a diffuse purulent infiltration, or even the formation of an abscess. Consequently there are some cases to which the designation œdematous laryngitis, others to which that of phlegmonous laryngitis is the more applicable. It is, however, necessary to use one name rather than the other in the present chapter, and perhaps the former is to be preferred as somewhat less narrow in its scope than the latter. I am disposed to think that a better name than either would be "submucous laryngitis."

I must not be understood to mean that there is no such thing as an *œdema* of the larynx in the sense of a simple non-inflammatory dropsy. On the contrary, that affection is very frequently seen in the bodies of those who have died of Bright's disease, or of heart disease, the epiglottidean folds forming watery swellings which may be of very considerable size; but so far as I have observed, such cases present no special symptoms during life, and the dropsy of the larynx has no clinical significance whatever.

Very different is the state of affairs in a true œdematous laryngitis, which is one of the most rapidly fatal of all diseases. The appearances, whether at the bedside or in the post-mortem room, vary somewhat with the exact locality of the inflammation. When the epiglottis is involved it forms a turgid round mass, perhaps as large as the end of one's thumb, and often of two lateral rounded halves pressed closely together so as to leave only a narrow gap between them. This may be either felt by the finger passed into the fauces or seen in the laryngeal mirror; it may even be directly visible when the tongue is depressed with a spatula. Sometimes the tissues in front of the epiglottis are included in the swelling. In other cases the parts most affected are the ary-epiglottidean folds, which may be converted into two large globular masses, tense and resisting, so that they feel like swollen tonsils. The mucous membrane covering the *cornicula laryngis*, and that between the arytenoid cartilages may also share in the morbid process, in which case the movements of the cartilages are greatly impeded. Within the larynx the inflammation usually affects the false cords, which bulge downward as well as inward, so as to overhang and conceal the true cords. The latter are themselves very seldom involved in the swelling, but Risch has recorded ("*Berl. Klin. Woch.*," 1866) a case in which, having actually removed the larynx within ten minutes of the patient's death, he found the true cords swollen to the breadth of half a centimetre and pressed against one another so as completely to close the glottis. A similar instance occurred at Guy's Hospital in 1873; the patient was a woman who was found moaning on the ground in the street, and who died before she could be brought into the ward. In some cases the effusion is limited to the structures below the cords, constituting what Gibb termed a "subglottic œdema." Mackenzie speaks of such cases as generally chronic rather than acute, but Ziemssen cites five instances, observed by Burow, Rauchfuss, and himself, in each of which the affection in question was clearly recognized by the laryngoscope, the symptoms being of a grave and urgent character and very rapidly developed. The color of the affected parts as seen during life is generally a very bright red. After death they look much paler, the ary-epiglottidean folds in particular appearing gelatinous, and having often a yellowish gum color, from infiltration of pus into their tissue. When they are incised in the post-mortem room, however, it is often found that no fluid escapes from them, even under gentle pressure. Not uncommonly the inflammation extends to the laryngeal muscles, which may be full of suppurating points.

Among the symptoms of œdematous laryngitis the most important is dyspnœa, which may increase with extraordinary rapidity until it destroys life by suffocation. Inspiration is commonly attended with a loud whistling sound. There is some pain in the throat, increased by speaking, and the larynx is tender when handled in the neck. The voice is not necessarily much altered, but it commonly soon becomes hoarse and extinct. The cough is hollow or it may be toneless. If the voice, as is sometimes the case, becomes affected before the breathing, the explanation seems to be that the interarytenoid mucous membrane has been the first to become swollen. Another sign that this part is attacked is, according to Störk, an incessant dry, jerking cough. If the epiglottis or other parts bounding the entrance to the larynx are inflamed at the beginning, there is intense pain in swallowing. Much distress is given to the patient by the accumulation of buccal and pharyngeal secretions, which he is unable to get rid of.

Störk lays great stress upon the significance of dysphagia, as the earliest indication of commencing laryngitis in some cases, and upon the importance of using the laryngoscope, whenever there is difficulty of swallowing, not obviously accounted for by an affection of the fauces. A thorough examination with the finger often at once clears up all doubt as to the nature of the disease. But this must be done with some caution, for it is very apt to set up an attack of suffocative dyspnœa.

Œdematous laryngitis occurs chiefly in young adults between the ages of eighteen and thirty-five; in males more often than in females. It is rarely seen in children. As a primary affection, it appears to be generally due to some septic influence, such as is commonly vaguely described as "blood poisoning." Dr. Mackenzie says he has met with it in hospital physicians, in medical students, in nurses, as well as in persons exposed to emanations from bad drains. Sometimes it appears to be directly dependent upon exposure to cold, as in Trousseau's case of a man who, having drunk too freely at a wine-shop, was turned out into the street on a cold night, and fell asleep there, to wake with a violent sore throat, which in an hour or two became attended with the most extreme dyspnœa. Sometimes it arises by direct extension from faucial erysipelas, sometimes it occurs in the course of smallpox, or of enteric fever. It is frequently the immediate cause of death in the *angina Ludovici*, a diffuse inflammation of the connective tissue of the neck, with brawny infiltration, which may or may not pass on to suppuration. In a case that occurred at Guy's Hospital in 1863 it was secondary to chronic suppuration in the fibrous tissues about the hyoid line. It is often developed by extension from perichondritis of the laryngeal cartilages, or follows chronic tubercular affections of the mucous membrane. Other frequent causes of it are scalds of the throat, the entrance of a foreign body into the larynx, the swallowing of mineral acids and other corrosive poisons.

I must not omit to mention that some writers describe it as being sometimes the immediate cause of death in cases of Bright's disease. Fauvel, indeed, maintained that it may be the earliest symptom of that disease. The only case in which I remember to have made an autopsy in which severe affection of the submucous tissue of the larynx occurred as a secondary complication, was in a man, aged twenty-seven, who died in 1878, of epileptiform convulsions. He had complained of sore throat and of shortness of breath, but no symptoms had been observed indicative of laryngeal mischief. Both epiglottidean folds were found infiltrated with pus, but especially the left one. There was also much exudation of puriform lymph round the pharnyx and at the base of the tongue. In the following year I examined the body of a man, aged thirty-four, who was in the hospital with cirrhosis of the liver, when he was attacked one day, at 11 A.M., with sore

throat, followed at 4 P.M. by severe laryngeal symptoms, and by rapidly fatal dyspnœa at 10 P.M. I found the left epiglottidean fold moderately swollen, with an effusion of serum, the right one smooth and shining, and greatly enlarged by infiltration with a semi-solid purulent substance. In a case of Bright's disease that terminated fatally by dyspnœa in 1866, the autopsy showed that the course was not an ordinary œdematous laryngitis, but the exudation of a shreddy, lymph-like material upon the mucous membrane of the larynx below the cords, extending down to about the eighth ring of the trachea.

It is surprising how rapidly the symptoms of œdematous laryngitis are sometimes developed. Ziemssen relates the case of a young man who was attacked with extreme distress of breathing after eating some bread, and who ran off to Ziemssen, thinking there must have been in it a needle which was sticking in his throat. The laryngoscope showed that the right "sinus pyriformis" (outside the larynx) contained a pointed splinter of wood, which was at once removed with a pair of forceps. Only a quarter of an hour altogether had passed, yet there was considerable œdema of the right ary-epiglottidean fold and the entrance of the larynx generally was much injected. In cases of poisoning by corrosive liquids, laryngeal symptoms sometimes, after setting in suddenly and quickly reaching an alarming height, so that the necessity for tracheotomy appears imminent, subside with no less rapidity.

In treating a case of œdematous laryngitis it is often well, at the commencement, to apply leeches to the neck over the sides of the larynx. Dr. Mackenzie recommends that bromide of potassium should be given freely and that the patient should constantly suck ice. Trousseau speaks highly of spray inhalations impregnated with tannin or alum. If, however, the entrance of the larynx is found to be very greatly swollen, the best thing to do is to scarify the tissues thoroughly, so as to give the exudation an opportunity of escaping. A number of shallow parallel incisions should be made, the best instrument for the purpose being a "laryngeal lancet" or small double-edged knife mounted on a curved stem; but in an emergency an ordinary bistoury, covered up with adhesive plaster to within a quarter of an inch of its extremity, answers very well. If relief does not speedily follow, tracheotomy must not be delayed. Above all the patient must not be left, even for a few minutes, until an opening into the trachea has been made, for a paroxysm of dyspnœa may set in at any moment, and may end fatally before there is time to fetch a surgeon.

Laryngeal Perichondritis.—Several writers in the latter part of the eighteenth or in the earlier part of the present century recorded cases attended with suppuration round, and destruction of, one or more of the laryngeal cartilages. And until a comparatively recent period, the usual opinion was that the disease began in the cartilages themselves, which (it was supposed) first became ossified and then necrosed. But now it seems to be generally admitted that the starting point of such cases is rather in the perichondrium. Indeed, as far back as 1850, Dittrich, a very keen observer, pointed out, in an interesting article in the "*Prager Veirteljahrschrift*," that in a young subject it is not uncommon to find, when a small portion of the cricoid, happening to lie bare in an abscess cavity, is converted into a dirty-yellow calcified mass, that the rest of it and all the other laryngeal cartilages are in a perfectly normal state. And the inference which is suggested by such observations is confirmed by a case at Guy's Hospital in 1859, in which the right ala of the thyroid and the right half of the cricoid were alike necrosed, whereas the left halves of their cartilages had escaped. Moreover, ossification of the affected

cartilages is not in reality always present, even in adult life. In a remarkable case of a child, eighteen months old, who died with "croupy" symptoms, I once found that part of the left half of the cricoid, which was bathed in pus, had undergone absorption, so that there was a gap in it, with thin, smooth edges of perfectly natural appearance. And a year previously, in 1874, I examined the body of a man, aged thirty-three, in whom the back part of the cricoid was necrosed, lying loose in an abscess cavity, while its anterior part was represented by a narrow edge of healthy cartilage, thinning off into fibrous tissue. Lastly, there is every reason to believe that disease of laryngeal cartilages is often not merely dependent upon an affection of the perichondrium, but secondary to ulceration which began in the mucous membrane. Probably this is the correct explanation of many of the cases in which such disease arises in the course of phthisis, enteric fever, smallpox, or syphilis. And Dittrich suggested, in the paper already referred to, that in certain cases, occurring in persons confined to bed, necrosis of the cricoid is an indirect result of the pressure of this body, especially when ossified, against the vertebral column. His idea was the pressure first caused ulceration and sloughing in the two opposed surfaces of the pharynx, and that then the affection of the anterior pharyngeal wall spread to the perichondrium. The morbid process would thus be strictly comparable with that which is concerned in the formation of ordinary bed sores; and Dittrich gave one case in which, the patient being a phthisical man, aged thirty-one, numerous bed sores were actually present at the time of death. He also recorded in detail two out of several cases in which, in bedridden patients, he had found that both surfaces of the pharynx showed local patches of ulceration, without the cricoid cartilage having as yet become involved in the disease. Störk satisfied himself that in severe enteric fever necrosis of the thyroid cartilage may arise in a similar way, from pressure against the spinal column. And Ziemssen says that in old people, in whom the cricoid is ossified, that cartilage may be affected with perichondritis as a consequence of the repeated introduction of œsophageal bougies. Sometimes such disease is produced by direct injury, as in a case, recorded by Störk, of a man who was struck in the right side of the neck by a piece of wood, which flew up while he was attending to a circular saw. In some cases, perhaps, it is the result of exposure to cold. In others no cause can be discovered. Laryngeal perichondritis is much more common in males than in females. An analysis of twenty cases, collected from the pathological records of the Berlin School, showed that the period of life at which it was most apt to occur was between twenty and thirty years of age. But probably this was dependent upon the circumstance that eighteen of the twenty patients were either tuberculous or died of enteric fever. For I find that at Guy's Hospital the disease, as a primary affection, has been more frequent in persons from thirty to fifty than in those who were younger. One case occurred in a girl under two years old, one in a boy of nine, and one in an old man of sixty-three.

Hitherto I have spoken of perichondritis as though its necessary result were to produce suppuration and destructive changes in the subjacent cartilage. But there is every reason to believe that this is not always the case. In alluding to the subject of anchylosis of the crico-arytenoid joints at p. 796, I have already remarked that such an affection may probably sometimes lead simply to a development of fibrous tissue. Ziemssen records the case of a young man, in whom, in the course of enteric fever, a dark-red, flat projection appeared over one processus vocalis, causing hoarseness and severe pain. During convalescence this gradually diminished, and under favorable circumstances it might possibly have entirely subsided; but he

insisted on going out, and after three days' exposure to weather and indulgence in alcohol, returned in a state of such severe distress that tracheotomy had to be performed.

When necrosis does occur, the cartilage sometimes remains *in situ*, sometimes it is extruded from the abscess cavity. An arytenoid is often expectorated entire; the larger cartilages commonly break up into fragments, which come away one by one. Störk speaks of having seen cases in which suppuration went on for years. If, however, the necrosed material is completely got rid of, the cavity may become closed up by fibrous tissue.

The *symptoms* of laryngeal perichondritis vary a good deal with the exact seat of the affection. At first, however, there is little to distinguish them from those of other subacute or chronic diseases of the larynx. The patient usually complains of hoarseness of voice or of aphonia; there may be dysphagia, cough, more or less definitely localized pain and tenderness; presently dyspnœa sets in, which may rapidly increase until it threatens suffocation. Sometimes the spontaneous evacuation of the contents of an abscess cavity affords great relief to this symptom. In some cases of perichondritis enlargement of the cervical glands is a marked feature. I have notes of one case at Guy's in which they were found at the autopsy to be of the size of plums. The putrid discharge which is formed in some cases is probably a direct cause of danger to the patient's life, by dropping into the air passages and setting up a pneumonia that may rapidly pass on into gangrene. I have notes of two cases which ended fatally in this way.

Perichondritis of the *thyroid* sometimes shows itself on the outer, sometimes on the inner surface of the cartilage. In the former case there is swelling, œdema, and at length fluctuation over one of the alæ or over the pomum Adami; the affected part is very tender when pressed upon. In the latter case a swelling usually appears in the position of one sinus pyriformis within the ary-epiglottidean fold on one side, or even below the vocal cord, as in an instance recorded by Störk, in which it was mistaken for a polypus. Not infrequently both surfaces are affected in succession, so that when the abscesses have discharged themselves, milk or any other colored fluid can be injected through a sinus in the neck into the interior of the larynx, or a probe passed from without inward until it is visible in the laryngeal mirror.

Perichondritis of the *cricoid* usually affects its posterior rather than the anterior wall. It causes marked dysphagia. Another effect to which it sometimes gives rise is paralysis of the crico-arytænoidei postici muscles, so that the cords appear fixed near the median line. In a case that occurred at Guy's Hospital in 1861 it is noted that the voice remained clear, although there was extreme dyspnœa. Sometimes the symptoms develop themselves with extreme rapidity. Ziemssen cites a case of Pitha's which ended fatally in a week from its commencement. Where suppuration occurs, the abscess may discharge itself either into the pharynx, into the larynx, or into both canals at once. In some cases a swelling can be seen in the laryngeal mirror, bulging below one of the vocal cords; such a swelling has been mistaken for a solid new growth.

Perichondritis of an *arytenoid* cartilage leads to swelling and œdema of the surrounding soft parts, which may, of course, be visible in the laryngeal mirror. The mobility of the corresponding vocal cord is more or less interfered with, and the voice may be much impaired. I must confess, however, that my experience in the post-mortem room has impressed me with the conviction that neither aphonia nor even any marked alteration of the voice is nearly so constant a symptom of disease of an arytenoid

cartilage as seems to be generally supposed. I have seen cases of phthisis, in which complete exfoliation had occurred, and in which no laryngeal symptoms had been present during life, so far as I could learn. In such cases there is a good deal of indurated fibrous tissue in the place of the cartilage, which seems to have fixed the cord and enabled the muscles to act upon it sufficiently to maintain its functions. Laryngoscopically, when an arytenoid has been exfoliated, there is often an obvious falling in of the soft structures around.

It is, of course, to be understood that more than one of the laryngeal cartilages are not infrequently affected at once; one or both of the arytenoids, for example, together with a part or with the whole of the cricoid.

The *treatment* of perichondritis, if the disease is detected early enough, may sometimes be begun with leeches, the application of an ice bag to the throat, and other antiphlogistic measures. Whenever an abscess is recognized, whether outside or inside the larynx, it ought at once to be incised. Störk relates a capital case in which, having punctured a swelling below one of the cords and let out a quantity of pus, he subsequently brought the cavity to close by the systematic application of nitrate of silver to its interior. In almost all cases, however, tracheotomy is required sooner or later, and when dyspnœa has once set in there is great risk in delaying it. The immediate result is almost always successful, but it rarely happens that the swelling of the laryngeal structures afterward subsides sufficiently to allow of the removal of the cannula. Schrötter has recently had much success in the treatment of such cases by mechanical dilatation, at first with vulcanite tubes, and afterward with pewter plugs, about an inch and a quarter in length, which can be left in the larynx for several hours at a time. Having been introduced through the mouth, the plug is held *in situ* by being bolted into the convex surface of the cannula which the patient is wearing.

Laryngeal Tumors.—New growths within the larynx are by no means of very rare occurrence, and may be of very different kinds. From a clinical point of view it will be convenient to describe first those which are benign, and afterward those which are malignant in character.

With regard to the *causes* of benign growths in the larynx, almost the only fact hitherto ascertained is that they seem often to arise out of the irritation connected with chronic catarrh of the laryngeal mucous membrane. They are most frequently seen in persons who use the voice a great deal; this might be a sufficient explanation of the fact that they are far more common in males than in females, were it not that, according to Causit, a similar preponderance of boys over girls is observed among children affected with them.

1. *Papilloma.*—This, which is sometimes more accurately designated as *fibroma papillare*, is the commonest of all laryngeal tumors. It consists of a series of pointed or bulbous papillary excrescences, sometimes of small size, sometimes forming a large mass like a cauliflower, which may almost fill the cavity of the larynx. Their most frequent starting point is from one or both of the cords, especially near their anterior extremities, or from the angle between the cords. But sometimes they arise from the false cords, or even from the epiglottis, seldom or never from the mucous membrane covering the arytenoid cartilages, or the parts adjacent to them. Their color may be either whitish, or pink, or red. They cause more or less alteration of voice, or even complete aphonia; cough, which may torment the patient greatly, and which may be of a "croupy" character; dyspnœa, which sometimes ends in actual suffocation. It now and then happens that

fragments of papillary growths become detached in the act of coughing and are expectorated. Otherwise it is only with the aid of the laryngeal mirror that their presence can be accurately diagnosed. They not infrequently occur in quite young children. When removed by operation they are very apt to return, sometimes within a few months. Störk relates a case which came again and again under his observation during a period of thirteen years.

2. *Fibroma*, or *Fibrous Polypus of the Larynx.*—This forms a round or pear-shaped swelling, generally pedunculated but sometimes sessile, smooth or more or less lobulated, hard or more rarely soft in consistence, whitish or bright red in color, varying in size up to that of a hazel nut, or even larger still. It is a solitary growth, its development is exceedingly slow, and it never recurs when it has once been removed by operation. Its most frequent starting point is from one of the vocal cords, but sometimes it is attached to some other part of the larynx. Ziemssen figures one, as large as a walnut, which arose from the mucous membrane covering the posterior surface of the cricoid cartilage. Growths of this kind most frequently occur in adult or middle-aged patients. Störk speaks of them as sometimes becoming ulcerated on the surface, so that they bleed. In some few cases a fibrous polypus has become detached spontaneously and has been expectorated. With the laryngoscope the existence and the seat of this sort of tumor are generally easily recognized. Almost the only thing that can cause a mistake in diagnosis is the occurrence of eversion of the "sacculus laryngis." Such a specimen, taken from the body of a man who had had no laryngeal symptoms, was shown to the Pathological Society, in 1868, by Dr. Moxon, and is now in the museum of Guy's Hospital; it appeared like a semi-elliptical tumor hanging down in front of one of the cords, and could easily be replaced. More recently Dr. Lefferts, of New York, had diagnosed this affection in the living subject.

The symptoms produced by a fibroma of the larynx vary with its seat. Unless it is at a distance from the glottis the voice is almost always more or less affected, one reason for this being that even if the growth does not actually interfere with the opposition of the cords the surrounding mucous membrane is sure to be affected to a greater or less extent with catarrh. When a polypus has a pedicle of some length it may rise between the cords during phonation, and rest upon their upper surface, whereas during inspiration it falls down between them. The occurrence of dyspnœa is very uncertain. Dr. Mackenzie had a patient who invariably slept with her hand resting under the neck, and who would immediately wake up with distress of breathing whenever by chance her hand slipped away. In a case recorded by Lieutaud about a century ago, the patient died of sudden suffocation, as the result of stooping out of bed to pick up a book which had fallen on to the floor. This man had been conscious for some time of the presence in the larynx of something which he could not get rid of by coughing.

3. *Mucous Cyst.*—This is sometimes found upon the epiglottis, as in a case which occurred, in 1863, to Mr. Durham, who has recorded it in vol. xlvii of the "*Med.-Chir. Trans.*" The patient was a boy, aged eleven, who had suffered for some months from dysphagia, from hoarseness and feebleness of voice, and from attacks of dyspnœa which came on especially during sleep. The cyst, which was situated upon the laryngeal surface of the epiglottis, was incised, and gave exit to a glairy, thick, muco-purulent matter; it is therefore evident that the cyst was inflamed, and, indeed, the epiglottidean folds themselves were swollen and œdematous. In other cases a similar cyst has been found in the ventricle of Morgagni. Dr. Edis has

recorded an instance in which there was a cyst of the size of a hazel nut in the larynx of an infant, who died of suffocation thirty-seven hours after birth.

4. In some rare cases a laryngeal tumor has been a myxoma, a lipoma, an angioma (Mackenzie), or an outgrowth of the thyroid body penetrating the crico-thyroid membrane.

The only *treatment* for benign tumors of the larynx is their removal by surgical operation. Dr. Mackenzie, however, advises that small growths on the epiglottis or on the false cords should be left alone if they give rise to no inconvenience; he has observed several cases in which small "warts," after reaching a certain size, have ceased to undergo further growth. Various instruments have been devised for the purpose of removing laryngeal tumors through the natural passages or (as it is termed) by the "endo-laryngeal method"—knives (guarded or unguarded), cutting forceps, crushing forceps, guillotines, écraseurs, the galvano-cautery, have all found their advocates. I do not think it advisable to enter into details with regard to them, because it is not likely that any medical man would attempt to use them without having had special training, nor without consulting the works of those who have devoted themselves to the study of laryngeal affections. In choosing an instrument for a particular case, the degree of hardness of the growth and the character of its pedicle form important considerations; they must be determined, as far as possible, by the use of a laryngeal probe. It is not advisable to use an anæsthetic unless tracheotomy has previously been performed, but a few whiffs of chloroform may sometimes be given with advantage. A point which must be remembered is that, *cæteris paribus*, more skill is required in the removal of a very small laryngeal growth than of one which is larger. Both in this country and abroad an extraordinary degree of skill has now been attained in the performance of endo-laryngeal operations. The immediate result of the introduction of laryngeal forceps, or of any other instrument, is the production of a violent spasm, with a feeling as of impending suffocation, but this quickly passes off. When there are a large number of papillomata in the larynx, repeated endo-laryngeal operations are, of course, necessary, which may run over a period of several weeks.

In cases in which it is difficult or impossible to operate through the natural passages, the question arises whether recourse should be had to "thyrotomy," or the division of the thyroid cartilage in the median line, with separation of its halves, enabling the surgeon to seize the growth or growths and to clear out the whole cavity of the larynx on a single occasion. This procedure, which had been adopted for the removal of foreign bodies nearly a century ago, was vigorously advocated by Mr. Durham in a paper read before the Royal Medical and Chirurgical Society in 1871. But subsequent experience seems to have greatly limited the range of cases within which alone its performance can be justified. At the International Congress in 1881 opinions were almost unanimous with regard to this question. It was urged that the operation is attended with considerable danger to life from hemorrhage, or from other consecutive evils, among which pneumonia and necrosis of cartilages with suppuration perhaps take the principal places. Further it was shown that a permanent impairment or loss of voice is a not infrequent result of thyrotomy, though, on the other hand, there are many recorded cases in which the voice has been perfectly restored. Lastly, it was pointed out that in some patients there is great difficulty in getting the alæ of the thyroid cartilage wide enough apart to enable the operation to be successfully completed, and that experience does not at all confirm the expectation that the risk of recurrence of multiple papillomata is diminished by

the adoption of this procedure as contrasted with endo-laryngeal methods. Most of those who attended the Congress thought that even in young children (in whom multiple papillomata are so common) thyrotomy is really very seldom necessary. Krishaber related the case of a child, aged six, in which he succeeded in rapidly removing a number of tumors without a laryngoscope, by sliding a pair of forceps along his index finger into the larynx. One criticism, I think, it is fair to make upon the speeches delivered at the Congress; it is that laryngologists, not being so much accustomed to ordinary cutting operations, have probably in the performance of thyrotomy met with greater difficulties and obtained less satisfactory results than those which might occur to surgeons in the like cases.

A mucous cyst in the larynx requires only to be incised and to have its interior rubbed with caustic. Contrary to what might have been expected, it seems seldom or never to fill again.

Malignant growths in the larynx are sometimes *Sarcomata*, generally of the spindle-cell kind. Ziemssen speaks of there being more than twenty recorded instances of such an affection; its seat is usually on or near one of the vocal cords. Dr. Mackenzie figures a sarcoma which he describes as growing from the posterior surface of the cricoid cartilage; it had a papillomatous character.

Carcinomata of the larynx usually belong to the keratoid variety, such as are commonly called epitheliomata. I think they must be rare in comparison with cancers of other parts, for in the post-mortem room of Guy's Hospital I believe that only some four examples have been met with since the year 1854. All of them occurred in patients between the ages of fifty-eight and sixty-five. According to Ziemssen, however, they are fairly common relatively to other laryngeal growths; he speaks of having collected 147 cases, of which thirteen had come under his own observation. Among the patients there were many more men than women. In one curious case the development of the disease was preceded, at an interval of some months, by a fracture of the thyroid cartilage, the result of an attempt at strangulation.

The larynx sometimes becomes affected with cancer by extension from the pharynx or from the base of the tongue. But in the cases now under consideration the starting point of the affection is in the laryngeal mucous membrane, its original seat being generally one of the cords, one of the ventricles of Morgagni, or one of the false cords. In a case that occurred at Guy's Hospital in 1875 the amount of the growth at the time of the patient's death was remarkably small; the left ary-epiglottidean fold showed a whitish thickening, with puckering, as of a healed ulcer, two or three lines in diameter; the thickening extended down to the false cord on that side; until the microscope revealed the structure of a carcinoma, it was doubtful whether a new growth was present. In another case, observed in 1862, there was a raised patch, somewhat papillary in character, growing from the left cord and the parts around. But in many instances, as the disease spreads, extensive ulceration occurs. The structures outside the larynx become infiltrated with the growth, which may protrude into the pharynx, or from an obvious tumor in the neck. The ulcerated surface within the larynx may pour out an abundant ichorous discharge mixed with blood, or may even be the seat of copious hemorrhage. In such cases the breath becomes horribly fetid. Perichondritis, leading to suppuration, and to necrosis of cartilages, often occurs as a complication. Death may be due to œdematous laryngitis, or (as in two out of four cases at Guy's Hospital) to pleuro-pneumonia and empyema.

The laryngoscopic diagnosis of carcinoma of the larynx is by no means

always easy. At an early stage, when there is little beyond a diffuse infiltration of the mucous membrane, the case may be taken for one of perichondritis; and at a later period, when an ulcer has formed, an affection really syphilitic may be supposed to be cancerous, as in a very remarkable case recorded by Ziemssen, in which he fortunately gave iodide of potassium, with rapid and complete success, the patient being an old man of sixty-eight. There is, of course, nothing very characteristic in the other symptoms. Of these, hoarseness of voice, seldom amounting to complete aphonia, is the most constant and generally the earliest. According to Ziemssen, indeed, it is almost the rule that there is a "prodromal hoarseness," lasting a year or two; and in several of the cases which he collected this was prolonged during three, four, or five years, and once even during twenty-six years. It is certainly difficult to suppose that the affection had a definitely malignant character throughout such long periods; and, indeed, Ziemssen's statements with regard to the duration of laryngeal cancer appear scarcely consistent with what one knows of the rate of progress of a similar affection of other parts; he speaks of several cases which lasted three or four years, and of some which lasted even six, ten, or fifteen years. Next to hoarseness, *pain*, which may either be seated in some one spot within the larynx, or referred deeply to the pharynx, is the most conspicuous symptom. And Ziemssen lays stress on the frequent radiation of pain into one or other ear; this pain in the ear he associates with the auricular branch of the vagus; he found it present in five out of thirteen cases in which inquiries were made about it; and, sometimes, when there was no pain in the larynx itself. As a rule, dyspnœa occurs sooner or later; it may be especially marked when the patient is lying down. There may also be dysphagia. In all cases of suspected carcinoma of the larynx, careful search must, of course, be made for enlarged cervical glands; but Ziemssen says that they can seldom be detected within the first six months, and often not for a year or even longer. He also says that cancerous affection of the viscera is of very rare occurrence. However, in a case which I saw in 1879 with Mr. Durham, and which was yet in an early stage, there were already two flat subcutaneous nodules, one near the right clavicle, the other over the edge of the left sterno-mastoid muscle.

The *treatment* of carcinoma of the larynx can often be only palliative; but Ziemssen's case, already referred to, shows that whenever there can be a doubt as to the nature of the disease, the patient should have the benefit of the chance afforded by a course of iodide of potassium. Indeed, at the London International Congress, in 1881, Dr. Semon spoke of having several times seen this salt produce, in cases of cancer, a subjective improvement, and even, apparently, a brief temporary arrest of the progress of the disease. Tracheotomy is generally required sooner or later; and the average duration of life after the performance of this operation is said to be more than a year.

It is chiefly in cases of malignant tumor that the question has to be considered of the "total extirpation" of the larynx. This operation, originally performed for syphilitic stenosis, by Dr. P. H. Watson, of Edinburgh, in 1866, was first introduced to the profession in 1873 by Billroth, who in that year carried it out in a case of cancer. In a paper read in 1881 by Dr. Foulis, of Glasgow, before the International Congress, reports of thirty-two cases are collected, in twenty-five of which the disease was a carcinoma. In fourteen out of the twenty-five death occurred within sixteen days after the operation; and in not one of the remainder was life known to have been prolonged more than nine months, the only patients who were stated to be alive when the paper was read being two who had been operated on three months previously. It is, I think, difficult not to agree with Dr. Semon,

who took part in the discussion which followed the reading of Dr. Foulis's paper, and who evidently was of opinion that the operation had not yet been shown to be justifiable. However, Bottini, of Turin, has had one very successful case in which the larynx was extirpated for sarcoma; in 1881, six years after the operation, the patient was well, and had been able to work in the fields and to act as a postman. This evidently suggests the possibility of a like success in cases of cancer, if they could be operated upon at a sufficiently early stage. But, on the other hand, it is important to note that the condition of those patients who have survived for any length of time has generally been very miserable, there being great difficulty in deglutition, in consequence of the large opening in the neck, which could not be closed. The voice, however, can be restored by the use of an artificial vocal apparatus, such as was originally invented by Störk.

Foreign Bodies in the Larynx.—Not an uncommon cause of severe laryngeal symptoms is the entrance into the upper air passages of foreign bodies of various kinds. As a rule, such bodies are sucked down within the larynx during a deep inspiration, as the result of coughing, laughing, sneezing, or talking while there is something in the mouth. In children the accident sometimes occurs during the night, in consequence of the foolish habit of sucking a toy before going off to sleep; and it may even happen to an adult who wears false teeth, unless he is always careful to take them out at bedtime. It is surprising what large things will sometimes enter the larynx. Dr. Mackenzie relates the case of a boy, aged six, who went to sleep with a toy engine in his mouth; during the night it was drawn into the air passages, and tracheotomy had to be performed. The cause of the sudden attack of dyspnœa which had occurred was not discovered at the time, but some months later it was found that the toy was impacted in the subglottic region, whence it had to be removed by thyrotomy.

The symptoms produced by the entrance of a foreign body into the larynx are generally at first very violent; there is a most distressing sense of suffocation, the face becomes cyanotic, the inspiration is prolonged and whistling, a cold sweat breaks out, the patient tears at his throat with the hands, under an irresistible impulse to try to relieve himself of the cause of his sufferings. Such a case may end fatally, within a minute or two, by asphyxia. But it is an important point that if the air passages are completely closed, there is sometimes no obvious trouble with the breathing; the patient falls dead at once, and it may be only at the autopsy that the real cause is discovered of what had seemed to be an attack of syncope. In the act of vomiting, for example, it may happen that there is inhaled into the larynx a soft, pulpy mass which entirely fills it.

In many cases a foreign body, having passed into the larynx, at once falls through into the trachea; the early indications of laryngeal irritation then, of course, soon subside, and are followed by a fresh set of symptoms, which will be discussed in the next chapter. In other cases, again, the foreign body is quickly coughed out into the mouth, after which it, perhaps, is swallowed, and ultimately passed *per anum*. It may then happen that laryngeal symptoms—brassy cough, more or less dyspnœa, alteration of voice—which were present for some little time, entirely disappear; and it is then difficult to decide whether there is still something in the air passages or not. Another class of cases in which a diagnosis is not always easy occurs in hysterical women; such persons seek advice for tickling or pricking sensations in the throat, which they declare to be due to the presence of a needle or a pin, or a bristle, but which are really "paræsthesiæ," of neurotic origin.

Finally, when a foreign body is of large size, or when it has pointed ends, it generally becomes fixed in the larynx, and may remain there, as in a case already alluded to, for a great length of time. At any period œdematous laryngitis may set in, attended with severe dyspnœa. But if the foreign body is impacted in the ventricle of Morgagni, there may be merely pain and cough, with perhaps some degree of hoarseness, so that both the patient and his friends are apt to think it impossible that any such cause for his symptoms can still be present. In such cases the diagnosis rests entirely upon the results of careful laryngoscopic examinations.

When there is a foreign body in the larynx, it has, of course, to be removed in one way or another, and generally by surgical interference. In a large proportion of cases tracheotomy is required as a preliminary measure.

OBSTRUCTION OF THE TRACHEA AND BRONCHI. PLASTIC BRONCHITIS.

Obstruction.—EXTERNAL COMPRESSION—THYROID, ANEURISM, ETC.—INTRINSIC STENOSIS FROM SYPHILIS, ETC.—OBSTRUCTION BY A FOREIGN BODY—SYMPTOMS, DIAGNOSIS AND TREATMENT.

Plastic Bronchitis.—NAME—RARITY—SYMPTOMS—PATHOLOGY AND TREATMENT.

The trachea and the main bronchi are liable to but few affections except such as they have in common either with the larynx alone or with the tubes within the lungs below. And of such affections the clinical importance attaches itself always to the narrower rather than to the wider parts of the air passages. Hence there is no need for me to give a separate account of tracheitis; the plastic form of it has been described under croup; the catarrhal form will be dealt with under bronchitis.

But there are a variety of diseases which at some point may narrow the calibre of the trachea or of the bronchi, with the result of producing a definite and characteristic group of symptoms. Of these diseases, some have their seat outside the walls of the air passages, and will be described in other parts of this work. Here I need only briefly enumerate them. But there are others which originally affect the walls of the air passages themselves; or which, as in the case of foreign bodies, obstruct their channel from within. Of these I must give a full account in the present chapter. Since, however, it is often an accident whether in a given case the part narrowed is the lower end of the trachea, or one or both of the bronchi, it is useless to attempt to separate the affections of these several parts from one another, and the more so inasmuch as two, or even all three of them, are often involved at the same time. All that is possible is to describe the special symptoms which in certain cases indicate that the obstruction is altogether limited to one of the bronchi, leaving the trachea free. The general designation, *obstruction of the trachea and bronchi*, will serve to contain the whole group.

(A) Of the diseases which, starting from outside, may obstruct the air passages and so cause what may be termed a *compression-stenosis*, the following are the most important:

1. *Tumor of the Thyroid.*—It is well known that a goitre may compress the trachea in the neck, flattening it usually from side to side, so that its outline comes to resemble that of a scabbard, but also often pushing it out of the straight line or bending it. And it is by no means the largest goitres which are most apt to have this effect; much depends upon the exact situation of the growth, and upon the condition of the overlying muscles, which ordinarily oppose resistance to its extension outward, but which were in one case found by Virchow to be in a state of complete fatty degeneration. Another point of great importance, for a knowledge of which we are also mainly indebted to this writer, is that the middle lobe of the thyroid when it becomes enlarged, sometimes passes down behind the sternum so as to compress the trachea backward against the spine. He

even maintains that such a "substernal goitre" may be present without there being any obvious swelling of the thyroid in the neck. Upon this point, however, Ross, of Zürich, has recently expressed doubts. This surgeon, in a very able paper in vol. xxii of the "*Arch. f. Klin. Chirurgie*" has drawn attention to a peculiar change in the tracheal cartilages which occurs as the result of the presence of a goitre, rendering them soft and flaccid. The way in which he recognizes this after death is by dissecting off all other structures from the larynx and trachea and then placing them upright; the tube collapses at some one point, bending sharply so that its channel becomes completely closed. A like collapse is believed by him to be the cause of the supervention of sudden fatal dyspnœa as the result of goitre; he supposes that patients instinctively have to maintain the head in such a position as to avoid this occurrence, but that the muscles become relaxed during fainting, or sleep, or chloroform narcosis, or as the result of weakness. Dr. Bristowe, for example, relates, in vol. iii of the "*St. Thomas's Hospital Reports*," the case of a woman who was admitted for feverish symptoms, but who was one day suddenly attacked with intense difficulty of breathing, followed in a minute or two by blackness of face and insensibility. Fortunately he was close at hand, and finding that she had a tumor in the front of the neck, part of which was evidently cystic, he had this punctured, with the result that two or three ounces of a reddish-brown fluid were removed, and that she was quickly restored to health. In other cases enlargement of the thyroid is due not to a mere overgrowth of its tissues, but to the presence of a hydatid or to the development in it of a malignant new growth, which may then perforate the trachea and protrude into its channel; of this a well-marked instance occurred at Guy's Hospital in 1873.

2. *Thoracic Aneurism.*—I have before me notes, taken without selection, from the post-mortem records of Guy's Hospital, of twenty-seven cases of aneurism, in each of which there was interference with the trachea or with one of the main bronchi. In fourteen of them the sac arose from the arch and pressed straight backward upon the lower end of the trachea itself, flattening it, and often adhering very intimately with its walls. Probably in several of these cases the pressure extended also to one or both of the bronchi. But what I must confess is a surprise to me is that in no fewer than seven cases the aneurism seems (from the description given in the books) to have pressed solely upon the left bronchus; in three of these the sac arose from the summit of the arch on its left side, and pressed mainly upon the upper or upon the anterior surface of the tube; in the other four it came from the descending part of the arch and pressed forward upon the posterior surface of the air tube. On the other hand, I find only two cases in which the sac, having its origin in the right side of the arch, compressed only the right bronchus. The remaining four cases were examples of what is commonly termed aneurism of the innominate artery; in them the sac pressed upon that part of the trachea which lies behind the upper part of the sternum or in the root of the neck. It is difficult to say how many of the twenty-seven cases were characterized by other symptoms which actually did indicate or might have indicated the real nature of the disease during life. But I may note that in three instances the sac was of very moderate size. One, which flattened the trachea, was a round pouch, "of the size of a walnut" (as seen at the autopsy), arising by a definite orifice from the posterior walls of an aorta severely affected with arteritisde formans. Another, which compressed and opened into the left bronchus, was "no bigger than a marble." The third, which likewise interfered with the left bronchus, was "of the size of a small plum."

3. *Mediastinal New Growth.*—In the period during which the twenty-seven

cases of aneurism were observed in the post-mortem room at Guy's Hospital, there occurred nearly an equal number of cases in which the great air passages were narrowed by mediastinal new growths; and among twenty-four of them in which details are given as to the exact seat of the lesion there appear to have been eight in which the obstruction affected the lower end of the trachea or both bronchi (sometimes with a great preponderance on one side rather than on the other), six in which it was limited to the right bronchus, ten in which it was limited to the left bronchus. In every instance the new growth invaded the walls of the air passages, thickening them, and not merely pressing upon them from without. Indeed, there are two other cases besides those already mentioned, in each of which it is expressly reported that although the bronchus on one side was penetrated by the tumor there was no narrowing of its calibre. Among the whole number of cases there seems to have been hardly one in which if marked symptoms of stenosis were present there were not also observed other symptoms and physical signs sufficient to show that the obstruction was due to disease beginning outside the air passages. The pathological reports seem to justify the inference that mediastinal growths seldom invade the trachea or the bronchi at an early period in their development. It must, however, be remembered that they are not likely to be seen in the post-mortem room at this stage, since, unlike aneurisms, they do not commonly destroy life suddenly and unexpectedly by hemorrhage. In an interesting case of lymphosarcoma of the mediastinal glands, recorded by Weil in the "*Deutsch Archiv*" for 1874, all the symptoms and signs of tracheal obstruction disappeared suddenly eight days before death; at the autopsy it was found that this was due to the giving way of the softened mass, which must have poured its substance into the air passages, although the sputa had shown no fresh appearance, even under the microscope.

4. *Scrofulous Disease of the Bronchial Glands.*—This is commonly given as one of the causes of obstruction of the trachea or of a bronchus, especially in children. Vogel, however, says that although there may be slight flattening or indentation, it does not go on to actual sclerosis. On the other hand, Widerhofer, in Gerhardt's "Handbuch," describes this occurrence and also cites instances in which after prolonged dyspnœa abscesses dependent on disease of the bronchial glands discharged into the air passages with relief to the urgent symptoms.

5. *Mediastinal Abscess.*—Abscesses of various origin may compress the trachea or a bronchus. One of the most striking cases that I have met with is recorded by Schnitzler, in the Wiener "*Kunik*" for 1877. The patient was four years old; an abscess of the size of a child's fist pushed forward and to the right; its starting point was caries of the second and third dorsal vertebræ.

6. *Carcinoma of the œsophagus* is mentioned by Riegel and other writers as an occasional cause of stenosis of the trachea. But although it frequently invades the air passages I cannot remember to have ever seen it produce in this direct manner symptoms indicating interference with the entrance of air. As I have already remarked at p. 794, it may cause a bilateral paralysis of the abductors of the glottis, and so render the performance of tracheotomy necessary. In all probability the emaciation which is so marked a symptom of œsophageal cancer is attended with a great diminution in the activity of the pulmonary functions.

7. *A dilated left auricle*, secondary to mitral stenosis, may compress the left bronchus, as was first pointed out by Mr. Wilkinson King, in 1838, and as may be still seen by his preparations in the museum of Guy's Hospital. Friedreich has recorded an instance in which pressure on the bronchus from this cause was actually diagnosed by physical signs four

years before the patient's death; at the autopsy, made by Virchow, it was found that only a very narrow channel was left. My own observations, however, would lead me to believe that cases in which this condition can be made out at the bedside are very rare.

(B) Of the diseases which, starting in the walls of the trachea or of the main bronchi, may narrow the calibre of the air passages, some are exceedingly rare. Demarquay, for example, is cited by Riegel as having observed a case in which such an affection arose from ulceration set up by the poison of glanders. And Langhans, in vol. liii of "*Virchow's Archiv*," recorded in 1871 an instance of primary carcinoma, having its origin in the mucous glands of the lower end of the trachea and right bronchus, which destroyed the life of the patient, a man of forty; it appeared as a warty affection of the lining membrane, extending also by infiltration into the muscular and fibrous external coats. Whether a simple local inflammatory process is capable of thickening the walls of the lower air passages, so as to obstruct their calibre, is, I should think, doubtful. Andral and Wilks are referred to by Riegel as having reported such cases; but the observations of Wilks, at any rate, were of a syphilitic stenosis only. The last-named affection is, indeed, by far the most important cause of obstruction of the lower air passages, if the diseases producing compression from without be excluded. Gerhardt, in vol. ii of the "*Deutsches Archiv*," alluded to twenty-two examples of it, of which he had made an analysis; and seven instances presented themselves in the post-mortem room of Guy's Hospital between 1861 and 1874. Sometimes the disease is limited to a single spot in the trachea, as in a specimen, taken from a patient of Dr. Bright's, which is contained in the museum of Guy's Hospital, and in which opposite the second ring there is a contraction, like that which might have been produced by a ligature. Much more often it extends for a considerable distance along the tube, and it may involve its whole length, and may even be prolonged into one or both of the bronchi. The bronchi themselves are but seldom affected when the trachea escapes. But Wilks, in the "*Guy's Hosp. Rep.*" for 1863, relates a case in which the right bronchus alone was stenosed; and in another case, observed at the hospital in 1875, the lesion was found to have attacked only the left bronchus and the upper branch of the right bronchus. The mucous membrane is commonly raised into a series of irregular bands and ridges, which, following Wilks, I have been accustomed to regard as the cicatrices of former ulcers. Gerhardt, indeed, has reported a case in which at the time when death occurred, from a form of chronic pneumonia, there was simply an unhealthy ulcer with raised edges, occupying the right bronchus and one of its branches for about an inch, and exposing the bronchial cartilages. But the view taken by German pathologists generally is that the fundamental lesion is a diffused thickening of the whole tracheal wall, raising its lining membrane into folds and prominences. They describe ulceration, more or less extensive, as of not infrequent occurrence, but they regard this as secondary. It may spread deeply, setting up a perichondritis, and so leading to ossification and necrosis of the tracheal or bronchial cartilages, which may even be exfoliated and discovered by the patient in his expectoration. Or it may penetrate to the tissues outside the air passages, forming an external abscess. In a case that occurred at Guy's Hospital in 1865 there was actually perforation of the aorta, which happened to be highly atheromatous, so that the patient died of sudden hemorrhage. In other instances, the tracheal rings, instead of being exposed and detached, become atrophied and bent on themselves or dragged one over the other. I do not know whether syphilitic stenosis affecting a bronchus ever leads to its complete obliteration. In certain instances in which such a condition has been found it has been regarded as congenital. Thus Ratjen, in vol. xxxviii

of "*Virchow's Archiv*," described a case occurring in a man, aged forty-nine, whose left bronchus was converted into a fibrous cord for an inch and a half of its length, the corresponding lung being quite airless, while the right lung was enormously enlarged and apparently in a state of true hypertrophy, its air cells being of normal size. But Cohnheim observes, with reference to this case, that the presence of pigment in good quantity in the collapsed left lung is clear proof that it had at one time been in a functionally active state.

With regard to the time of life at which syphilitic stenosis of the trachea proves fatal, it is, perhaps, worth noting that the large majority of cases at Guy's Hospital have been in persons between forty and fifty years old, and it has occurred in men far more often than in women. Among the cases collected by Gerhardt there was a far wider range of ages; one was in a patient under ten, and another in a patient under twenty; probably these are the two cases to which he alludes as having been apparently instances of inherited syphilis. Two instances of syphilitic stenosis in children twelve years old are given by Widerhofer.

(c) Obstruction of the lower air passages may be due to a *foreign body*. As I have already remarked at p. 842, a foreign body which enters the larynx through its upper orifice rarely remains fixed there, unless it is either very large or pointed in shape. Beans, peas, nut shells, pebbles, small coins, fragments of bone, commonly fall into the trachea. Sometimes they remain free for a time, moving up and down as the patient coughs. One may then be able to feel the impact of the foreign body against the side of the trachea with the fingers placed outside the patient's neck, as was observed by Mr. Lucas in the case of a little child with a pebble in its air passages whom I saw with him in 1880 ("*Clin. Soc. Trans.*," xv). Even in that instance there were physical signs which rendered it probable that the pebble lay in the right bronchus in the intervals between the fits of coughing. And, as a rule, such bodies soon become fixed in the right bronchus or in one of its main divisions; the reason why they enter it rather than the left bronchus being that the fork between the two is slightly to the left of the middle line, so that the opening into the right bronchus is rather the more direct. Sometimes, however, the left bronchus is the one into which a foreign body passes; and sometimes each bronchus in turn, the body becoming dislodged by cough and falling now into one, now into the other. In some cases the cause of obstruction is not, strictly speaking, a foreign body at all; it may be a tooth or a fragment of uvula, or of a pharyngeal polypus separated by the hand of the surgeon; it may even have found its way into the air passages by ulceration from the living tissues, as when it is a necrosed laryngeal cartilage, a concretion from a bronchial gland, or an echinococcus vesicle from the liver. An accident which has several times happened after tracheotomy is that a portion of the tube has become detached from the rest and has dropped into the trachea. Altogether the literature of foreign bodies in the air passages is very extensive, no fewer than 374 recorded cases having been collected and analyzed by Kühn.

As may be supposed, foreign bodies are especially apt to enter the air passages of children and of lunatics. Others besides such patients, however, when attacked by sudden and violent symptoms as the result of this accident, may be altogether ignorant of the cause. Hamberger is cited by Riegel, in Ziemssen's "Handbuch," as having recorded the case of an old man, aged seventy, who fainted after a journey, and was found in a state of dyspnœa, with evident obstruction of the right bronchus. An emetic was given, which led to the expectoration of a green pea swollen to the size of a bean. Subsequently it was learned that when he was eating peas, one day, he had swallowed one the wrong way.

Of the *symptoms* of stenosis of the lower air passages, the most important is dyspnœa; as contrasted with laryngeal stenosis it may be said in the main to be characterized by difficulty of breathing without loss of voice. One must, however, remember that the power of speaking well and even loudly is not in itself proof that the seat of an affection attended with severe distress of breathing is not in the larynx. For in bilateral paralysis of the abductors of the vocal cords, precisely this combination of symptoms is met with, as has already been shown at p. 796. On the other hand, it frequently happens that the voice in cases of tracheal stenosis is weak, thin, and devoid of sonorous quality, from deficiency in the stream of air reaching the larynx from below. A further point to be borne in mind is that syphilitic disease of the larynx is often combined with a like disease of the trachea; a patient may have lost his voice as the result of a syphilitic affection of the larynx, but the dyspnœa from which he suffers may nevertheless be dependent on mischief lower down, so that, if tracheotomy should be performed, the operation may turn out a failure, and afford no relief whatever. Again, in many cases of aneurism or of mediastinal growth, tracheal stenosis is accompanied by paralysis of laryngeal muscles, as the result of pressure upon one or both of the recurrent laryngeal nerves.

Whether or not the voice is affected, it is, therefore, essential to make a thorough laryngoscopic examination in all cases of suspected stenosis of the trachea or of the main bronchi. But, moreover, it is often possible, especially if the larynx is healthy, to make a direct diagnosis of the nature of a tracheal lesion by examination with the mirror. The lower part of the windpipe and its bifurcation, with the orifices of the two bronchi, are said to have been first seen in the person of Czermak himself, by Elfinger. Türck has pointed out the conditions most favorable to a successful exploration of these parts. The patient should be seated with the body and the neck upright and the head bent slightly forward, the object being to bring the axis of the larynx and that of the trachea into a straight line. The mirror must be placed against the soft palate, rather further forward than usual, and with its surface nearly horizontal. The observer should sit at a lower level than the patient. The illumination must be very bright and the light should be thrown into the mouth horizontally, or rather from below. An aneurism may sometimes be seen bulging into the trachea, as in a case of innominate aneurism which was examined by my friend, Mr. Lane, when house physician at Guy's Hospital. It must not, however, be supposed that a mere slight pulsation of the lower end of the trachea necessarily indicates a morbid condition, for Gerhardt and Schrötter have shown that such pulsation is present in many healthy persons as the result of the pressure of the great arteries coming from the base of the heart.

Local Diagnosis.—An important distinction between stenosis of the lower air passages and that of the larynx was first pointed out by Gerhardt. It is that in the former affection the larynx does not during inspiration make the rapid and extensive movement downward which occurs when the larynx is itself the seat of obstruction to the entrance of air. According to this observer, if with severe stenosis the range of descent of the larynx is not more than one centimetre, one may confidently assert that the disease is either in the trachea or possibly in both bronchi, but not in the larynx. He also remarks that the position of the patient's head differs in the two sets of cases. When the obstruction is laryngeal, the head is thrown backward as far as possible. When it is tracheal, the head is stretched forward, and the chin slightly depressed, so as to relax the trachea. The character of the dyspnœa in stenosis of the lower air passages is in the main inspiratory, like that in laryngeal stenosis. It is less often extreme in degree, on account of the greater calibre of the trachea as compared with that of the

glottis. Consequently, the breathing is not usually greatly reduced in frequency; nor are the lower ribs and the other unsupported parts of the chest walls very much sucked in. But should the disease go on to actual suffocation, all these phenomena may be as marked as they possibly can be in any case whatever. On the other hand, there is generally from an early period the loudest and most noisy stridor, or (as the French term it) *cornage*. It is heard not only through a stethoscope placed over the trachea or over the back of the neck, but also more or less on auscultation over every part of the chest, drowning the normal breath sounds. Indeed, it is commonly obvious to every one standing near the patient. According to Gerhardt, the only cases in which any safe conclusion as to the seat of the obstruction can be drawn from observations as to the spot at which this sound is heard loudest through the stethoscope, are those in which this spot is directly over the trachea in the neck. When there is stenosis of the lower part of the trachea, it often happens that the sound is audible with greater intensity over the larynx than over the sternum. Sometimes a râle is constantly discoverable over some particular point in the trachea. A sign to which Demme has drawn attention is that in prolonged cases of constriction of the lower air passages the circumference of the upper part of the thorax becomes lessened.

In most cases of stenosis of the trachea, the dyspnœa undergoes aggravation from time to time, there being paroxysms of the most extreme distress which are attended with cyanosis, and one of which generally at length proves fatal. It was formerly supposed that the cause of such attacks was paralysis, or perhaps spasm, of the vocal cords from implication of one or both of the recurrent laryngeal nerves, and I think that Dr. Bristowe, in an admirable paper in vol. iii of the St. Thomas's Hospital "*Reports*," was the first to point out the incorrectness of this opinion. They are probably due either to acute swelling of the mucous membrane at the seat of pressure, or to an accumulation of mucus there which cannot be disloged, or perhaps in part to spasm of the muscular tissue of the trachea itself. It is important to notice that no relief is to be expected from the performance of tracheotomy.

The patient commonly complains more or less of subjective sensations of oppression of the chest, of soreness behind the sternum, or of actual pain. There may or may not be cough, with expectoration of mucus, perhaps tinged with blood, according to the nature of the disease which produces the stenosis.

In contrasting the physical signs of obstruction of one bronchus with those of stenosis of the entire lower air passages, the first point to be remarked is that much depends upon whether the obstruction is complete or partial. In the former case there is absence of vesicular murmur over the corresponding side of the chest, with impaired movement of the ribs and of the diaphragm, deficient vocal fremitus, and a normal percussion sound. After a time the side may actually be found to have fallen in, and to measure less than the other side. In the latter case a snoring, whistling, or humming sound may be heard over the root of the lung between the scapula and the vertebræ, or there may be moist sounds there. A thrill may sometimes be felt with the hand placed upon the surface of the chest.

One clinical peculiarity of the obstruction of a main bronchus caused by a foreign body is that it is far more sudden, as well as more complete, than that due to any other cause. Consequently, its effects may be supposed to approximate more closely than those of any other morbid condition likely to be observed in man, with those of the plugging of a bronchus by wedges of laminaria, which were studied by Lichtheim in a series of experiments on rabbits recorded in vol. x of the "*Arch. f. exp. Pathologie*." The opposite

lung in these experiments became enormously distended. Very often it gave way, so that pneumothorax resulted; even when this did not occur, the animal usually died within twenty-four hours. What proved that the rapidly fatal issue was immediately dependent upon the state of this lung rather than of the one which was deprived of air, was that no such consequences followed when the pleura was laid open on the side of the obstructed bronchus. A bean or a pea is capable of swelling, like the laminaria plugs used by Lichtheim, although more slowly; and his results are worth bearing in mind, because it may be that in the failure of all attempts to extract a foreign body from a bronchus, to admit air into the pleura might sometimes be a justifiable procedure; even if it did not prolong life it might greatly relieve the dyspnœa.

Sequelæ.—Every form of disease producing obstruction of the lower air passages is liable to be attended with inflammatory changes in the pulmonary tissue, as well as in the walls of the air passages themselves. Thus when an aneurism has pressed upon the trachea, or upon a bronchus, I have repeatedly seen the mucous membrane ulcerated and some of the cartilages exposed and partially detached, even though there may have been no indication of an approaching rupture of the sac. Stenosis of a bronchus, from whatever cause, is not infrequently accompanied by dilatation of its branches within the lung. Purulent fluid is apt to accumulate in them, and the result is the occurrence of more or less extensive pneumonia, which often goes on to gangrene. A foreign body fixed in a bronchus often sets up ulceration and sloughing of the part of the tube against which it presses. Sometimes this ends in perforation of the pleura, with pneumothorax, and the foreign body itself may become loosened and fall into the serous cavity. Sometimes it leads to a pneumonia, which may spread from the root of the lung far into its substance. The occurrence of fetid expectoration, and the development of the appropriate physical signs, may reveal these various changes, but in some cases they are first detected in the post-mortem room, there having been no suspicion of them during life. Even after expulsion or removal of the foreign body, it sometimes happens that the case nevertheless ends fatally, as a consequence of the pneumonia that had been set up; but, happily, this sometimes subsides and the patient makes a permanent recovery.

With regard to the *diagnosis* from one another of the several affections that may cause obstruction of the lower air passages, I think it is worth remembering that the two diseases in the course of which stenosis of the trachea is most apt to occur without the presence of any other symptoms are syphilis and thoracic aneurism. When the obstruction is limited to a bronchus, aneurism is still probable, especially perhaps on the left side; a mediastinal growth is a more likely cause than a syphilitic stricture. The possible presence of a foreign body must never be disregarded, especially if the symptoms have come on suddenly.

The *duration* of syphilitic disease of the trachea after symptoms have set in ranges, according to Gerhardt, from two months to four years. That of a compression stenosis, from whatever cause, would probably be found to be confined within comparatively narrow limits of time. Foreign bodies sometimes remain for a very long period—for months, even for years—in the lower air passages, and yet are, after all, expectorated.

As to the *treatment* of the various affections that may cause stenosis of the trachea or of the bronchi there is little to be said. Whenever there is a possibility that it may be due to syphilis, mercury and iodide of potassium should be employed actively. Gerhardt relates, in vol. ii of the "*Deutsch. Archiv*," the case of a man, aged thirty-six, who had had constitutional symptoms after a hard chancre eight years before, and who consulted him

on account of cough with scanty, muco-purulent expectoration, a tickling sensation behind the sternum, a little alteration of voice, and slight interference with the breathing. These symptoms had been present for about six months. The patient had lost flesh to some extent; his face was somewhat puffy and livid. Nothing could be discovered with the laryngoscope, and only râles behind the manubrium with the stethoscope. A permanent cure was effected by the administration of full doses of iodide of potassium during several weeks. Unfortunately, however, the cases which are usually seen, and in which the affection has already led to the formation of cicatricial bands and ridges, appear not to be amenable to anti-syphilitic remedies.

When there is a foreign body in the air passages the only proper course is to perform tracheotomy at once. Until this has been done, it is not safe to place the patient head downward, on the chance that the body may fall out through the glottis, as happened (but after tracheotomy) in the case of Mr. Brunel, which is so graphically told in Watson's "Lectures." Nor does it appear to be prudent to administer an emetic, on account of the risk that the body, if dislodged from its position in a bronchus, may become impacted in the larynx and cause suffocation.

Fibrinous or Plastic Bronchitis.—In discussing the subject of inflammation of the bronchial tubes, I shall have formally to exclude from the disease, as I shall then describe it, an affection which anatomically is certainly nothing else than a bronchitis, but which yet has characters so peculiar that from a clinical point of view it would be absurd to group it with the ordinary catarrhal forms. It consists in the exudation of a fibrinous material from the walls of the air passages which forms "casts" of their channels. But here, again, a further limitation is needed. A like exudation may occur by extension of a morbid process downward from the larynx in diphtheria or in membranous croup, and also by extension upward from the pulmonary alveoli in pneumonia. It is not my intention to include cases of either kind in the description which follows.

It would almost have been better for the disease to have a name altogether unmeaning, since it is not likely that any possible designation invented could precisely convey the right conception. But for practical purposes the terms "plastic bronchitis" and "fibrinous bronchitis" do well enough, and are, I think, to be preferred to "croupous bronchitis," which suggests an association with laryngeal croup. Some of the earliest recorded cases went under the inappropriate name of "bronchial polypi." The disease is one of the rarest that are known to physicians.* At Guy's Hospital I am not aware that a single instance of it has been observed within the last quarter of a century. The experience of Dr. Watson is perhaps unique, who says that he has had under his observation five well-marked examples.

Anatomy.—In almost all cases of plastic bronchitis the patient soon begins to expectorate masses of the peculiar exudation. It usually appears rolled up into a sort of ball, with a good deal of mucus and blood covering it. All this is easily removed by floating it out in water, and one then sees that there is a complete cast of some part of the bronchial tree, extending, perhaps, down to its finest subdivisions, so that, according to Biermer, the minute terminal filaments may actually have bulbous ends moulded in the infundibula themselves. The color of the cast is whitish yellow or gray; its consistence is tough and elastic; it is almost always made up of a number of concentric laminæ, separated here and there by narrow spaces and with a more or less definite central cavity, containing mucus or bubbles of air. Only the more delicate fila-

* [" Höchstselten:" Riegel, "Aeusserstselten:" Biermer.—Ed.]

ments are said to be generally solid. This laminated structure affords a distinction from the branching clots which are sometimes formed in the air passages as the result of hemorrhage, and which are quite homogeneous. Biermer, indeed, is disposed to deny that blood ever coagulates so as to form casts of the bronchial tree. But Dr. Walshe speaks about it in the most positive terms, and there could hardly be a better authority. The casts in cases of fibrinous bronchitis, when examined microscopically, are seen to consist of a hyaline or slightly fibrillated base, in which are imbedded large numbers of leucocytes. They seldom contain red blood discs in any quantity. Several observers have noticed Charcot's crystals in them. In one case Waldenberg found that the thicker parts contained only a few formed elements, but very abundant fat globules.

The length of a bronchial cast is commonly from one and a half to two and a half inches, but sometimes it may be four or five inches, or even (as in a case of Riegel's) six or seven inches. The diameter of the thickest part of it is seldom greater than that of a goose quill, being, in fact, considerably less than that of the space in which it was formed. Biermer has pointed out that from its appearance one can sometimes draw an inference as to the part of the lung from which it came, whether from the short, rapidly-branching tubes of the upper lobe, or from the comparatively longer tubes of the lower lobe. The masses expectorated at different times by the same patient often resemble one another so exactly in size and in the arrangement of their subdivisions as clearly to show that they have all in succession been derived from the same tract of mucous membrane. For example, in a case recorded by Kretschy seven casts appeared one after the other, all of which came from the middle and lower lobes of the right lung. In fatal cases it is not usually found that the tubes which have poured out the fibrinous exudation show any marked morbid changes. The mucous membrane is sometimes reddened, sometimes pale and healthy looking. In two instances Biermer found the epithelium still remaining beneath loose casts; but he was himself disposed to think that these cases might be exceptional; and Kretschy has since stated that in his case there was no trace of epithelium in that part of the air passages which contained the plastic material. The submucous tissue may be swollen and infiltrated with serum. The pulmonary alveoli are healthy, except that some of them are sometimes in a state either of collapse, or of over-distention, or "acute emphysema."

Symptoms.—The expectoration of casts of the lower air passages is generally attended with severe cough and dyspnœa, the occurrence of which may be the first indication that the patient is otherwise than well. But in many cases there is an antecedent stage during which he appears to be suffering from ordinary bronchial catarrh; and this may last for a long time. Sometimes the disease sets in with rigors, loss of appetite, thirst, oppression of the chest, and pyrexia, so that it may be supposed that an attack of pneumonia is impending. Presently a dry, hard cough appears, which may cause extreme suffering; the breathing becomes rapid, up to 40 or more in the minute; it may be attended with the greatest anguish, as of impending suffocation, with lividity, and with a small, tense pulse. There may be some pain in the side, and a feeling of soreness within the chest, but, on the whole, the attack is more distressing than acutely painful. At first nothing is expectorated, or only a little mucus. The cough may even, it is said, go on for days before any fibrinous masses appear. More often a cast is detached and got rid of after a few hours, and by this the cough and dyspnœa are generally at once relieved, at least for a time. Hæmoptysis often occurs at intervals during the paroxysm, not only at the time when the cast is being expectorated, but also previously. It must, therefore, be borne in mind that the mere fact of there having been spitting of blood in any particular case in

which casts of the tubes are afterward ejected affords no ground for maintaining that they must necessarily be coagula, and cannot have been formed as the result of a process of exudation from the walls of the tubes. But I do not know that the quantity of blood is ever very large; perhaps it amounts to a tablespoonful at a time. The case of the late Prof. Daniell, recorded by Watson, is the only one which I remember to have read of in which it was from two to eight ounces on each occasion.

Physical Signs.—Examination of the chest throws but little light upon cases of plastic bronchitis. If a large tube is blocked, absence of vesicular murmur may be made out over some part of one of the lungs. The fact that the violent cough fails to clear away the obstacle might, perhaps, suggest to a keen observer the presence of something more than a plug of mucus such as may prevent the entrance of air in ordinary cases of catarrhal bronchitis; and the diagnosis as to the cause of the obstruction would then lie between fibrinous casts, a foreign body, and stenosis of the walls of the tube. In practice, however, it scarcely ever happens that any suspicion of the real nature of the case arises until a cast has actually been expectorated. There is not usually any change in the percussion sound, but Dr. Walshe says that he has had repeated occasion to observe dullness, as complete as that of pneumonic consolidation, dependent upon collapse of the lung substance. He also says that local pneumonia now and then occurs, attended with crepitation and with bronchial breathing, as well as with rusty sputa. When there is very extensive blocking of tubes, the movements of the corresponding side of the chest may be distinctly impeded, and the lower ribs may even be drawn in during inspiration. Râles are sometimes audible over the affected part of the lung, especially when the cast is becoming loose; some writers have described special sounds as arising in such circumstances, but it does not appear that they are really pathognomonic.

Course.—It very rarely happens that the expectoration of a single cast brings to an end an attack of plastic bronchitis. As a rule the relief is only temporary. After some hours the cough and the dyspnœa return and are followed by the appearance of another cast. This process is usually repeated about once in twenty-four or in forty-eight hours for several days, and then the affection slowly subsides and the patient gets well. Smaller pieces may be spat up at very frequent intervals; being imbedded in mucus they sometimes remain unnoticed, unless specially looked for.

Prognosis.—It may well be supposed that the expulsion of such large masses as sometimes come from the air passages in this disease is not altogether unattended with danger. In 1865 I showed to the Pathological Society a cast which was taken from the body of a girl, aged seven, having been found lying across the bifurcation of the trachea, with its branches extending into the ramification of the right bronchus, but with its broad end occluding the left bronchus. She had been expectorating similar masses for three days; and on the very day on which she died she had already, at three A.M., brought up a cast of about the same size as that which killed her at three P.M. in a violent fit of cough and dyspnœa. It is, therefore, clear that Dr. Walshe and others go too far in giving a favorable prognosis in cases of plastic bronchitis without any reservation. Lebert, in a paper in the "*Deutsch. Arch.*" for 1869, divides his cases (collected from various sources) into acute and chronic; of seventeen of the former four ended fatally; of twenty-seven of the latter only three. The distinction, however, appears to me to be artificial; and one of the four fatal acute cases, that recorded by Nonat, was, I suspect, really a case of diphtheria. As a rule, when death occurs, it appears to be the result of the extension of the morbid process into so large a part of the bronchial tree that due aeration of the blood can no longer be effected. In such instances it is preceded by stupor and som-

nolence. Riegel, however, relates a case in which, although the patient died in an attack of asphyxia, after having been spitting up large casts nearly every day for three weeks, the air passages were all found empty after death. Lebert has placed in a separate category cases in which plastic bronchitis has run on to a fatal termination without any fibrinous masses having been expectorated. As they occurred chiefly in children, and generally in association with broncho-pneumonia after measles, they should probably be regarded as different from plastic bronchitis.

Recurrence.—When an attack of fibrinous bronchitis has passed off, leaving the patient apparently well, it by no means follows that the disease is really at an end. One of the most curious points about it is its liability to return again and again at irregular intervals, sometimes during a very long period. Dr. Walshe met with an instance in which the expectoration of casts continued, with occasional brief intermissions, from the spring of 1843 to June, 1857, when he lost sight of the patient. In the course of this time she married, and she once resided at Buenos Ayres for four months. Other observers have recorded cases which were scarcely less protracted; in many of them the general health seemed to remain entirely unaffected.

Treatment seems generally to be altogether ineffectual. Waldenberg, however, saw a case in a girl, aged eight and a half, who for more than four years had been coughing up fibrinous masses at intervals of a few days, and in whom a whey-cure and the daily inhalation of lime-water succeeded in arresting the disease in from six to seven weeks. Indeed, a spray of lime-water, or of a solution of an alkaline carbonate, should always be employed in plastic bronchitis; the only doubt is whether they reach the lower air passages in sufficient quantity. Emetics appear to be sometimes useful; probably it is best to use apomorphia hypodermically. Biermer recommends an active mercurial treatment; others have prescribed iodide of potassium, with apparent advantage.

Ætiology.—With regard to the causes of plastic bronchitis, scarcely anything can as yet be said. It is more common in males than in females, the proportion being as three to two, if not as two to one. The period of life at which it is most frequent is between ten and thirty. One case has been recorded at the advanced age of seventy-two; it had lasted seven or eight years. One remarkable circumstance, all the more striking because of the extreme rarity of the disease, is its occurrence in different members of the same family. Fuller met with it in two sisters; Watson relates the cases of two brothers, both of whom were affected within a twelvemonth. It is supposed to be rarer in southern countries than in the north of Europe. Riegel says that, like acute pneumonia, it is most apt to occur toward the end of spring, when there are great daily variations of temperature. In one instance the recurrence of the attacks appeared to be connected with the catamenial periods. Eisenlohr met with a case in which fibrinous casts were expectorated during the second week of enteric fever.*

* [An able report on this remarkable affection by the late Dr. Peacock occurs in the 5th vol. of the "*Pathological Transactions*," where he tabulates 31 cases. In the 11th vol. Dr. Salter describes and figures another specimen. These "bronchial polypi" were known very early. Dr. Nicholas Tulp (the lecturer in Rembrandt's famous painting, "The Lesson of Anatomy") records and figures two specimens brought up by a Dutch sea captain suffering from hæmoptysis: "*Effudit duos insignes venarum ramos, adæquantes singulos expansæ manûs magnitudinem*" ("Obs. Med.," Amst. Elz., 1652, cap. xiii, p. 122, tab. iii, iv). Afterward cases were recorded by the younger Bartholin, Cheselden, de Haen, Morgagni, Hunter, Cheyne of Dublin, and Stokes. Of 55 cases (including Dr. Peacock's) 42 occurred in men and 13 in women (Biermer's figures are 39 male to 19 female cases). Of 37 patients of whom the age is given, I find five between 5 and 10, twelve between 11 and 20, ten between 21 and 30, eight between 31 and 50, and two between 50 and 60.—Ed.]

DISEASES OF THE LUNGS.

Symptoms common to various Chest Affections—DYSPNŒA—VARIETIES—PHRENIC DYSPNŒA—COUGH—BRONCHIAL—FAUCIAL—GASTRIC—CEREBRAL—PLEURODYNIA.

Percussion—HISTORY—METHODS—TERMINOLOGY—PHYSICAL THEORY—SIGNIFICANCE.

Auscultation—HISTORY—METHODS—THE RESPIRATORY MURMUR—BRONCHIAL AND TUBULAR BREATHING—PHYSICAL EXPLANATION—RALES—THE VOCAL RESONANCE IN HEALTH—BRONCHOPHONY—PECTORILOQUY—ÆGOPHONY—TACTILE VIBRATION—INSPECTION AND MEASUREMENT OF THE CHEST.

In passing on to consider the affections of the lower air passages and lungs, there are certain symptoms common to several of them which demand notice; and the methods of percussion and of auscultation have also to be discussed.

I. DYSPNŒA.—The use of this term is commonly limited to cases in which a sensation of "shortness of breath" is experienced, with more or less discomfort or distress. But I think that we ought rather to follow the physiologists, and to understand by it simply that the respiratory movements are deeper than natural, or more frequent, or both deeper and more frequent, without regard to whether the patient is or is not conscious of any disturbance of his breathing. Now, it is a remarkable fact that persons affected with extensive disease of the lungs, provided that such disease develops itself gradually, may continue to breathe as slowly as in health, and with no more effort, so long as they are at rest. The amount of oxygen supplied to the blood is no doubt considerably reduced under such circumstances; but they manage to make it suffice for the wants of the system. It might be imagined that a deficiency of oxygen would probably interfere with the due completion of the chemical changes in which this element is concerned; that sugar, for example, would be likely to appear in the urine, and that urea would to a greater or less extent be replaced by less perfectly oxidized bodies, such as uric acid. But a series of experiments on animals recorded by Senator in '*Virchow's Archiv*' for 1868 appear to show that this is not the case; and his conclusions are quite in accordance with clinical experience. In reality, the body adjusts its requirements to the necessities of its condition. In all probability one reason why persons affected with chronic bronchitis or other pulmonary disease almost always grow thin is that they instinctively learn to take very little food. But a far more important method of adjustment seems to be the avoidance of all bodily effort. So soon as such a patient begins to walk, especially on rising ground, dyspnœa sets in. Muscular exertion at once involves a demand for more oxygen than is contained in his arterial blood. So, again, the supervention of pyrexia in a case of this kind necessarily leads to a disproportionate increase in the rapidity and in the depth of the breathing. In illustration of this principle, Cohnheim instances the remarkable subsidence of dyspnœa which often occurs immediately after the crisis

in acute pneumonia, before the affected lung has even begun to recover itself.

Sometimes, however, dyspnœa of a very marked kind arises without ever leaving any reason to suppose that the arterialization of the blood is at all defective. This is the case, for example, in *diabetic coma*. And in *Bright's disease* a distressing shortness of breath may be the very earliest symptom of which the patient is conscious. Of this a very striking instance came under my notice some years ago. I was one day seeing my out-patients at Guy's, when the attendant asked me to listen to her chest, because her breathing had become so difficult and labored that she felt unfit for any exertion. After the most careful investigation I was unable to detect anything amiss with either the lungs or the heart. Then, as she told me she was thirsty, I thought I would examine her urine for sugar, and the result being still negative, I went on to test it with nitric acid, which brought down a large quantity of albumen. A few months later dropsy set in, and her case soon ended fatally.

Another form of dyspnœa, independent of any disease of the thoracic organs, appears to be of *nervous* origin. It is often ascribed to hysteria; but, according to Walshe, it is not always accompanied by other signs of that disease, although he has never seen it except in the female sex.

This will be the most convenient place for a description of a peculiar form of dyspnœa which depends upon *paralysis of the diaphragm*. The characters of this affection were first recognized by Duchenne, and they deserve careful study, because its true nature is very likely to be overlooked. So long as the patient is at rest his breathing is perfectly easy. But the slightest effort at once begins to distress him and to increase the frequency of his respirations per minute; when he walks he may experience a sense of suffocation as soon as he has made a few steps; in mounting a staircase, and even in speaking, he may be obliged to stop every instant to take breath. When he sighs he may experience a sensation as though the abdominal organs were being drawn up into his chest. The act of defecation may be much embarrassed. His voice may be weak; there may be more or less difficulty in coughing and sneezing, because he cannot take the deep, full inspiration which is a necessary preliminary; even a slight attack of bronchitis is attended with very great danger If one looks at the surface of his body while he breathes, the characteristic indication of paralysis of the diaphragm is generally at once apparent. During inspiration, when the ribs rise and the chest expands, the epigastrium and the hypochondriac regions become drawn in; during expiration they are pushed forward. In other words, their relation to the thoracic movements is exactly the reverse of what it normally should be. Sometimes it is not so easy to see the alteration as to feel it with the two hands placed just below the rib cartilages of the patient. If only one side of the diaphragm is paralyzed, as is sometimes the case, the corresponding hypochondrium may be drawn in while the other one protrudes in the natural manner. Among the examples of this affection recorded by Duchenne there are some in which it appeared at an advanced stage of progressive muscular atrophy, others in which it was associated with paralysis of many other muscles as the result of lead poisoning, and one in which is was hysterical. Walshe says that he has seen it in a well-marked form as a sequela of diphtheria. Erb cites Oppolzer as having observed it at the stage of puberty without any cause being discoverable; he also speaks of it as having been sometimes regarded as "rheumatic" and due to the influence of cold upon either the phrenic nerve or the substance of the muscle itself. Another cause which is mentioned by Duchenne as having been observed by Aran, is the extension of inflammation from the peritoneum or from the pleura. And he gives a case of empyema in which

the muscular tissue of the corresponding side of the diaphragm was of an orange-yellow color, and in which the fibres microscopically had undergone complete fatty degeneration. But for my own part, I must confess that I should hesitate to diagnose an actual paralysis of the diaphragm in every case of thoracic disease in which I saw one or both of the hypochondriac regions drawn in during the act of inspiration. That seems to me to be a very frequent occurrence under a great variety of circumstances, and to be often due to a mere inaction of the muscle, which surely ought to be distinguished from paralysis of it. The application of electricity seems not to be likely to afford any assistance in clearing up the difficulty, for in all cases in which the diaphragm has been observed to be paralyzed, it appears to have retained its power of responding to faradic stimulation of the phrenic nerves. The best method of galvanizing these nerves is, according to Duchenne, as follows: By two fingers, placed just outside the edge of one sterno-cleido-mastoid muscle, the skin is first drawn slightly inward; they are then separated, leaving between them an interval, upon which a small conical metal rheophore is pressed, so as to be just over the spot where the phrenic nerve lies upon the *scalenus anticus*. The rheophore is now given to an assistant to hold, and the same procedure is repeated on the opposite side of the neck. When both rheophores are fixed the operator takes one in each hand. He passes through them an interrupted current, which should instantly give rise to a contraction of the diaphragm, shown by the abdominal walls being pushed forward, while the lower ribs are separated from one another. Sometimes, however, the platysma interferes with this result, contracting with such force as to jerk the rheophores out of position. And sometimes it is necessary to shift them a little from spot to spot before one can succeed in acting upon the phrenic nerves. Erb recommends a somewhat different method; he places one pole upon the neck, and the other over the attachment of the diaphragm to the costal cartilages.

Whatever position may be adopted for the rheophores, the galvanism should be so used that the resulting contraction of the diaphragm may fall in with the natural respiratory movements. The current should be stopped as soon as the muscle has acted, and a few seconds later it should be reapplied. It seems to possess considerable therapeutical value. By means of it Duchenne succeeded in restoring completely the functions of the diaphragm in a man named Bonnard, who had advanced progressive atrophy of other muscles, but in whom the paralysis of the muscle in question was as yet recent and incomplete, as was shown by the hypochondriac regions receding only when he breathed deeply. After a few weeks of treatment he became able to ascend stairs and to walk long distances without discomfort.

II. Cough, as is well known, is produced in the following manner: A deep inspiration is first taken, the glottis is then closed, and a sudden expiratory effort being made, the glottis is allowed to open, causing a loud sound and allowing a blast of air to pass out, which may carry away with it any secretion or other substance present in the air passages. In describing laryngeal diseases I have already had to mention how they may modify the characters of cough, giving it a hoarse, or rough, or metallic quality, or rendering it almost noiseless. But in its ordinary forms cough is a symptom of affections of other parts rather than of the larynx itself, and, indeed, may almost be taken as an indication that the larynx is healthy.

The nervous mechanism by which cough is effected is reflex in its action. As a rule, the irritation which gives rise to it starts from the respiratory mucous membrane, as is evidenced by the consequent expulsion of mucus or pus in greater or less quantity. But sometimes the most violent and

repeated efforts of coughing bring away nothing. The cough is then said to be *dry;* and in the last century the distinction between a "dry" and a "humid" cough seems to have been regarded as one of the most fundamental points in regard to chest complaints. It is, however, quite possible for the air passages to contain mucus which is too viscid and too firmly adherent to be expectorated; and probably what is still more frequent is that some part of the respiratory mucous membrane is affected with slight catarrh, and that this condition either itself constitutes an "irritation" or else renders the surface sensitive to the passage of air over it, or to the disturbance produced by the laryngeal movements in breathing or speaking. But, on the other hand, there is no doubt that the starting point of cough is sometimes altogether outside the air passages, and, as may well be supposed, the recognition of this fact is of great importance in medical practice. The question has been worked out in experiments upon animals by several physiologists, the last having been Kohts, of Strasburg, whose observations appeared in "*Virchow's Archiv*" for 1874. To some of them I shall presently refer; but it is to be observed, in regard to cough, as to all other reflex phenomena, that although positive experimental results are of great clinical value, negative results prove very little. For under morbid conditions afferent nerves may transmit impressions with more than usual energy, or reflex centres may be unduly excitable, so as to be stimulated by impressions which normally should not disturb them.

The following appear to be the chief varieties of cough which have to be recognized, apart from affections of the respiratory organs:—

1. *Throat Cough.*—Kohts found, both in animals and in man, that irritation of the pharynx had the effect of producing cough in many individuals, but not in all. There is, therefore, no theoretical difficulty in admitting that catarrh of the fauces may be attended with cough, without there being a corresponding affection of the larynx, but the parts being continuous, it must always be difficult, if not impossible, to say that this is actually the case, especially as Kohts showed that the glosso-epiglottic folds, the ary-epiglottic folds, and the lateral edges of the epiglottis were among the most sensitive structures of all, so far as the production of cough was concerned. It is a somewhat different question whether or not a relaxed and elongated uvula frequently gives rise to cough, by coming into contact with the parts behind the base of the tongue. Dr. Mackenzie speaks of this as giving rise to a "distressing tickling cough continuing all day," and some physicians have, I believe, adopted the practice of having the uvula snipped off whenever a patient has complained of such a cough, for which no other cause could be discovered. But while not denying that this treatment often succeeds (as, for instance, in cases alluded to by Dr. Garrett, of Hastings, in the "*Lancet*" for 1872), I have been more impressed by the failures which I have from time to time heard of. Dr. Mackenzie speaks of an elongated uvula as being sometimes drawn into the larynx, when a person is sleeping on his back, so as to wake him up with a suffocative attack.

2. *Ear Cough.*—That cough can be excited by irritation of the external auditory meatus had been known long ago, but I think that it was generally forgotten until Dr. Cornelius Fox, of Ilfracombe, drew attention to it in the "*Lancet*" for 1867. He examined a number of persons and found that this peculiarity existed in about one among every five or six of them. He mentions the case of a gentleman who always experienced a feeling of irritation in the larynx, and had a violent suffocating cough, whenever he introduced a toothpick into the left ear; in him, too, a somewhat similar action was capable of being exerted in the reverse direction, for long-continued singing would cause him pain in the ear. Dr. Fox shows that the ear may sometimes be the starting point of a cough under such circumstances that the relation

may be overlooked. Thus, a healthy-looking woman, aged fifty, had for eighteen months had a most distressing cough. As she was deaf in the right ear, the meatus was examined, and was found to contain a hard plug of cerumen and to have a small ulcer in its floor. Almost immediate relief to the cough followed extraction of the wax and the application of nitrate of silver to heal the ulcer. In a patient of Mr. Toynbee's a cough was cured by the removal of a piece of necrosed bone from the external ear. It is obvious that a foreign body, such as a bead, might have a similar effect. Dr. Fox is no doubt right in maintaining that the afferent nerve in all such cases is the auriculo-temporal branch of the fifth, and not (as had been suggested) the minute auricular twig of the vagus.

3. *Tooth Cough.*—I know very little of cough as a result of dental irritation. But Dr. Fox incidentally mentions that it is well known to dentists that the stump of a tooth may be the starting point of a cough, and he also refers to cough in infants during the first dentition as ceasing when the gum lancet is used. In investigating an obscure case, therefore, one must not fail to examine the teeth.

4. *Stomach Cough.*—In the last century it was a favorite dogma that dry cough, and even humid cough, are very often produced by disorder of the digestive organs. The most recent exposition of such a view with which I am acquainted is to be found in "Copland's Dictionary." But, as so often happens in like cases, what has long since ceased to be taught by the faculty has become an article of faith among the public. Thus mothers still commonly refer to the stomach coughs in their children which are really due to catarrh of the upper air passages. Or, committing a fatal error, they set down to the same cause the dry cough of early phthisis, attended (as it often is) with nausea and loss of appetite and pain in the side. In his lectures on the pneumogastric nerve Dr. Habershon takes up only this side of the question, and no one is more likely to have met with examples of stomach cough, if it were really of frequent occurrence. I have heard the statement made that the characteristic sign of a cough due to gastric irritation is either that it comes on when the stomach is loaded with a full meal and disappears after the completion of digestion, or else that it occurs chiefly when the patient is in bed at night. The second of these criteria corresponds well with the fact that persons in whom intermission of the pulse and palpitation of the heart are caused by irritation of the stomach experience these symptoms when they lie down more than when they are sitting or standing. Kohts in his experiments failed altogether to excite cough by irritating the stomach. He cites from "Brücke's Physiology" a case in which a boy coughed day and night with the utmost violence and obstinacy until he vomited, whereupon the cough at once ceased; but he adds that Brücke, who himself made the observation, believed the starting point of the affection to have been, after all, something in the air passages which became dislodged when the stomach expelled its contents. Another instance, which Kohts had from Professor Leyden, is that of a patient who had repeated attacks of biliary colic, and who every time became affected with dry cough and with pain in the right hypochondrium twenty-four hours before the jaundice set in. Walshe says that he has known the trifling irritation due to the presence of an ascaris lumbricoides keep up reflex cough for several weeks.

5. *Centric Cough.*—Kohts found that he could sometimes excite cough in animals by mechanical or electrical stimulation of the floor of the fourth ventricle, and he thinks that the centre for this reflex act is situated rather above that for respiration. In hysteria, as is well known, a hard, dry, barking cough is common, which may be supposed to be centric in its orgin. A very remarkable instance of this was recorded by Dr. John Harley

in the "*Med. Times and Gazette*" for 1863. The patient, a girl aged fourteen, uttered a short bark seventy times a minute without intermission, so that, according to calculation, she must have coughed 40,000 or 50,000 times in the course of the day. She had had the cough a fortnight when she came under observation. She was treated with valerianate of zinc and with a cold douche and frictions to the spine, and in three days the cough ceased. A very similar case, in a child aged eight, was described by Dr. Whytt, more than a century ago, under the name of nervous cough. A remarkable feature in each of these cases was that the cough ceased instantly when the patient lay down. Dr. Whytt made an elaborate series of investigations into the effects of posture upon his patient, finding (for example) that the cough did not return when she sat up in bed so long as the feet were extended straight out, but that as soon as they were inclined at an angle she began to cough. He also observed that putting the feet in hot water, and even the contact of hot water with the soles, at once arrested the cough.

III. *Pain* is a symptom of various thoracic diseases, but it may also occur in the same positions when it is the sole indication that anything is the matter with the patient, and when, therefore, it can only be regarded as a substantive affection.

Sometimes, perhaps, the seat to which pain is referred is the interior of the lung itself. Walshe speaks of "pains deeply felt within the chest, and shooting in the direction of the pulmonary branches of the vagi and sympathetic," as existing "independently of any other deviation from health not only local but general." He also refers to "various anomalous and more or less painful sensations, felt deeply within the chest by phthisical patients."

But in the large majority of cases thoracic pain is referred to the chest walls, and especially to one or both of the infra-mammary or infra-axillary regions. Various names are given to pain in these situations, according to the views held with regard to its nature. Walshe describes in succession three separate affections, which he terms "pleurodynia," "thoracic myalgia," and "intercostal neuralgia." The distinctions which he would draw between them seem to be chiefly in reference to the intensity and duration of the pain, to its being accompanied by superficial tenderness, and to the presence or the absence of the "points douloureux" of Valleix. But in discussing neuralgia in general, I have already expressed doubts as to the value of any such differences. Walshe limits the term *pleurodynia* to attacks of pain of extreme severity, generally setting in suddenly and lasting only a short time. Of it he says first that it is "an actual rheumatism of the walls of the chest, affecting their muscular and fibrous textures," and then, a little further on, that "nerve fibres are implicated, and that rheumatic neuralgia of the intercostal nerves forms an element of it." Is it not clear that the distinction is only arbitrary? For my own part, I should be content with the term "pain in the side," unless *pleurodynia* may be used as a comprehensive name for all varieties of it.

Another difficulty, which seems to be completely ignored by systematic writers, but which in practice is of great importance, is to determine whether the respiratory organs are concerned in the production of a pain in the side, or whether it may not depend upon disorder of the heart or even of the stomach. The stomach has appeared to me to be very frequently its starting point when it is on the left side, which (according to Walshe) is the case in the majority of instances in pleurodynia; and I think that another frequent cause is ovarian irritation, especially in women who are hysterical.

The spine, too, must be thought of, even when the pain is unilateral. Lastly, a pain in the side, if recent, may be the precursor of an attack of shingles.

Again, it is necessary to bear in mind the possible presence of disease or injury of a rib. In July, 1877, I was consulted by a lady, the wife of an old schoolfellow, who told me that, having had a cough all the previous winter, she had one night felt something crack in her left side while she was coughing. Ever since then she had suffered from a continuous gnawing pain there. On examining the side I found considerable enlargement of one of the lower ribs, which seemed to me to be clearly the callus of a fracture. Under suitable treatment she got well, but for as long as six months afterward she still experienced some pain in coughing, which, however, was no longer limited to one spot and extended as high as the shoulder. She also said that she sometimes felt pain in the side toward night when she was fatigued, and that changes of weather seemed to increase it. I suppose that the fracture of a rib in coughing happens very rarely indeed. In a lecture reported in the "*Lancet*" for 1882, Mr. Marshall relates the case of a woman, aged thirty-five, who in the severe weather of the spring of 1881, owing to exposure to draughts, caught cold, shivered, and was attacked first with pain in the left side and then, a month later, with equally severe pain in the right side. In the previous year she had had acute rheumatism, and this had also been attended with pains in each side alternately. Her case was regarded as neuralgic, and powerful remedies were administered, but with only temporary benefit. At length she came to Mr. Marshall, who found two firm oblong swellings, one along the lower border of the right fifth rib, and the other at a corresponding spot upon the eighth rib. When they were pressed upon she experienced very acute pain shooting through to her back. They gradually softened into abscesses and were opened, when parts of each rib were found to be eroded and softened. Ultimately some pieces of dead bone came away and she did perfectly well. In another instance, also recorded by Mr. Marshall, an abscess evidently connected with disease of a rib, arose in a patient who had phthisis. Syphilitic periostitis is another affection that must be borne in mind, although it is much less common in the case of the ribs than of the sternum. Mr. Marshall speaks of it as occurring nearly always in women.

Methods of Physical Examination of the Chest.—Even now, when more than half a century has passed since the introduction of percussion and auscultation into England, the history of the discovery of these methods of diagnosis is full of interest. For one cannot help feeling that, besides directly revolutionizing the then existing knowledge of diseases of the chest, they also gave so powerful an impulse to the scientific spirit among medical men that they have indirectly brought about changes scarcely less important in every other department of medicine.

I. Percussion.—This method was discovered in the middle of the last century by an Austrian physician, Auenbrügger, who published in 1761 his "Inventum novum ex percussione thoracis humani abstrusos interni pectoris morbos detegendi." This method, however, seems to have been adopted by Stoll alone among contemporary physicians of eminence, and it had passed into complete oblivion when Corvisart, in 1808, brought out in Paris a translation of Auenbrügger's work, with commentaries of his own, based upon extensive practice at the Charité. Into this country it was introduced in 1825 by Sir John Forbes, then physician to the Chichester Dispensary.

The way in which Auenbrügger performed percussion was by striking the

chest with the fingers "brought close together and stretched out straight." He also directed that a glove of unpolished leather should be worn, and that the patient should be clothed in a shirt drawn tightly over the chest. Even now the whole of the fingers of one hand are sometimes used when one wishes to ascertain broadly what is the state of the backs of the lungs, but for the finer objects of percussion such a procedure is altogether inadequate. In 1828, Piorry, who was himself afterward physician in the Charité at Paris, published a work upon what he termed "mediate percussion." This he described as consisting in the use of a thin plate of ivory, called a *plessimètre*, to be held by the left hand in contact with the surface of the chest, while a gentle blow is given to it with the tip of the right forefinger, or with the tips of the fore and middle fingers. Since that time a hammer, which is known as a *plessor*, has also been introduced, and on the Continent these instruments are employed more or less generally, and in England sometimes for class teaching. But in this country the usual practice is to simplify Piorry's method by using the left forefinger in the place of a plessimeter. It is curious that in his original work Piorry himself speaks of that plan as having been already adopted by certain English and American physicians who had attended his lectures.

It must not be supposed that percussion in this simple form is very easy. On the contrary, a great deal of practice is necessary to enable one to obtain with certainty correct results. I find in the wards that clinical clerks commonly continue to make blunders in percussing long after they have mastered the difficulties of auscultation. It is very important that the blow should come from the wrist, that it should be sharp and sudden, so as not to damp the sound which is produced, and that the fingers at the moment of striking should be as nearly vertical as possible. The amount of force that should be employed, and the extent to which the finger should be raised before striking, vary with the thickness of the soft tissues over the part of the chest which is to be struck, and I think that every physician, even without being aware of it, modifies his manner of percussing in different patients and upon different regions of the chest according to what experience has taught him to be necessary in order to elicit the best possible sound under various conditions. As a rule, percussion can be practiced by a skilled observer without causing any sensation that is complained of by the patient as being painful, but in delicate women, and even in some very thin men, the sternum and the ribs may be so exquisitely tender that a satisfactory sound can be elicited only with very great difficulty. Sometimes cough is excited by every attempt at percussion, and a forcible blow may even lead to blood spitting, so that it is wise to abstain as far as possible from this method of physical examination when there has been recent hemorrhage from the lung.

It must not be supposed that in different persons one can always elicit the same sound by percussion of the chest, if the organs are healthy; nor, again, that the sound ought to be the same over different parts of the chest of any one individual. But, in health, the range of possible sounds is almost limited to a simple series. One extreme is heard where a thick mass of solid tissue lies behind the ribs, as over the centre of the heart just below the fourth left rib, or over the liver at about the right seventh and eighth ribs. This sound is said to be "dull." The other extreme is heard where the ribs cover a thick substance of lung, as in front below the clavicles or behind below the scapulæ. This sound is commonly called "clear" or "resonant." Both sounds are difficult to describe satisfactorily, but they are easily recognized in practice. With regard to the names for them it is important to bear in mind that "resonance on percussion" means altogether a different thing from what is called *resonance* in works on acoustics. Such a technical and conventional way of using the term would, no doubt, have

been better avoided, since it is apt to engender confusion; but in England it has been universal, and I do not know of any good substitute. Between the extremes of dullness and resonance, there are all gradations, for which the expressions "partial dullness," "incomplete resonance," "muffled resonance" are employed. These varieties of percussion sounds are heard at the borders of a resonant or dull area, especially where the lung overlaps a solid organ by a thin edge. At such points one obtains many different sounds according to the amount of force used in striking. A gentle blow elicits sound only from the parts immediately below the spot which is struck; a more forcible blow affects deeper parts as well. Thus it is usual to speak of "superficial" and of "deep percussion." But it must be remembered that in "deep percussion" the sound is also modified by the structures which are adjacent laterally, and (as I think) in this particular manner, that a resonant area interferes with the sound yielded by a dull area on forcible percussion, but not that a dull area interferes with the sound yielded by a resonant area. For example, by "deep percussion" over the heart one can often elicit a more or less resonant sound even when no lung covers the heart. But on deep percussion over the lung it would not be possible to detect any degree of dullness, however slight, as the result of the presence of the heart or of the liver by the side of the lung. When a solid organ is overlapped by lung, it is often essential to employ deep percussion as the only means of detecting the distance to which the solid organ extends. But under other circumstances superficial percussion should be used when the object is to map out the relative positions of the viscera. It is otherwise when one wishes to determine the presence or absence of disease of the pulmonary tissue. In such cases one has to ascertain by repeated trials what amount of force is necessary to bring out an altered percussion sound most distinctly.

As a rule, it is by comparing the two sides of the chest together in the person under examination, rather than by an absolute standard, that one judges of the results obtained by percussion. But if both sides are diseased, this method may altogether fail. And even when only one side is diseased it is necessary to have some kind of standard, since without it one could not tell, by percussion alone, on which side the disease lay. Now, I have already remarked that in health the percussion sound is different in different individuals. The thinner a man is, the more "resonant" his chest is likely to be; in thick set, very muscular, or very fat persons it is sometimes by no means easy to elicit a clear sound anywhere, particularly over the backs of the lungs. The differences in percussion sound at different parts of the chest in health depends upon obvious anatomical conditions, but they nevertheless require careful study in actual practice. In front the sound is modified by the position of the liver and of the heart on the right and left sides respectively; and immediately beneath the clavicles it is more resonant near the sternum than it is further outward. Behind, in the supra-scapular regions, one sometimes has to use a good deal of force in order to elicit anything but a dull sound; and even between the scapulæ the sound generally becomes gradually more resonant as one passes downward. Below the scapulæ the sound is generally not very much less clear than in front; the resonant area is on the left side slightly larger than on the right side, extending about a finger's breadth lower. While the back is being percussed the patient should have his shoulders drawn forward as much as possible, crossing his arms over the chest.

In a former paragraph I spoke of the variations of the percussion sound in health as being *almost* limited to a simple series. The qualification was necessary for two reasons. 1. When the blow is struck upon the clavicle or the sternum or upon one of the ribs or rib cartilages, a high-pitched tone

is added to the sound, giving it what is termed an "osteal" character. 2. In a very young child, especially when it is screaming, one sometimes elicits what will presently be described as a "cracked-pot sound."

With regard to the theoretical interpretation of the sounds generated by percussion there have been great differences of opinion. Even now scarcely any two writers seem to express the same views. Nothing is more confusing than to read in succession three or four works, and to attempt to compare together the statements contained in them, even as to some matters of fact, such as the pitch of particular sounds, etc. And the variations in nomenclature are absolutely bewildering. Dr. Walshe insists that the use of the terms "dull" and "resonant," or "clear," is inaccurate, because they represent conditions which, instead of being "simple, are in reality made up of several elements, capable of separate analyses;" and, of course, it is true that writers on acoustics, in enumerating the properties of sound, allude to no such distinction. But in my judgment the analysis which Dr. Walshe himself has attempted throws but little light on the subject. The view which to me appears the most satisfactory—if I may assume its theoretical accuracy, as to which I am not competent to express an opinion—is that enunciated by Dr. Gee. He declares that the terms in question are perfectly capable of scientific definition. According to him, dullness means *absence of tone;* a part is dull when the sound which it yields on percussion is a mere noise, without any regular succession of impulses such as constitutes tone. On the other hand, varying degrees of clearness or resonance correspond with the admixture, in different proportions, of noises and tones. No percussion note is ever perfectly pure; and thus beyond the "resonance" yielded by the healthy chest of even the thinnest person, there are "hyper-resonant" sounds which may accompany certain diseases.

Another question upon which writers differ is as to the seat of the vibrations causing the tone which is elicited by percussion over the healthy chest. Dr. Gee refers this tone to the middle-sized and largest bronchia; following Wintrich, he thinks that the pulmonary vesicles and the bronchioles are too small to yield it. But it certainly seems to be a great obstacle in the way of the acceptance of that view that in bronchitis resonance is seldom, if ever, impaired, however completely the tubes become filled up with pus or mucus. I suppose that Dr. Gee would attribute the *noise* or toneless part of every percussion sound to the thoracic walls, seeing that its amount, in proportion to that of the tone, varies with their thickness and with the extent to which they are loaded with fat. Now, Dr. Bristowe believes that the whole of the sound is "mainly due to the vibration of the thoracic walls alone." He assumes that "so much of each half of the thorax as bounds lung tissue vibrates bell-like when any part of that half is struck, and that the impure musical sound which is elicited comprises a fundamental tone due to the vibration of the whole or a large portion of the side, and harmonic tones due to the vibration of aliquot parts of it." It is a strong argument in favor of this doctrine that deformity of the chest, without any apparent thickening of the parietes, may give rise to absolute dullness on percussion, notwithstanding that the lung beneath is quite healthy. When there is lateral curvature of the spine, for example, the rounded projection formed by the ribs on one side of the back generally, if not always, yields a dull sound. Another argument is afforded by a peculiar alteration in the percussion sound beneath the clavicle, observed when a certain quantity of fluid is effused into the lower part of the corresponding pleural cavity. This is a matter which I shall have to discuss elsewhere. But I may say at once that no explanation with which I am acquainted seems to me so satisfactory as that suggested by Dr. Bristowe, namely, that the vibrating area is diminished, and that consequently it yields a

fundamental tone which is raised in pitch. Now, the subclavicular percussion sound is not affected in the same way when there is pneumonia of the lower lobe of the lung. One must therefore assume, if one adopts Dr. Bristowe's view, that hepatization of the lung, notwithstanding that it gives rise to a more or less dull sound when percussion is made directly over it, does not, like pleural effusion, completely damp the vibration of the corresponding part of the chest wall and prevent it from joining with the rest of the side in emitting a tone when a distant part is struck. One point in which I cannot agree with Dr. Bristowe is his rejection of the distinction between "superficial" and "deep" percussion. He evidently thinks that his doctrine is incompatible with the admission that a percussion sound can in any way be modified by the presence of solid matter within the chest, except in so far as it is in direct contact with the inner surface of the chest wall. But about the reality of this distinction there is, I think, no doubt whatever.

I have already spoken of the chest as being sometimes *hyper-resonant;* but have abstained from using the term *tympanitic,* which has often been employed as meaning the same thing. Originally, a percussion sound was called tympanitic, when it was such as would be yielded by an abdomen in which the intestines are distended with gas; for tympanites has, since before the days of Celsus, been a name for that condition. And, as a matter of fact, the sound generated by percussion over an emphysematous (or over-distended) lung, when the chest walls are rounded and thin, often approaches, if it does not actually reach, a tympanitic quality. But there are other cases in which a tympanitic is far from being identical with a merely hyper-resonant percussion sound. We now reach a point which has been a stumbling block to all writers on percussion, and about which I always myself felt the greatest difficulty, until I carefully read Dr. Gee's work. We have seen that, according to this writer, *dullness* means an absence of the tone which would be yielded by percussion over healthy lung, the sound which is generated being a mere confused noise. Hitherto I have scarcely alluded to the *pitch* of this tone; within certain limits it varies in different individuals. But here comes the point which seems to me to be essential to a right understanding of the matter. It is obvious that the absence of the tone yielded by percussion over the healthy chest is by no means incompatible with the presence of other tones having a different origin. Thus, although dullness and hyper-resonance are at opposite ends of the scale, it is quite possible for a sound to be at the same time dull and tympanitic, if, besides the toneless noise yielded by chest walls when there is solid matter beneath, it contains a tone due to the vibration of some part of these structures, or of air in a space within the chest. I do not go with Dr. Bristowe when he assumes that in a case of pneumothorax, or when the lung is excavated into vomicæ, the tones contained in the sound elicited on percussion over the affected part are still due to the vibration of the thoracic parietes alone. It seems to me that they may fairly be referred to the vibrations of the air, and of the walls of the space in which it is confined. Now, Dr. Gee classifies percussion sounds containing adventitious tones as follows, according to their pitch. Those which are highest pitched he terms *Osteal,* because they are yielded by the hard, solid tissues, cartilage, and bone, as has already been mentioned. Those which are somewhat lower he terms *Trachæal* or *Tubular,* being more or less like the note yielded by the trachea when the mouth is a little open. Lower still are tones which he proposes to call *Subtympanitic;* they are such as are usually yielded by percussion over healthy lungs in their natural state of distention. The lowest pitched tones of all are the *Tympanitic.*

I have still to mention certain modifications of the percussion sound

which are noticed under special circumstances; they are (1) the "metallic ring" or "amphoric note," and (2) the "cracked-pot sound" or *bruit de pôt fêlé*. The *metallic ring* is described by Dr. Gee as consisting in an "overtone, existing either apart and alone, or as a harmonic superadded to the fundamental tone, which itself may either be clear or muffled." It is commonly heard when percussion is made over a stomach distended with air. It may, also, be elicited by giving a sharp fillip to the cheek when it is blown out to a certain point. The *cracked-pot sound* is exactly like the chinking of coins, or the sound produced by clasping the hands loosely together and striking them upon the knee. I am disposed to believe that it always depends upon the propulsion of air out of a space through a more or less narrow opening. It was originally noticed by, and received the name of *bruit de pôt fêlé* from, Laennec.

Lastly, it must be stated that, besides the sound which is yielded by percussion and which is audible to bystanders as well as to the person who percusses, he is himself conscious of differences in the degree of resistance offered to his fingers, and that this is often of considerable assistance in enabling him to draw correct inferences from his observations.

As to the practical significance of the various modifications of sound elicited by percussion of the thorax, it is not necessary for me to say much in this place, since whatever remarks I might make would necessarily have to be repeated elsewhere, when I come to discuss the various diseases of the lung and of the pleura. But I may point out, in general, that dullness on percussion may mean either of two things: (1) consolidation of the pulmonary tissue, which may itself be due to several different causes; (2) compression of it by fluid or by some adjacent organ enlarged. As a rule, the diagnosis between these various conditions is mainly based upon other considerations, independent of percussion. But the dullness resulting from the presence of liquid in the pleura has the peculiarity of being more complete or absolute than that which arises in almost any other way; and the sense of resistance is also far greater, especially when the quantity of liquid is large. A clear tympanitic sound, if it be too well marked to be due to emphysema of the lung, can hardly depend upon anything except the presence of air in the pleural cavity, though it is, perhaps, right to allude to another very rare affection, namely, diaphragmatic hernia, with escape of the stomach through the diaphragm into the chest. The various combinations of dullness with tympanitic, subtympanitic, tracheal, and osteal tones are met with chiefly when there are cavities or vomicæ in a part of the lung which is separated from the surface by a thickened and adherent pleura or by a layer of consolidated pulmonary tissue, or when air is present in a pleural cavity of which the walls are indurated. The significance of the metallic ring and of the cracked-pot sound will have to be fully considered elsewhere.

This will be the most convenient place for me to mention an effect of "immediate" percussion, which is not infrequently seen in emaciated persons, namely, the production of contractions in muscles which receive the blow, especially in the pectoralis major. A rounded knot rises up at the spot which is struck, and from this a smaller ridge spreads away, wave-like, along the fibres toward each of their ends. This "idio-muscular contraction" has been supposed to be characteristic of phthisis. But the fact is that it occurs in wasting diseases generally.

Auscultation.—Dr. Gee points out, and it is a very interesting fact, that this, a still more important method of physical examination than percussion, arose directly out of Corvisart's researches. Among those who followed the practice of that professor at the Paris Charité were two friends, Bayle and

Laennec. In endeavoring to distinguish between active and passive enlargement of the heart when percussion showed its size to be increased, they were in the habit of carefully observing the character of its impulse; and they became accustomed to apply the ear to the cardiac region for this purpose rather than the hand. One day Laennec was consulted by a young woman who had the general symptoms of disease of the heart, but in whom, as she was fat, he was unable to feel the impulse satisfactorily. For reasons of delicacy, he was hesitating to put his hand to her chest, when he remembered the fact, that by applying the ear to one end of a plank one can hear the scratch of a pin at the other end. So he took a quire of paper and rolled it very tight, And then, placing one end on the præcordial region, and leaning his ear on the other end, he found, to his surprise and pleasure, that he could actually hear the beating of the heart more plainly than when the ear was in immediate contact with the thoracic wall. He soon began to employ the new method of investigation, which he termed *l'auscultation médiate*, in phthisis and in other pulmonary diseases, read a memoir on the subject in 1818, and published a work in the following year. In 1826 he died at the age of forty-five, having worked out the subject, so far as concerns lung affections, in such a way that there has been very little for later observers to do. It is remarkable that of the very few references to sounds heard by listening over the chest which have been traced in medical writings of an earlier date, two are in the works of Hippocrates, the father of medicine; namely, the well-known allusions to the leather sound of pleural friction and to succussion splash. The only other notice of any sound produced in the lung is, according to Dr. Gee, one relating to pneumonic crepitation, in van Swieten's "Commentaries," published in 1774.

The Stethoscope.—In examining the backs of the lungs, we still sometimes apply the ear directly to the surface of the chest, or rather so that only a towel or the patient's night dress separates one's ear from his skin. In this way one can rapidly judge of the state of a large part of each lung; and it is often more convenient than using an instrument when the person is very ill and has to be lifted up in bed by attendants. But in all other cases we employ some form of stethoscope. That which was originally designed by Laennec has long ago been discarded; it was of a clumsy shape and needlessly elaborate in construction. What is now used is generally a light hollow stem of wood or metal, having a flat ear piece at one end and spread out into a concavity at the other end, which is placed upon the spot to be examined. In applying it, one must be careful not to hurt the patient by pressing too hard or by bearing unevenly upon one side of the rim of the concavity. The ear must always be moved to the stethoscope and not the stethoscope to the ear. Recently, it has become the fashion to employ a stethoscope with a flexible stem and with two long metal and ivory ear pieces, one of which is introduced into the meatus of each ear, and is held in position by an elastic band. To the use of this binaural stethoscope, as it is called, every one should accustom himself, because of the facility with which the back and sides of the chest can be explored by it in persons who are too ill to sit up. Students, I find, like the instrument on account of the loudness with which sounds are transmitted through it. But I am not yet satisfied that when they reach the ear they are as clear and distinct as when an ordinary stethoscope is employed. And the slightest movement of the flexible part of the binaural stethoscope produces noises which are apt to be perplexing. Whatever form of stethoscope may be employed, one must take care that the patient's clothes do not rest against it nor against the surface of the chest near the spot to which it is applied. And another point to be kept in mind is, that if the instrument is placed on a part of a man's chest where hair is growing, a fine crackling sound is often

produced which may be very like what will be presently described as crepitation. Guttmann observes that all difficulty from this source may be avoided by wetting the surface.

So far as the respiratory organs are concerned, the practice of auscultation falls under two divisions, that of the breathing and that of the voice.

1. *Auscultation of the Breathing.*—On listening over the lungs of a healthy person, one hears each time he breathes a delicate, rustling sound. Of this no further description is needed, since the only way of really learning to know it is to have heard it again and again and in many different individuals. It is commonly called the "vesicular murmur," having been so named when the idea that it arose in the air cells of the lung was accepted without question. Such an association with a theory which is disputable, though (I believe) correct, is, of course, an objection to the term, and I have no doubt that Dr. Walshe is right when he says that it has also often led to an erroneous impression with regard to the character of the sound itself. But there seems to be no possibility now of securing formal acceptance for the name "pulmonary respiration sound," which he proposes in its place. The greater part of this sound accompanies the act of inspiration; expiration is either altogether noiseless or attended with a much shorter and fainter murmur, which, however, is almost continuous with the inspiratory. In some persons the vesicular murmur is much louder than in others. In children it is particularly loud, so that when under morbid conditions an equally intense murmur is heard in an adult this is sometimes dignified by the title of "puerile breathing." But in very thin adults it is often scarcely less loud, even when all parts of the lungs are healthy. On the other hand, there are some people in whom the act of breathing is attended with scarcely any sound, even when one tries to make them breathe deeply, which should have the effect of exaggerating the vesicular murmur. One must, therefore, listen over different parts of a patient's chest before one draws conclusions from the degree of loudness of the murmur at a single spot. It is naturally louder where the thoracic walls are thin than where they are covered with thick muscular masses, and over the edges of the lung it is less loud than elsewhere; in other words, its intensity in health is generally proportionate to the degree of resonance on percussion at various parts of the chest.

At the bases of the lungs, especially in persons who are confined to bed, by whatever cause, the vesicular murmur is sometimes found to be mixed with a crackling sound, which may be mistaken for crepitation, but which disappears when the breath is drawn deeply two or three times in succession, so that it must be due to slight collapse from disease. Of course, it has no clinical significance. On the other hand, there are certain spots at which in most healthy individuals the breathing is attended with a sound that differs from the vesicular murmur. They are (1) the spaces between the scapulæ over an area of variable extent, but somewhat lower in situation on the left side than on the right; (2) the regions below the sterno-clavicular joints, especially on the right side and in females; (3) over the spines of the seventh cervical and a few adjacent vertebræ, extending sometimes a little outward toward the supra-scapular region, especially the right. The sound heard over these parts is called "Bronchial breathing," because it is transmitted from the main bronchi. It differs from the vesicular murmur in having a blowing character, in the fact that the expiratory part of it is as loud as (if not louder than) the inspiratory, and in there being an interval or pause between them. A similar but louder sound is heard on auscultation over the trachea, and also over the larynx, where it is of a more "whiffing" quality.

To be able, in practice, to distinguish a vesicular murmur from bronchial breathing is the most essential step in auscultation. For the latter sound, with certain modifications of it, may be heard, in disease, over any part of the chest, and constitutes one of the chief signs of nearly every important pulmonary affection. In bronchitis, indeed, bronchial breathing does not occur, but it may accompany any disease in which the lung is either compressed or consolidated, or hollowed into cavities. In other words, its range is generally conterminous with that of percussion dullness though the two phenomena by no means necessarily accompany one another in each individual case. The modifications of bronchial breathing concern its quality. A blowing character belongs to them all; each of them consists of an inspiratory part and of an expiratory part, separated by an interval. What distinguishes them is that the blowing sound is more or less hollow. In its most extreme form it resembles the sound produced by breathing into a large, empty glass bottle; it is then named *amphoric*. A less marked degree of the same quality of sound is called *cavernous*, because it is commonly heard over vomicæ, which are sometimes spoken of as caverns in the lung. When it is merely whiffing, it is by most writers termed *tubular*, although others employ "tubular breathing" as a mere synonym for "bronchial breathing." It must be understood that between these several modifications of bronchial breathing all gradations exist, so that it is often difficult to know whether to call a sound "tubular" or "cavernous," "cavernous" or "amphoric." But there are, nevertheless, good grounds for maintaining, as far as possible, the distinctions between them; and a point of some importance is that if tubular or amphoric breathing is heard, at whatever part of the chest, there can be no doubt that disease is present, even though the spot should be one in which "bronchial" breathing is audible in health.

With regard to the physical causes of the vesicular murmur, of bronchial breathing, and of its various modifications from tubular up to amphoric, there has been much speculation. Of late the tendency has been to apply to them strictly the theory of the *veine fluide*. According to this theory, a blowing sound is generated whenever a fluid (whether liquid or gas) passes from a narrow space into a wider one. Now, during inspiration, this condition is fulfilled at two points in the respiratory tract: (1) when the air enters the trachea from between the vocal cords; (2) when it emerges from the extremity of each bronchiole into the ampulla formed by the air sacs around. On the other hand, during expiration, the only point at which a *veine fluide* can be formed is at the upper orifice of the larynx; but since the false vocal cords form a lip on each side, the resulting sound should be audible not only above the spot at which it is formed, but also below. It is obvious that these facts accord perfectly with what has been stated with regard to the respective characters of the vesicular murmur and of the sound heard on auscultation over the larger air passages. The former belongs mainly to the act of inspiration; the latter is divided into more or less equal parts, of which one attends inspiration and the other expiration. That the vesicular murmur is generated somewhere below the larynx has, indeed, been demonstrated, in a series of experiments of which I gave an account in the "*Med.-Chir. Review*" for July, 1873, by certain French observers, especially Bergeon, Chauveau, and Boudet. They cut through the trachea of a horse and drew the lower end of the tube outward through the wound in the skin so that no *veine fluide* could possibly arise in it; after this operation they found, on listening over the animal's chest, that the vesicular murmur still remained audible and that its intensity was little, if at all, diminished. On the other hand, by dividing the pneumogastric nerves in another horse, they succeeded in abolishing the vesicular

murmur. The explanation of this is supposed to be that the muscular walls of the bronchioles were paralyzed, so that they opened into the air sacs by wide, funnel-shaped mouths—which would yield no *veine fluide*. In this last experiment the laryngeal sounds heard over the trachea are, of course, still persistent. The theory of the *veine fluide*, however, in the form in which it is stated by the French experimenters, does not account for the fact that in most persons expiration is attended with a murmur, although a faint one. Moreover, in pulmonary emphysema, the expiratory murmur becomes greatly prolonged and very noisy, while the inspiratory murmur is much diminished. But it seems to me that these difficulties may be met as follows. It is a very slight extension of the results obtained by Bergeon, in experimenting with a tube provided with a lip or rim where it was narrowed, to assume that a lip, at the orifice of a contracting cavity, would generate a *veine fluide* in the cavity itself. Now, in emphysema it is fair to suppose that the mouth of the bronchiole projects some distance forward into the space formed by the dilated air sacs around it; and even under normal conditions it may do so sufficiently to produce the faint expiratory murmur which is heard in healthy persons.

The French observers whose views I have been quoting suppose bronchial breathing, whether in health or in disease, to consist of sounds generated in the larynx and transmitted downward with more or less modification until they reach the ear of the auscultator. Indeed, they seem to have proved this experimentally. A horse was affected with pneumonia, and an intense *souffle tubaire* was audible. Chauveau and Boudet cut through the trachea, so as to allow air to enter the lungs without passing through the glottis, and the *souffle* at once disappeared. It would be of great interest to observe the same point in man, either in cases of cut throat or after tracheotomy; but the requisite conditions can scarcely ever be satisfactorily fulfilled, for, unless the orifice into the trachea is as wide as the calibre of the tube itself, a *veine fluide* may always be generated, at least during inspiration. Stokes, however, long ago pointed out that when the larynx is diseased it is often difficult or impossible to determine whether the lungs are or are not healthy. And it certainly seems very probable that extensive ulceration, destroying the attachments of the vocal cords, may prevent the formation of a *veine fluide* in the air which passes the glottis, and so render impossible the development of bronchial breathing in diseased lung below.

As to the question why laryngeal sounds should be transmitted to the surface of the chest better when the lung is consolidated or compressed than when it is healthy, there has been much discussion, and I am not sure that it can even now be answered quite satisfactorily. It will be better, however, to postpone the consideration of it until we come to the subject of bronchophony. Of the "hollow" modifications of bronchial breathing, from tubular to amphoric, all that need be said in this place is that the more marked the hollow quality the more one is justified in asserting that a space filled with air, of larger size than even a main bronchus, has been formed in the substance of the lung, unless, indeed, the pleural cavity itself should contain air. But even in acute pneumonia, when there has not been the slightest destruction of pulmonary tissue, it is surprising how hollow the bronchial breathing sometimes is. And Dr. Walshe speaks positively of having heard sounds to which he would assign the name of cavernous, in cases, whether of pneumonia or of pleurisy, in which there was no excavation and in which the lung was simply consolidated or compressed over large bronchial tubes.

Hitherto, in describing the signs derived from auscultation, I have spoken only of sounds which are identical with, or are at least derived from, those that can be heard on listening over the healthy lungs or air

passages. But there are other sounds which are altogether adventitious and have no physiological representations. Thus in pleurisy a *friction sound* is heard, which, of course, has its origin on the surface of the lungs; but as this is almost, if not quite, peculiar to that disease, I may with advantage leave it to be discussed hereafter. With regard to the remaining sounds which are found within the lungs themselves there has unfortunately been much confusion of names. Some writers describe them all indifferently as "Râles" or as "Rhonchi," the latter term having probably been originally intended as a Latin equivalent for the French term which had been introduced by Laennec, and which was rendered into English as "rattle" by those who first promulgated his views in this country. According to this use of the words in question, each of them is applicable indifferently to two kinds of sounds, which are very unlike one another, and which may be distinguished by those of the one kind being *continuous*, whereas those of the other kind are *interrupted*, and "crackling" or "bubbling" in character. Very often the two kinds are spoken of as being respectively "dry" and "moist;" but this is better avoided, for reasons which will presently appear. Now, there is another meaning of the term rhonchus, which, I believe, was first given to it by the late Dr. Latham, of St. Bartholomew's Hospital, and which afterward was sanctioned by Sir Thomas Watson. In that sense it is limited not merely to continuous or "dry" sounds, but to a single variety of dry sounds. For my own part, I always like, as far as possible, to have simple substantives for names, and therefore I propose to follow the writers whom I have just named in their use of the term *rhonchus*, especially as there is another term, *sibilus*, which is exactly applicable to the only other sound known to auscultators which is certainly dry in its origin. Both these sounds belong mainly to bronchitis, and I may therefore postpone my descriptions of them.

On the other hand, if the two continuous sounds are to have each a name of its own, the term râle may fairly be confined to interrupted sounds, of which, however, there are several varieties. They occur in almost every disease to which the lungs or the air passages are liable. They are generally attributed to the disturbance of fluid lying in a bronchial tube, or in a vomica, or in the pleural cavity, by air bubbling through it; and on that account they are known as moist sounds. But, as I shall have occasion to point out when speaking of the "crepitation" of acute pneumonia, there have long been observers who have maintained a different opinion with regard to the origin of some at least of these sounds, ascribing them to the sudden separation of surfaces that had been in contact, just as one can make a series of clicking sounds by pulling away the lower lip from the gum several times in rapid succession. And in 1871, Traube, in the "*Berlin Med. Wochenschrift*," applied this view still more widely, referring a great number of the râles found even in the larger tubes to the momentary detachment of portions of viscid mucus from their sides, by the air current passing into or out of them. It is, indeed, difficult to conceive that in such thick fluids as are ordinarily found in the air passages bubbles can be made and broken with sufficient frequency to account for the abundant râles that are often heard. And, as Traube remarks, râles are often to be detected in cases of pleurisy or of hydrothorax when there is not the least reason to suppose that any fluid is present in the tubes, and when, therefore, they can only be attributed to the separation of the sides of the tubes that had been forced against one another in compressed parts of the lungs. He says that he has several times determined the absence of fluid in such cases at an autopsy. A further point to which he draws attention is that a fine râle can be produced by pressing gently with the stethoscope upon the surface of a healthy lung, even of a recently killed animal.

Wintrich has shown that a like result may also be brought about by inflating the collapsed lungs after death. ("Virchow's Hdbh.," Bd. v, Abth. 1.)

The usual mode of classifying râles is by what may well at first sight appear to be an arbitrary principle, namely, according to the impression which they give as to the size of the bubbles which might be concerned in producing them, or, perhaps, rather of the spaces in which they are found. This distinction, however, of "fine" or "small râles" from "coarse" or "large râles" is one which is in practice easily drawn.

But there is another distinction between different kinds of râles which is, I think, of far greater importance than that of their apparent size, and which depends upon whether the tubes in which they are found are surrounded by spongy or by consolidated lung tissue. In the latter case they have a peculiar quality which in the former is wanting, and which the ear easily recognizes, although to describe it in words is very difficult. Perhaps one might say that râles arising when the lung is solid differ from other râles in being "bright" or "clear" or "ringing." But the name which is commonly given to them is that of "consonating râles," originally proposed by Skoda. Nor do I know that there is any great objection to the continued use of it, even if we reject the doctrine that their peculiarities depend upon *consonance* in the sense to which the word is strictly limited by writers on acoustics. We have already found ourselves obliged to employ the term "resonance," as regards the results of percussion, with an arbitrary technical meaning. And precisely in the same way we may, perhaps, adopt consonance in dealing with the phenomena of auscultation. It will, of course, be understood that consonating râles are often associated with bronchial breathing, both in its simple form and in its "hollow" modifications. They are also associated with a sign which I shall describe a little further on as bronchophony, and in speaking of which I shall have to discuss somewhat fully the whole question of consonance.

Not only does the "consonating" quality divide râles into two well-defined groups, but it also appears to me to be essential to the definition of certain names which are in clinical practice applied to particular varieties of them. Thus for the largest râles of all, *gurgling* is often used as a synonym, but only, I think, where they possess the "consonating" quality in a more or less marked degree. The same thing may be said, if I am not mistaken, of those smaller râles which are termed "crepitant râles" or sometimes "crepitations." Of these one particular kind almost (if not quite) peculiar to an early stage of acute pneumonia is termed *fine crepitation*. This sound will be described elsewhere, and its origin discussed.

To non-consonating râles, such as belong chiefly to bronchitis, it does not appear to be necessary to apply any special designations. But it is, perhaps, worth while to notice that the term "mucous râle," which is now very generally abandoned, was confined to such râles as, besides being of medium size, were devoid of the consonating quality.

The statements made in the previous paragraphs must inevitably appear perplexing to the tyro; and it would be easy to draw up a far simpler classification of the adventitious sounds heard on auscultation of the lungs. But to every simple classification that has yet been proposed there are two inseparable objections. One is that in endeavoring to convey to other persons definite ideas as to the auscultatory phenomena observed in a case of pulmonary disease, or in receiving from them the same kind of information, we are at once baffled by uncertainty as to the sense in which we or they employ different terms, unless we are familiar with the various meanings that are assigned to them by practical men. The other objection is that in studying medical literature, as soon as we pass beyond the scope of a single text-book, we fall into the gravest mistakes if we imagine that the

language used by different writers has always the same sense. And, as for the future, all experience goes to show that if the most distinguished physicians of the day were to meet week after week until they adopted a uniform terminology, and agreed to impose it on others, nearly every one of them would within a year forget in his own practice to make use of it.

2. *Auscultation of the Voice.*—If, while one is listening over the lungs of a healthy man, one tells him to speak, one generally hears an indefinite humming or buzzing noise. In a woman the sounds come to the ear much more sharply, but still so that one is unable to distinguish the words that are uttered. Only over the upper part of the interscapular region on each side is there in some persons a space in which one can hear the voice clearly, and in which the separate words are perceived almost as they are when one places the stethoscope over the larynx and trachea, but, of course, with less loudness. Now, in disease the voice may be carried to any part of the chest thus distinctly, and may be heard far more loudly than is ever the case in health. For this "increased vocal resonance," as it is often termed, Laennec invented two names according to its degree of intensity. When the voice, however distinct, gave the impression of still coming from a distance, he spoke of "Bronchophony;" when it appeared as though it were formed within the chest immediately below the spot at which the stethoscope was applied he used the term "Pectoriloquy." Bronchophony, as a rule, is associated with bronchial or tubular breathing and with crepitations. Pectoriloquy accompanies cavernous or amphoric breath-sounds and gurgling. In other words, bronchophony attends those diseases, or those stages of diseases, in which the lung tissue is solidified or condensed by pressure; pectoriloquy occurs chiefly when it is hollowed out into a cavity or when the pleural space contains air. Now, Laennec was originally disposed to lay more stress upon auscultation of the voice than of the breathing, and pectoriloquy having thus been the first-fruits of his great discovery (for he did not introduce the term bronchophony until after the first edition of his work), it is not surprising, as Dr. Gee remarks, that he always clung to this sign with peculiar affection. It accordingly became his object to define it in such a way that it should become an unerring indication of a vomica. For this purpose he added to the characters of what he termed perfect pectoriloquy that of being limited to a very small part of the chest. But subsequent experience has shown that, like all other "hollow" sounds, the most typical pectoriloquy is capable in some very exceptional instances of being generated when there is no cavity of abnormal size within the thorax. Thus pectoriloquy no longer carries the exaggerated importance which Laennec endeavored to attach to it. And at least one modern writer, Guttmann, omits it altogether, and includes all degrees of increased vocal resonance under the term bronchophony.

On the other hand, an attempt has of late been made, while retaining the use of the term pectoriloquy, to assign to it a new meaning. It has long been known that under certain circumstances an increase in the vocal resonance is discovered more readily when the patient whispers than when he speaks aloud, and the term "whispering bronchophony" has been employed by Dr. Gee and by others to indicate that the sign is elicited in this way. But Dr. Bristowe proposes that in future the transmission of the whisper should be regarded as the special characteristic of pectoriloquy. By bronchophony he would understand that tones generated in the larynx—by pectoriloquy that the articulate sounds formed in the cavity of the mouth —are conveyed downward to the stethoscope with abnormal distinctness. According to this way of using the terms, when the patient speaks aloud, and when the words he utters are clearly perceived by the auscul-

tator, both bronchophony and pectoriloquy are heard at the same time. But it seems to me that the adoption of Dr. Bristowe's proposal is strongly to be deprecated, notwithstanding that it has the advantage of introducing a real distinction into the meanings of the two terms in question, whereas, hitherto, the difference has been only one of degree. It would render useless, except to those who kept themselves well informed as to the change in the sense of the word pectoriloquy, all the literature of the subject during the last sixty years. And, after all, there is no suggestion that transmission downward of the whisper enables any conclusion as to the state of the lung to be drawn which cannot be drawn from ordinary bronchophony. The practical importance of whispered bronchophony lies solely in the fact that its presence is sometimes easily recognized when there is difficulty in detecting an increased resonance of the ordinary voice.

With regard to bronchophony and pectoriloquy, discussions have arisen as to *why* they should be heard when the lung is solid or when it is compressed rather than when it retains its spongy structure. But the answer to this question involves also the explanation of the occurrence of bronchial breathing under similar circumstances, of the peculiarly bright and clear quality of râles, and of the loud transmission of the cardiac sounds to the most distant parts of the chest. Now, Laennec was content to assume that spongy lung substance was a bad conductor of sound. Skoda, however, as the result of direct experiments upon the dead tissues, declared the conductivity of hepatized lung to be actually less than that of the healthy organ; but he could not reproduce the condition under which auscultation is practiced during life, and it is certainly very difficult to believe that a homogeneous material should not convey sound better than one which is full of spaces containing air. Thus almost all recent writers reject Skoda's conclusion. But even if we admit that bronchial breathing and bronchophony are due merely to increased conduction downward of sounds produced above, we have still to account for the hollow modifications of the breath sound and for pectoriloquy. Now, Skoda maintained that *consonance* was the cause of all these phenomena. In acoustics, however, consonance has been understood to mean a power of vibrating in unison with some particular tone, or of producing sounds in harmonic relation to it. And it has always appeared to me that in his work on the subject Skoda does not really limit his application of the term to what is known as consonance in a strict sense. He makes allusions both to the effect of the sounding board of a guitar or violin, and to the augmentation of the sound produced by a tuning fork when it is placed upon a table. But each of them is an example of what writers on acoustics have termed "resonance" rather than "consonance." I observe, however, that in Tyndall's "Lectures on Sound" nothing is said about consonance, everything being included under resonance. Probably, therefore, there is little or no risk of error resulting from our continuing to speak of "consonating" sounds in auscultation, and we need not intend to imply that we adopt a definite acoustical theory as to their origin. It may be sufficient if we suppose that either bronchial tubes surrounded by solidified lung tissue, or the walls of a cavity, are capable, with the air which they contain, of reverberating sounds conveyed to them from above, so that they are subsequently transmitted to the ear of the auscultator with great loudness, or more or less altered in character besides.

It is necessary to make special mention of one modification of vocal resonance, that which from the time of Laennec has been known as *ægophony*, on account of its resemblance to the bleating of a goat. Its characters, and the conditions under which it occurs, will be discussed elsewhere when I am describing pleurisy; but I must so far anticipate what will then be stated as to remark that the presence of a moderate quantity of fluid effusion, forming

a somewhat thin layer and separating the lung from the chest wall, is believed to be almost essential to the production of this sign. Dr. Stone has recently given a very ingenious explanation of ægophony. In a course of experiments with a pitch pipe placed between the lips of various patients and made to utter a musical note by their drawing a deep inspiration, Dr. Stone found that when the spoken voice, or even the whisper, yielded marked ægophony to a stethoscope placed over the affected part of the chest, there was no transmission of the sound of the pitch pipe. So, also, if the patient could be made to sing or to intone a good musical note, no ægophony was to be heard. And among spoken words a difference was found to exist as to the degree in which they gave rise to this modification of vocal resonance according to the vowel sounds contained in them. The French A yielded hardly any ægophony; it was more marked with the E, still more so with the I, and most of all with U. Now, as is well known, Helmholtz showed that the different vowel sounds are formed by the addition of certain harmonic overtones in varying degrees of intensity to a fundamental tone which may be the same for all of them. And, according to Dr. Stone, the cause of ægophony is that the fundamental tone is intercepted in its passage through a layer of pleural exudation, while the overtones are allowed to pass, and, being heard by themselves, give the peculiar character to the sound. In some further experiments he succeeded in imitating ægophony. This was effected by transmitting the voice through a wide india-rubber tube, over which was placed a bladder containing water. When a stethoscope was applied to the upper surface of the bladder, an ægophonic twang became exceedingly distinct.

Dr. Stone's theory of ægophony seems to me to be fully established. And it is of the more interest because it brings into complete accord with the auscultatory phenomenon another physical sign, which (so far as I am aware) had never been thought of in connection with it, but which has long been known to be one of the chief indications of pleuritic effusion; this is called "loss of tactile vibration" or of "*vocal fremitus*." When the hand is placed upon the bare chest of a healthy man, a tremulous sensation is felt, especially if the voice is low pitched; in a woman or a child it is often not to be perceived. On the right side of the back it is almost always more distinct than on the left side. Now, the presence of fluid in the pleura, even in moderate quantity, invariably annuls this sensation, whereas it is not interfered with by hepatization of the lung, the other signs of which resemble those of effusion so closely. The practical importance of loss of tactile vibration seems to have been first pointed out in the "*Journal Hebdomadaire*" for 1829 by a French physician, Reynaud, who also discovered pleuritic friction sound. Its occurrence when the pleura contains fluid affords further confirmation of Dr. Stone's view with regard to ægophony; for the fundamental tone, which, according to him, is intercepted, is the one which would be felt under normal circumstances, whereas the overtones consist of waves too rapid to be perceptible to the touch. I am not sure, however, that loss of tactile vibration is, in all cases, to be detected whenever the voice has an ægophonic character. To make out the former sign satisfactorily, one must lay the hand on a broad surface; whereas the latter is often limited to a very small area. Commonly, tactile vibration is often absent when no ægophony can be heard. This occurs whenever the quantity of fluid is large, so that it cuts off the harmonic overtones, as well as the fundamental tone, of the voice.

INSPECTION.—Beside percussion and auscultation, physical examination of the chest includes an inspection of its shape and also of its movements. A rough estimate with regard to these points is, indeed, commonly made when one

first glances at the patient after he has stripped. But accurate observations are almost always postponed until after one has listened to the breathing over different parts of the lungs. It is not necessary for me in this place to go into any details with regard to the varieties of shape that the chest may present under different conditions. But I may remark that bulging or flattening of one of the infraclavicular regions is more easily detected by standing behind the patient while he is sitting, so as so look downward over his shoulders. A general enlargement of one side is often easily appreciable by the eye. But one must make quite sure that the patient is sitting or standing perfectly upright, especially if the case is that of a child or of a young woman, with a thin, flexible spine. In infants, Dr. Gee remarks, that it is a good plan to grasp the chest with the two hands, placing the thumbs tip to tip upon one of the vertebræ. In adults a measuring tape is often used. But, as Dr. Gee remarks, circumferential measurements are apt to be fallacious, because considerable increase in the sectional area of one side of the chest may leave the length of the periphery unaltered, by "the passage of the elliptical form into the circular." It is this which renders his "*cyrtometer*" so useful, an instrument by which the outline of the chest in any horizontal plane can be accurately determined. It is made of two long pieces of very narrow metal gas tubing, of an eighth of an inch in diameter, which are fastened together by a short piece of caoutchouc tube, slipped over their ends. The central caoutchouc piece is placed over the spinous process of a vertebra, and the hollow metal rods are then carefully bent round the patient's body so as to meet over the sternum. It is now easy to remove them without altering their shape; and by laying them upon a sheet of paper one can obtain an accurate tracing, which shows exactly the configuration of the two sides of the chest, and enables them to be compared. One must not forget that the half circumference of the chest on the right side is in many healthy persons greater than that on the left side, the difference being sometimes as much as an inch.

Various instruments, called *stethometers*, for the measurement of the movements of the chest have been devised by physicians, the latest being one by Dr. Arthur Ransome, of which a description may be found in the "*Med.-Chir. Transactions*" for 1873. But although they have yielded information as to the exact degree of impairment of mobility of different parts of the chest wall in various diseases, I am not aware that any one of them has been employed in actual practice by other observers than their inventors; the reason being that they are troublesome to use, and that they bring to light no facts that may not be ascertained without them.

Nor does it appear that in clinical practice any results worth speaking of can be attained by the use of an instrument invented by Dr. John Hutchinson many years ago for the purpose of measuring the amount of air that can be expelled from the chest by the fullest possible expiration. This instrument, which he called the *spirometer*, may, perhaps, be of value in the examination of recruits for the army, or of "lives" for insurance, by suggesting doubts as to the advisability of accepting such candidates as, for their height, fall very short of the standard. But there are great practical difficulties in obtaining correct results; very few persons succeed in "blowing" their full amount of air into the instrument until they have had some practice.

BRONCHITIS.

GENERAL SYMPTOMS: COUGH, DYSPNŒA, PAIN—PHYSICAL SIGNS: RHONCHUS, SIBILUS, RALES—MORBID ANATOMY.

Acute Bronchitis—CAPILLARY FORM—PROGNOSIS—PULMONARY COLLAPSE—BRONCHO-PNEUMONIA.

Chronic Bronchitis—VARIETIES—EMPHYSEMA—BRONCHIECTASIS—FETID BRONCHITIS—ÆTIOLOGY AND TREATMENT OF BRONCHITIS GENERALLY.

The disease which is termed bronchitis is so common, and it is now so familiar, that probably almost every one is at first surprised to learn that medical literature contains no allusion to it before the publication of works by Peter Frank in Germany, and by Badham in England, in the years 1812 and 1814. Up to that time it had been known as "catarrh," except that some of the more severe forms of it were often designated by the cumbrous name of "peripneumonia notha," invented by Sydenham. It is generally understood to include inflammations of all parts of the air passages below the larynx. When the windpipe is very obviously affected, tracheitis is sometimes said to be present in addition; but, as might be expected, the artificial boundary lines recognized by the anatomist find no application in clinical practice; and in a very large number of cases the morbid action reaches to a greater or less extent above the bifurcation of the trachea, without its being considered necessary to speak of the disease as anything more than bronchitis. On the other hand, it is usual to exclude altogether such forms of inflammation of the trachea and bronchi as are attended with plastic exudation, or lead to deep ulceration and cicatrization. These are described in separate chapters of this work (see pp. 847 and 852).

There are few other affections of which there are so many varieties as of bronchitis; and these differ, both in symptoms and in course, to the most extreme degree. It therefore seems to be hopeless to attempt to describe them in common. I shall first enumerate such of the symptoms and physical signs as belong alike to them all, and afterward I shall give separate accounts of certain of the more important of them.

Foremost among the symptoms, then, is *cough*. This is never absent, and it is often exceedingly severe, and of a very loud, barking, or ringing character. It may consist of isolated explosions, succeeding one another more or less regularly, and sometimes with extreme frequency. Or it may occur in paroxysms, which sometimes end in retching or actual vomiting. It may be worse when the patient lies down, or it may come on especially when he first gets up in the morning, being excited by an accumulation of mucus or pus in the air passages during the night. Sometimes the irritation which sets it up is definitely referred to some one spot along the course of the trachea, which is felt to be raw or tender; sometimes there is a vague tickling sensation, which cannot be localized. The characters of the sputum, if the cough leads to expectoration, differ so widely in different forms of bronchitis that it is useless now to allude to them.

Another symptom which belongs to all but mild cases of bronchitis is

dyspnœa. It depends, in the main, upon the mechanical obstruction to the entrance of air into the lungs, which we shall presently see to be a natural result of the pathological changes that occur in the mucous membrane of the finer tubes. Thus, it is especially associated with inflammation of the lower air passages, as contrasted with that of the trachea and main bronchi, the calibre of which is, perhaps, never sufficiently diminished, at least in the kind of inflammation with which we are now concerned, to interfere with the access of air to the pulmonary tissue. A curious circumstance, to which Riegel seems to have first drawn attention, is that in all affections of the bronchioles the dyspnœa is expiratory rather than inspiratory. Sometimes the act of inspiration is quite short and easy, while that of expiration is very prolonged and difficult; sometimes they are both alike embarrassed; it is, I believe, never the case that the inspiration is alone obstructed, as often occurs in affections of the larynx of the trachea. The explanation of this special tendency for the expiration to be interfered with, when the smaller air tubes are inflamed, is by no means obvious. Another peculiarity of the breathing, which may often be noticed in children, is that each expiration is instantly followed by an inspiration, the pause in the act of breathing taking place at the end of each inspiration and not at the end of the expiration, as it does normally. In severe cases there is often complete *orthopnœa*, the patient having to be propped up with pillows all night. The reason why it increases his distress to lie down seems to be partly that the weight of the abdominal viscera is thrown upon the diaphragm and renders its descent less easy, partly that the pectorals and other accessory muscles of respiration cannot be so easily brought into play as when he is upright.

When dyspnœa is at all considerable, there is almost always more or less lividity or *cyanosis*. This shows itself upon the face and hands. In extreme cases, the face becomes very turgid, flushed and bloated. The veins of the neck are dilated and throb. The superficial veins of the body generally are fuller than natural.

Pain is by no means constantly present in bronchitis. Many patients, however, complain of a sore feeling behind the sternum, or in the upper part of the chest on either side. Or, again, the harassing cough may give rise to a more or less severe myalgia in some part or other of the thoracic walls. Not infrequently such a pain, muscular in its origin, is felt at the epigastrium. But another cause of pain in this position is fullness of the liver, resulting from obstruction to the venous circulation.

The *physical signs* of bronchitis are less numerous than those of almost any other disease of the respiratory organs. They are mainly auscultatory, the percussion sound being quite unaltered, unless the case is complicated with pleural effusion or with some affection of the pulmonary tissue, such as emphysema, or collapse, or broncho-pneumonia.

With the stethoscope it is found that the vesicular murmur is more or less altered in character, or that it is replaced by, or has added to it, certain adventitious sounds. Sometimes the change in the vesicular murmur is that it is faint and indistinct; it may even be temporarily absent over a part of the lung, in consequence of the corresponding bronchial tube being plugged by mucus. In this case one can bring it back by making the patient cough vigorously two or three times. Or the vesicular murmur may be rough and harsh in quality, and the expiration may be accompanied by a very similar sound. There is, then, sometimes considerable difficulty in drawing a distinction from bronchial breathing, which, however, has generally a more blowing character, besides being strictly limited to certain parts of the chest, whereas in bronchitis the sound is heard over both lungs alike and very widely. It is a point which cannot be too strongly impressed on those who are learning the use of the stethoscope, that neither bronchial

breathing nor any of its modifications occurs in bronchitis except when some complication is present. Nor, again, is the transmission of the voice in any way altered.

The adventitious sounds which occur in bronchitis are those that have been already enumerated at p. 872, under the names of "rhonchus," "sibilus," and "râles." *Rhonchus* (or "sonorous rhonchus") is a loud snoring or cooing noise, often audible by the patient himself and by those about him, and due to vibrations that can be felt by the hand placed upon the surface of his chest. It is formed in the larger tubes, and in bronchitis its cause is the presence of a mass of viscid mucus partly obstructing the entrance of air, and producing a *veine fluide*. The proof of this is that it can very generally be made to disappear, at least for a time, by the patient's coughing once or twice, and, indeed, it comes and goes of its own accord, being heard first in one part of the chest and then in another, as mucus happens to accumulate in different branches of the bronchial tree.

Sibilus (or "sibilant rhonchus," as some prefer to term it) is a high-pitched, whistling sound. It is formed in the bronchioles, and is, therefore, of much graver import than rhonchus, inflammation of the smaller air passages being far more dangerous than of the larger. It seems to be due to the narrowing of the calibre of the affected tubes which results from swelling of their lining membrane. Consequently, it cannot be got rid of by coughing, and it usually remains in the same spot for hours or days together.

The *râles* which accompany bronchitis may be of every variety of size. They are not of "consonating" quality, inasmuch as the lung tissue round the tubes in which they are formed still remains more or less spongy. As a rule, this distinction is very obvious. But I do remember one case in which the autopsy showed that no disease beyond bronchitis was present, but in which I had thought that there must be either phthisis or pneumonia, from the character of the râles. In general, if râles are not universally distributed through the lungs, they are most marked over the lower lobes and behind rather than in front. Signs that might suggest the presence of bronchitis in the upper lobes only—especially if limited to the upper lobe on one side—should always arouse a strong suspicion of phthisis. A point which is worthy of mention is that it is sometimes impossible to detect any râles in cases in which the profuse expectoration would certainly have led one to expect to find them.

Diagnosis.—The symptoms and physical signs above enumerated are not sufficient in themselves to justify a diagnosis of bronchitis. It is necessary to add to them certain negative points by which the presence of other affections of the air passages or of the lungs is excluded. And in all cases of which the clinical history is such as to render it possible that the pulmonary parenchyma may contain scattered tubercles, whether of acute or of chronic development, great caution should be exercised in forming an opinion. Sometimes, but very rarely, the occurrence of secondary nodules of a malignant new growth in the lungs offers another source of error. As a rule, however, the difficulty is not so much in saying that bronchitis is present as in determining whether it is the principal affection from which the patient is suffering or only a complication. It is, in fact, exceedingly apt to arise in the course of a great variety of diseases, among which may be mentioned the exanthemata, enteric fever, rickets, obstructive lesions of the heart, and Bright's disease of the kidneys.

Morbid Anatomy.—In all mucous membranes, and in the skin, the morbid appearances produced by inflammation are far less conspicuous after death than during life; and the reason why such should be preëminently the case with the bronchial mucous membrane is afforded by the abundance of elastic

fibres in its structure. Indeed, it is sometimes by no means easy to determine the presence of bronchitis at an autopsy, even when it has been the principal disease from which the patient suffered. A very good method of detecting puriform secretion in the finer tubes is to slice off the extreme edge of the lung, and then to press the tissue upward toward the cut surface, where a yellow bead appears at each little orifice. But in many cases there is extreme redness and swelling of the mucous membrane, which may have a velvety appearance, so that it resembles the petal of a damask rose. And every part of the air passages, up to the trachea, may be almost full of a yellow or brown opaque fluid.

The histological changes in bronchitis have recently been studied by Socoleff ("*Virchow's Archiv*," vol. lxix), and by Dr. Hamilton ("*Practitioner*," 1879). The former set up inflammation in dogs and in rabbits by the insufflation of bichromate of potass or of a weak solution of chromic acid; the latter based his investigations upon cases that presented themselves in the post-mortem room of the Edinburgh Royal Infirmary. Both observers are agreed that a very early change is the detachment of the ciliated epithelium, which seems to be thrown off in flakes, and which remains absent during the whole course of the disease, being regenerated when recovery takes place. In a young man who died of opium poisoning, in from ten to sixteen hours, the ciliated cells were already to a great extent shed, although Dr. Hamilton speaks of the morbid process in that case as having been rather acute congestion than actual inflammation. He says that the cells themselves undergo fatty degeneration, which probably destroys many of them. Probably some are expectorated; others are inhaled into the finer air tubes, where they may be seen lying in large detached masses among the other catarrhal products. There is an obvious analogy between this exfoliation of the *formed layer* of the bronchial epithelium and the separation of the cuticle in the roofs of blisters or vesicles when inflammation affects the skin. During the further progress of the attack, the basement membrane is covered only by a layer of flat cells, from which there project here and there pyriform or oval cells, of transitional character, which are covered by a more or less abundant mass of leucocytes, embedded in a mucoid fluid. A point on which Dr. Hamilton lays great stress is that the basement membrane itself becomes greatly thickened and swollen, apparently as the result of œdema. Both he and Socoleff are convinced that the leucocytes which appear in such large numbers upon the free surface of the mucous membrane are not derived by emigration from the blood vessels, but are formed by germination from the flat cells that lie immediately in contact with the basement membrane. Socoleff's chief reason for maintaining this opinion was that in animals killed twenty-four hours after the commencement of the morbid process he found leucocytes on the free surface of the mucous membrane, although its substance was at that time entirely free from them. Dr. Hamilton insists especially on the difficulty which blood leucocytes would have in traversing the thickened basement membrane, and on the fact that in his preparations he could discover no indication that this was taking place. But it is, perhaps, worthy of notice that Socoleff himself figures ciliated epithelial cells having in their interior red blood discs, which must have made their way through. And I must confess that I should hesitate to accept any observations upon deep-seated tissues as overthrowing the results of investigations made upon the cornea and other superficial structures, for the special purpose of determining the nature of the inflammatory process, as detailed in pp. 66, *et seq.*

In all but very early and very slight cases of bronchitis the mucous or submucous tissues are, in their whole substance, more or less thickly filled with leucocytes, which are collected in lines along the lymph spaces between

the fibrous bundles and around the vessels. Dr. Hamilton is satisfied that these also are in great part derived by germination from the flat endothelial cells of the lymph spaces or from connective-tissue corpuscles. Another very important change occurs in the mucous glands. They become swollen, so as to be sometimes as large as hemp seeds, according to Riegel. Their epithelium undergoes very active proliferation, and the newly-formed cells become distended with a mucin-holding fluid, and appear to be the source of the mucus that often forms so large a part of the expectoration. This mucus, however, becomes mixed with serous exudation from the walls of the bronchial tubes themselves, and with leucocytes and cells of "transitional" form, as has already been stated.

When bronchitis has existed for a great length of time before death the changes found post-mortem are somewhat different. The mucous membrane is often pale and gray, with but few vessels visible. In many cases it presents a number of delicate longitudional ridges, which Rindfleisch has shown to consist of an overgrowth of connective tissue, containing very numerous cells, and bundles of elastic fibres running in various directions. According to Dr. Hamilton the muscular coat is sometimes found to be hypertrophied, sometimes atrophied. The cartilages shrink and disappear, the change in them being exactly the same which occurs in cartilage tissue under so many other conditions, namely, the absorption of the matrix from the periphery inward, with the formation of "medullary spaces" filled with leucocytes. In many cases the mucous glands also are destroyed. At an earlier period their orifices are widely dilated, giving the mucous membrane a finely-pitted appearance when looked at with a good light. And sometimes they become inflamed, forming minute funnel-shaped ulcers.

Of the various forms of bronchitis some run an *acute*, others a *chronic* course.

Acute Bronchitis.—This often affects mainly the larger air passages, so that it may fairly be called a "tracheo-bronchitis;" and in such cases the inflammation is sometimes derived by extension from the nose and throat. The cough may then be exceedingly distressing, being especially violent when the patient attempts to lie down. He may complain greatly of a sore sensation along the sternum; and pressure upon the trachea may be painful and may at once excite cough. This form of the disease, however, is not dangerous.

Very different is the course of acute bronchitis when it attacks the bronchioles throughout the lungs, for this may be one of the most rapidly fatal of all diseases. It is sometimes distinguished as "Capillary Bronchitis," and another name for it used to be "Suffocative Catarrh." It usually sets in with a sensation of chilliness, or even with a rigor; according to Niemeyer its onset differs from that of pneumonia in the circumstance that repeated rigors often occur, whereas in the latter disease there is seldom more than one. The degree of pyrexia varies widely; the temperature may range up to 104°, especially in children; more often it is at a lower level; it does not run any typical course. The head and the upper part of the body often become covered with sweat. The hands and the surface generally feel hot, the face is more or less deeply flushed. The pulse is very rapid, sometimes so rapid that it cannot be counted. It is often tense and full, this being doubtless the result of the obstruction offered by the systemic capillaries to the passage of blood containing an undue quantity of carbonic acid.

But the most prominent symptom of this form of bronchitis is the dyspnœa. The patient sits up, with chest heaving and with nostrils quivering, unable to utter more than two or three words at a time, using his

shoulders and arms in violent efforts to breathe. On carefully inspecting the thoracic movements one finds that obviously there is a great obstacle to the entrance of air into the lungs. The epigastric and the hypochondriac regions of the abdomen recede at every inspiration; in children all the lower ribs and the lower part of the sternum may be forcibly sucked in. The supraclavicular and the suprasternal spaces also recede, but, on the other hand, as Seitz has pointed out, the upper ribs often remain almost motionless in a position which is that of a forced inspiration, giving to the corresponding part of the chest a vaulted shape.

The cough of capillary bronchitis is often exceedingly harassing. At first it is usually "dry," there being nothing in the air passages to be expectorated. Afterward it is accompanied with a more or less abundant sputum. The bronchial mucous membrane, indeed, when it is inflamed, goes through stages very similar to those that may be observed in the nose under like circumstances. It begins by being swollen and dry, then it pours out a transparent mucoid fluid; after a time this becomes muco-purulent, and finally almost pure pus. The dry stage sometimes lasts several days, or even throughout the whole of the duration of the disease. Thus, Dr. Latham, in his "Lectures on Subjects connected with Clinical Medicine," speaks of the case of a boy, seven or eight years old, who for six days remained in a condition of extreme suffering, with a shrill sibilus audible all over his chest, and then gradually recovered without expectorating anything. A point, however, which must not be forgotten is that infants and growing children commonly swallow whatever they cough out of the air passages into the mouth. In older patients, when sputum first appears, it may be very scanty and dislodged with great difficulty, the patient, perhaps, coughing a number of times in rapid succession, and until he is purple in the face, before he can get relief by bringing up a little translucent pellet of mucus. But in other cases the spitting jar becomes filled in a few hours with a considerable quantity of a grayish-white, glairy liquid, which has numerous air bubbles entangled in it, and is so viscid that, if there is not too much of it, the vessel may be turned bottom upward without any escaping, with so great tenacity does it cling to the bottom and to the sides. Under the microscope this kind of sputum is found to contain remarkably few formed elements. I have already remarked that in bronchitis the tubes cease for the time to be lined with a columnar epithelium. It is, therefore, probable that when a few cells of that type are seen in the matters expectorated (except at the very commencement of the disease) they have been derived from healthy and not from inflamed parts of the air passages, just as flat epithelial cells are often seen which come from the throat or the mouth. Cells of transitional form, however, and mucous corpuscles are present in small numbers, and as the case advances pus cells abound more and more until the sputum becomes quite opaque and of a greenish-yellow color. It may now come up freely, and with scarcely any effort. Under these circumstances the cough is said to be "loose."

In many cases, after acute bronchitis has lasted for some time, the quantity of expectoration begins gradually to diminish from day to day; the other symptoms become less and less severe, and presently the patient recovers entirely from his attack. But in other cases the inflammatory exudation accumulates in such large amount as to threaten death by suffocation. Râles then become audible all over the chest, and are so loud that no trace of vesicular murmur can be anywhere detected. Indeed, they are often heard at a distance from the patient, and by those about him as well as by the physician. Still more important as a warning of danger is the supervention of cyanosis; the flushed cheeks, the lips, the hands assume

at first a faint lilac, but ultimately a leaden color. Another very grave symptom is the failure of effort on the part of the respiratory muscles; the breathing gradually becomes more and more shallow, until at last it may be represented only by a slight flickering movement of a few of the ribs, or by a faint jerking contraction of the diaphragm. With this, too, the patient ceases to be conscious of the necessity for activity on the part of the breathing apparatus. Instead of remaining upright he sinks down in bed, with his head in any position in which it may happen to be placed. His mind may wander for a time and then he becomes unconscious. Sometimes death is preceded by one or more convulsive seizures.

As a rule, if acute bronchitis is to end fatally it does so in the course of the first fortnight. There are even some cases in which the patient succumbs within twenty-four or forty-eight hours, but it not infrequently happens that when the disease has apparently been subsiding quite favorably, a relapse occurs which dashes all hopes to the ground. It need not be said that the patient's muscular strength is one of the most important points that one has to take into account in attempting to form an estimate as to the probable issue of a case of acute bronchitis. In very old persons the prognosis is always doubtful; it is so, likewise, in those who are very fat, or who are already weakened by previous illness, or who have progressive muscular atrophy affecting the shoulder or trunk muscles, or any considerable deformity of the spine or of the chest. In infants, on the other hand, the chance of recovery is better in proportion as the age is older; the gravity of the disease is greatly increased by the presence of rickets. A good deal of caution, however, is required in giving an unfavorable prognosis as regards children. It is surprising how rapid may be both the pulse and the breathing, for two or three days together, in cases that ultimately end in recovery.

In many instances of acute bronchitis the digestive organs are disturbed in a manner that is not readily accounted for, the degree of pyrexia affording no adequate explanation of it. The tongue is often coated with a whitish-yellow fur, of surprising thickness. There may be nausea and vomiting. The bowels may be obstinately constipated. In children it is sometimes difficult to say whether the chest symptoms or those belonging to the abdomen constitute the more essential part of the case.

But the most important complications of acute bronchitis when it attacks the finer tubes are those that concern the substance of the lungs themselves. One of them is known as *collapse* of the pulmonary tissue; another is an inflammatory affection, for which the best name appears to be *broncho-pneumonia.*

Pulmonary Collapse is identical with a state of lung which is seen in infants as the result of imperfect respiration, and which is nothing else than a persistence of the fœtal condition of the tissue. That, however, is properly termed Atelectasis (ἀτελής = imperfect, ἔκτασις = expansion) or Apneumatosis. It may affect the whole of both lungs if the child has never breathed at all, or parts of the lungs (especially the anterior edges) if it has breathed incompletely, from having been prematurely born, or being weakly, or having its air passages obstructed by mucus.

A German writer, Jörg, is generally credited with having been the first to point out, in the year 1832, the real nature of the atelectasis which had before been supposed to be a congenital pneumonia. And it is commonly said that collapse also was up to that time confounded with red hepatization. But Bright, in 1828, speaking of the morbid appearance found in the lungs of two children who had died of whooping-cough, showed that he clearly recognized the difference, and Dr. Alderson, also, is

said to have drawn attention to it. The distinctions between these two affections are unmistakable. A collapsed part of the lung is, indeed, reddened, and the color of its cut surface is reddish brown, or, when covered with pleura, reddish purple or violet. A section of it, however, looks perfectly dry, smooth, and homogeneous; it has not the dull, lustreless appearance of hepatization. Moreover, its surface lies below the level of the adjacent air-containing parts of the lung: if it reaches the free edge of the organ it forms a notch there. Lastly, inflation from the bronchus will usually restore to it its normal appearance. Sometimes, indeed, collapsed pulmonary tissue is at the same time œdematous, and then its characters are less marked, its cut surface being moist and emitting serum when gently squeezed.

Another airless state of lung which has received a separate name is the "carnification" caused by compression by pleural effusion; what characterizes it is that the tissue is bloodless as well as airless, and the color is slaty or mouse colored instead of being reddish brown.

The way in which collapse arises was well illustrated in a case in which I made an autopsy in 1874. A child, aged two years and two months, died four days after the performance of tracheotomy for a chronic laryngeal affection. Upon the under surface of the left lung there was a narrow red line of collapsed lung tissue. This had running through it a tube which (like all the other tubes in the same part of the organ) happened to have become dilated as the result of the chronic obstruction to the child's breathing. That tube was plugged at its upper part by a piece of sponge, about a quarter of an inch long, which had evidently fallen into the trachea at the time of the operation. The limitation of the collapse to the part of the lung served by the obstructed tube was perfect. As a rule, collapse is secondary to closure of the corresponding tube by viscid mucus or muco-pus. How this brings about the affection has only lately been well understood. Gairdner, in 1850, suggested that the plug acted like a ball valve, allowing air to escape during expiration, but preventing its entrance during inspiration. But I have always thought the explanation unsatisfactory and incapable of accounting for the complete disappearance of the air, inasmuch as the elastic force of the pulmonary tissue and that of the confined air must soon become inadequate to raise the valve. And Lichtheim, of Berne, in an important research recorded in the "*Arch. f. exp. Path.*" for 1879, has recently shown that in rabbits collapse very quickly (within twenty-four hours) follows the plugging of a bronchus by a piece of laminaria, which becomes swollen, so as to prevent all passage of air in either direction. It is, therefore, evident that the affection must depend mainly, if not entirely, upon absorption of the air by the blood which circulates in the walls of the alveoli; and Lichtheim gives reasons for believing that the several gaseous constituents of the atmosphere are taken up with different degrees of rapidity, the carbonic acid and the oxygen first, and afterward the nitrogen. A further result of his investigations is the proof that the elasticity of the pulmonary tissue is not exhausted until it has become completely devoid of air. For without the aid of lung elasticity, absorption by the blood must cease before collapse would become complete.

The state in which a lung is found when there has been slight narrowing of the space in which it lies (whether from pleural effusion, or enlargement of the heart, or pushing up of the diaphragm) is inexplicable unless it be admitted, as a general principle: that whenever even a small part of the organ fails to be acted on by the forces which are concerned in inspiration, its elasticity brings about a total collapse of its substance, notwithstanding that the tubes which serve it may be patent. This, I must admit, is a hard doctrine to accept; but I believe that there is no doubt about its truth. Now, in bronchitis, at

least in children, it often happens that large portions of the lower lobes of the lungs are unacted upon by inspiratory forces, for (as we have seen) the lower ribs and even the sternum are commonly drawn inward, instead of rising, during the act of breathing. And the same thing occurs in croup and in other diseases attended with laryngeal obstruction. I am therefore disposed to think that collapse of the lower and anterior edges of the lungs, which is so often seen under such circumstances, is generally, if not always, due directly to the cessation of inspiratory traction upon those parts of the organs. And Bartels, as far back as 1860, showed by dissection that it is in fact often impossible to demonstrate any plugging of the tubes passing to collapsed areas of pulmonary tissue. So again, in enteric fever, we may refer the collapse of the bases of the lungs, which is so commonly seen in the bodies of those who have died of that disease, to the shallowness and imperfection of the respiratory movements. Even when collapse depends upon obstruction of tubes, an important factor in its production is a deficiency of power in the muscles of the thorax. For, with strong muscles, there is not only the chance that a fit of coughing may expel a plug of mucus, but also that a vigorous inspiratory effort may succeed in drawing air into the tissue in spite of it. Accordingly, I believe that in adults collapse scarcely ever occurs as the effect of primary bronchitis. Even in children its development is greatly favored by a rachitic state of the ribs, and perhaps, also, by the muscular weakness resulting from measles or any other acute disease. And the younger the child, the more likely is it to show collapse of the lungs under a bronchial attack.

The view that inspiratory retraction of the lower part of the chest is generally the cause of collapse of the lungs, when the two things are associated together, is not incompatible with the opinion that an extensive collapse due to obstruction of the corresponding bronchial tubes, may sometimes in its turn lead to a falling in of the thoracic walls. Dr. Gee, for example, describes a unilateral shrinking of the chest as resulting in some cases from collapse of the whole of a lung in consequence of plugging of its main bronchus. Generally speaking, however, the space in the pleural cavity vacated by a collapsed portion of pulmonary tissue is filled up by an overdistention of other portions of the organ. And, if an entire lung should become emptied of air, the opposite lung undergoes enlargement and displaces the mediastinum. This condition is sometimes confounded with emphysema, as I shall have to point out further on.

Some writers believe that pneumonia is very apt to arise in collapsed portions of lung tissue, in which case the anatomical distinctions between collapse and hepatization must necessarily fail. Lichtheim, indeed, found in his experiments that an acute œdema of the affected lung sometimes occurred, so that, although airless, it was bulky, soft, and moist. The retention of secretion in obstructed tubes is suggested by Jürgensen as a probable cause of inflammatory irritation. And Lichtheim, in some instances in which an animal survived for a considerable time after obstruction of the bronchus, describes the lung as looking almost like a sacculated kidney full of dilated channels distended with pus. But, as a rule, I think that pulmonary tissue collapsed as the result of disease in the human subject remains uninflamed. If there are patches of broncho-pneumonia in the same organ, their presence is merely a coincidence.

So far as I know, collapse, whether arising from bronchitis or from laryngeal obstruction, is always a temporary condition, the affected parts of the lung again receiving air, if recovery takes place from the primary disease. I have never seen in the post-mortem room any appearances that appeared to be traceable to the occurrence of collapse at a former period.

With the exception of the collapse which occurs at the extreme bases of

the lungs as the result of inspiratory retraction of the chest walls, this condition, when secondary to bronchitis, seldom affects more than small portions of each organ, lobules or groups of lobules scattered here and there, some on the surface, others in the interior. Hence it does not often give rise to very definite physical signs; though if many patches should exist in close proximity to one another, it is possible that there may be more or less dullness on percussion, a deficiency of vesicular murmur, and even perhaps bronchial breathing. As regards symptoms, all that can be said is that collapse aggravates the dyspnœa already produced by the bronchial affection.

Broncho-Pneumonia (catarrhal pneumonia, lobular pneumonia).—In children acute bronchitis affecting the finer bronchial tubes is often accompanied by inflammation of the pulmonary alveoli. And even in adults the same thing is sometimes observed, although very rarely. Among children it is especially at an early age that broncho-pneumonia is apt to occur. Ziemssen, out of ninety-eight cases, found that sixty-seven, and Steffen, out of seventy-two cases, found that fifty-two, were in patients under three years old. In very many instances the affection is secondary to some infective disease. Measles and whooping cough furnish by far the largest number of cases of broncho-pneumonia. But it is also sometimes seen after diphtheria, rötheln, smallpox, scarlet fever. And Dr. Wilks, in the "*Guy's Hosp. Reports*" for 1860, pointed out that it is a common cause of death in children (even up to puberty) suffering from severe burns. Jürgensen insists, as other writers had before, on the greatly increased liability to broncho-pneumonia, as a complication of bronchitis (whether after measles or independently of any infective disease) in those who are obliged to breathe impure air, among the poor, in their close, narrow rooms. He is even disposed to attribute the greater frequency of the affection in winter to defect of ventilation rather than directly to inclemency of season. Nor is it unlikely that the existence of rickets may render a child more apt to become affected with broncho-pneumonia if it is attacked with bronchitis.

As to the special causes of the exceptional cases in which a broncho-pneumonia like that of infants occurs in adults, I am not aware that it is at present possible to say anything definite. I have notes of a few cases. One was in a woman, aged thirty, who, having miscarried eight days before, was attacked with shivering and headache, and died after an illness that lasted ten days. Another was in a man, aged twenty-five, of dissipated habits, who was said to have been under a course of mercurial treatment for syphilis, when he got drunk and received a blow on the nose. This was followed by epistaxis, which continued until recourse was had to plugging of the nares; the mucous membrane then suppurated profusely, the discharge being very fetid; and he sank and died at the end of a week. A third case was that of a man, aged twenty-eight, a singer at a music hall. He had a fall from a cart, and this led to an illness which proved fatal in three weeks. At first he tried to go on with his singing, but he was soon obliged to give it up. When admitted, shortly before his death, he was comatose. In this instance some of the patches were sloughing in their centres. But in the reports of the other two cases it is expressly stated that the appearances were identical with those that are commonly seen in children. It seems probable that in the second case the inflammation was set up by the inhalation of fetid pus from the nasal cavities into the air passages. Indeed, I have before me the report of another case in which a similar affection of the lungs was met with as a complication of tubercular meningitis in a woman aged thirty-four, and was attributed to the entrance of food into the bronchial tubes while she was struggling in her delirium.

Breathing irritant gases, especially chlorine in large quantity, sometimes sets up inflammation of the pulmonary tissue, simultaneously with a very intense bronchitis. It is not improbable that the immediate cause of the ordinary broncho-pneumonia of children is often, if not always, the entrance into the alveoli of inflammatory products formed in the finer tubes, as the result of violent inspiratory efforts.

Broncho-pneumonia is also said to be not infrequent in extreme old age, giving the *coup de grace* to those who have long labored under senile bronchial catarrh.

The most obvious indication that pneumonia in a given case has arisen by extension from the bronchial tubes is, its occurring in scattered patches throughout one or both of the lungs. These patches are more or less rounded in form; they are usually of about the size of peas, but sometimes it is said they may be as large as hazel nuts. They are reddish-brown in color, or more or less gray, or even grayish-yellow, according to the stage to which the inflammation has advanced in them. As seen upon the cut surface of the lung they appear slightly raised above the rest of the parenchyma. They have a dull, lustreless appearance, and I agree with those writers who describe them as very often somewhat granular on section, although no doubt the granulations are less obvious than in the red hepatization of ordinary acute pneumonia. The substance of the patches is soft and friable, and when squeezed they emit a more or less opaque fluid. It sometimes happens that they are so closely collected together in some one part of a lung that a whole lobe may seem to be consolidated. Even then, however, the lobular markings are usually very distinct and the cut surface is devoid of that homogeneous, even appearance which characterizes common "lobar" hepatization. Still, I do not think it is advisable to keep up the use of the term "lobular pneumonia" as a synonym for the affection now under consideration. One great objection to the name is that it was formerly applied as well to the scattered masses of infiltration that result from pyæmia.

I must not omit to mention that more or less of pleurisy is very commonly associated with broncho-pneumonia, there being a thin layer of lymph upon the pleural surface, especially over any patches that happen to lie just beneath the serous membrane. Indeed, both at the bedside and in the post-mortem room, I have been much struck with the fact that, altogether apart from the presence of any obvious pneumonia, pleurisy is a far more frequent complication of bronchitis than I should have anticipated. In adults affected with bronchial inflammation a pleuritic friction sound may very often be detected, if it is listened for.

Histologically, the morbid process in broncho-pneumonia is a *catarrhal* inflammation; that is to say, the cells which fill the affected alveoli are many of them epithelial in character, large, of irregular shapes, with bold nuclei. Some writers, therefore, prefer to call the disease "catarrhal pneumonia;" but I think that this is better avoided, because the name is ambiguous, inasmuch as a catarrhal pneumonia may occur under many other circumstances; we shall, for instance, have to discuss elsewhere the relations of such an affection to acute tuberculosis and to phthisis.

Moreover, in some cases the inflammation appears to spread to the connective tissue by which the pulmonary lobules are united together. Jürgensen speaks of "thick, whitish-gray bands, which are seen crossing one another upon the cut surface of the organ." In 1878 I met with a well-marked instance of this in a child, aged three, who died after an illness of five weeks' duration, which, perhaps, began in whooping-cough. The left lung contained many patches of broncho-pneumonia. The right lung was almost entirely consolidated. Its tissue, however, felt hard; and the fibrous tissue in it had obviously undergone a great increase. Such cases seem to

me to suggest an explanation of an appearance which one not infrequently sees in making autopsies of persons at all periods of life, namely, a marbling of the texture of a part, or the whole, of a lung by fibrous bands which intersect one another and divide it up into irregular areas. Such an affection is commonly regarded as indicating the commencement of cirrhosis of the organ. But it has not at all the look of a progressive or active lesion; and I think it is far more likely to be a residue of a former attack of inflammation. Ordinary acute pneumonia, so far as I am aware, never gives rise to it.

The *diagnosis* of broncho-pneumonia is usually more or less uncertain; the recognition, that is to say, of its presence, in addition to that of the acute bronchitis which precedes and gives origin to it. Physical examination of the chest often helps but little. If several lobules side by side beneath the pleura are consolidated there may be impairment of resonance (or even dullness) on percussion, bronchial breathing and bronchophony, the latter being especially obvious when the patient, if a child, is crying. The bronchitic crepitations and râles are often clear and "consonating" in quality. Jürgensen lays stress on the occurrence of a crepitating râle rather like that which is heard in ordinary acute pneumonia, but slightly less fine and audible on expiration, as well as on inspiration.

Nor, again, is broncho-pneumonia attended by any marked *symptoms*. Ziemssen has rightly laid stress on the importance of a sudden rise of temperature, as an indication of its supervention in the course of acute bronchitis, if the pyrexia should previously have been moderate. But, as I have already stated, the thermometer may indicate 104°, or even higher, in a child affected with bronchitis, independently of any complication. Jürgensen says that broncho-pnuemonia—as for example, after measles—may be accompanied with a temperature of 105.8° for days together with scarcely any remissions. In fatal cases the temperature sometimes rises before death, reading, perhaps, 107° or 108°; sometimes it falls below normal. The pulse is often extremely rapid, so that at the wrist it cannot be counted. In children a pulse rate of 150 to 200 is by no means incompatible with subsequent recovery; and one must be careful not to allow it to lead one to give an absolutely fatal prognosis. It is surprising, too, how hurried the breathing may be in cases which yet do perfectly well. What is really alarming is that the pulse should be full and "running" in character, from emptiness of the arteries. When the disease ends in recovery the pyrexia and the other symptoms subside gradually; there is never a "critical" fall of temperature. Herpes on the lips seldom accompanies this form of pneumonia. Albuminuria is not infrequent.

As regards what may be termed "chest symptoms," it often happens that with the supervention of broncho-pneumonia the cough from which the patient had been suffering ceases, or becomes less loud and shorter in character. Usually no expectoration makes its appearance. There is generally an extreme restlessness, the patient tossing about in bed, or requiring (if a child) to be taken up by its nurse and shifted in position every few minutes. The breathing is often shallow. The face and the lips are apt to become pale, with perhaps a livid blush upon the cheeks.

One point, of great clinical importance, is that broncho-pneumonia sometimes gives rise to cereberal symptoms of so prominent a character that one may easily suppose the case to be one of tubercular meningitis. Delirium, coma, retraction of the occiput, vomiting, strabismus, convulsions, may all appear in turn. Jürgensen makes the formal statement that "in a child affected with broncho-pneumonia it may be absolutely impossible to say with certainty whether or not tubercles are also present in moderate quantities in the brain." Of course, the question, then, is whether the lung affection also is not really an acute tuberculosis.

Prognosis.—The ordinary duration of broncho-pneumonia is about a fortnight; but sometimes it runs on for three or four weeks, or even longer. It may destroy life very rapidly—within a few days, or even in twenty-four hours. Sometimes the cause of death seems to be marasmus, all acute symptoms having passed off. Jürgensen gives some figures from which it appears that the disease is fatal to from half to two-thirds of those attacked by it. In cases consecutive to measles the risk is said to be smaller than this; and in general, he lays down the rule that the more acute the attack the less the danger. Very young infants are far more likely to die than older children; and the prognosis is also more serious in those who are weakly, rachitic, or very fat.

Chronic Bronchitis, like the *acute* form, varies widely in its degree of importance and of severity in different cases. Some of its mildest forms are seen in children and in young adults, who from time to time have attacks of what is termed "bronchial catarrh," until, as they get older, they perhaps ultimately "grow out" of them. Other cases, chiefly in persons advancing in age, take the form of what is called a "winter cough." Year after year, during the cold season, these people become troubled with a cough, which leaves them entirely in the summer, and which is attended with more or less free expectoration of a muco-purulent material. Sometimes it occurs chiefly in the morning, when they rise from bed; sometimes it goes on at intervals throughout the day; sometimes it is very bad at night, disturbing their rest. For a time there is not the least dyspnœa. Gradually, however, they find that in muscular exertion, as in walking up hill, or in mounting stairs quickly, the breath becomes short and hurried. Still, it is surprising how little heed is paid to such symptoms, which, among the poorer classes, seem to be taken almost as a matter of course. In making autopsies, I have repeatedly found the tubes in the lower parts of the lungs filled with pus, and even dilated, and the lungs themselves markedly emphysematous in the bodies of patients who had, perhaps, died in the surgical wards of the hospital, and who had never made any complaint of pulmonary symptoms. But after a few years this cough continues even during the summer; and there is constant dyspnœa, the breathing being hurried and even wheezing, especially during any exertion. For a long time there is no loss of flesh; but at length wasting occurs, and it may reach an extreme degree.

It is only during the early stages of chronic bronchitis that it is possible for the patient to recover, so as to remain henceforth free from the liability to the disease, when exposed to cold or damp. But even in advanced cases, occurring in persons of easy circumstances, it may often be kept at bay by their avoiding all changes of temperature, and spending every winter in a warm climate, or else remaining indoors throughout that season of the year. In this way life may often be preserved to its natural term, or even beyond it.

When death occurs, it is sometimes as the result of an intercurrent acute attack. Indeed, those who suffer from chronic bronchitis are exceedingly apt to have such attacks, attended with more or less pyrexia, which always cause more or less anxiety, but which may pass off, leaving behind them only an increased susceptibility for the future.

In other cases chronic bronchitis ends fatally by the supervention of dropsy, exactly like that of heart disease. The right chambers of the heart are thin, dilated, and hypertrophied; and the trunk and branches of the pulmonary artery are thickened, just as in mitral stenosis. I have notes of an instance in which the wall of the pulmonary artery was actually thicker than that of the aorta, and it was also atheromatous. Such changes never, I suppose, take place until emphysema of the lungs has developed

itself; and I should have thought that the obstruction to the pulmonary circulation was sufficiently explained by the defective aeration of the blood which occurs under such circumstances, and by the diminution of capillary area in the pulmonary system of vessels. But Traube has laid stress upon a third factor, namely, the deficiency of the movements of expansion and retraction of the lungs in breathing, which, under normal conditions, are supposed by him to further the flow of blood through the pulmonary capillaries. Ultimately the liver becomes nutmegged, the spleen and the kidneys indurated, the stomach congested. One important element in the bringing about these more remote changes is the occurrence of granular degeneration in the muscular substance of the right side of the heart. Another, which has not yet received its due share of attention, is a like degeneration of the diaphragm, as pointed out by the late Mr. Callender in the "*Lancet*" for 1857, and by Zahn, in vol. lxxiii of "*Virchow's Archiv.*"

Certain varieties of chronic bronchitis demand separate mention. One of them is that which Laennec called *catarrhe sec.* I have already spoken of a like form of acute bronchitis. But the cases that have now to be considered are described by Riegel as having a duration of several months, and as often ending fatally, at least in children. The most prominent symptom is a paroxysmal cough, which is so violent that the face becomes purple and the cervical veins swell out as thick cords. Yet there is no sputum, except, perhaps, a little tough mucus. Pyrexia is very slight, or altogether absent.

Another variety of chronic bronchitis is attended with a remarkable flow of a thin, watery, albuminous liquid from the mucous membrane, so that the name of *bronchorrhœa serosa* has been given to it. Laennec relates the case of an old man of seventy, who, for ten or twelve years, spat up such a fluid to the extent of about four pints every day, and yet remained fairly well nourished. But in other instances, as Andral pointed out in his "*Clinique Médicale*," extreme emaciation occurs, and weakness and pallor, almost resembling that which might have been caused by profuse hemorrhage.

Chronic bronchitis, if it lasts for any length of time, gives rise to certain secondary affections of the pulmonary parenchyma and of the air passages themselves, which affections add greatly to its gravity. They are respectively known as "emphysema of the lungs," and "bronchiectasis" or "dilatation of the bronchial tubes."

Emphysema of the Lungs—or "emphysema," as it is often called without any addition, when there can be no doubt that a pulmonary affection is intended—must not be confounded with that condition of the subcutaneous and other connective tissues which also bears the name of emphysema, and which depends upon an infiltration of them with air as the result of injury to some air-containing structure, so that it is more commonly seen by the surgeon than by the physician. What adds to the confusion is that in the lung itself an infiltration of air into the subpleural connective tissue does sometimes, though rarely, occur, and may go on until, passing along the root of the lung, the air diffuses itself through the mediastinum, and reaches even the superficial fasciæ of the neck and of the chest; this last affection is technically known as "interlobular" or "interstitial emphysema."

A very complete description of emphysema, both as regards its anatomical character and as regards its symptoms, was given by Laennec; before then it had almost escaped notice. Its real nature was first pointed out by Rokitansky, who showed that it consists in part of an over distention of the pulmonary alveoli, but also in part of an atrophy of their walls, causing their cavities to run together into irregular spaces, sometimes of very large

size. The name is, therefore, very appropriate, being derived from the Greek ἐν and φυσάω, I blow into.

Over distention of the lung tissue does not in itself justify its being spoken of as emphysematous. In children who have died after a few days' illness of laryngeal diphtheria, or croup, or acute bronchitis, it is common to find the lungs very bulky, and looking far more open textured than usual. One is very apt to speak of this condition as emphysema. But it is clear that there has been no time in such cases for the occurrence of atrophy of the alveolar walls, and in all probability if the patients had recovered, the lungs would quickly have returned to their normal state. Even when some amount of emphysema seems to be really present, it may happen that immediately after an attack of dyspnœa, a great apparent increase of the condition can be made out by physical signs, which yet subsides again within twenty-four hours. Hertz, in Ziemssen's "Handbuch," mentions the case of an asthmatic patient, aged thirty, in whom he observed such a transitory over distention of the lungs on several distinct occasions.

Anatomy.—The presence of emphysema of the lungs in the dead body is recognized partly by touch and partly by sight. The substance has a peculiar soft feel, like that of a down pillow; it scarcely crepitates, if at all, when squeezed between the finger and thumb; and pressure upon its surface readily causes a deep pit, which remains after the pressure is removed. This last circumstance depends upon the loss of the normal elasticity of the pulmonary tissue. Another effect of the same cause is failure of the lungs to collapse when the chest is opened. They often remain fully distended, and the left lung covers the heart, so that scarcely anything is to be seen of that organ. In a case, of which I have notes, which was observed at Guy's Hospital in 1868, one lung overlapped the other behind the sternum by an inch and a quarter. A similar condition which must, I think, have been pathological, was found in a body which I froze for the purpose of making a transverse section of it, when I was demonstrator of anatomy; a wax model of that preparation, which is now in the museum, shows the lungs covering one another for some little distance; there was advanced phthisis, and it is very likely that emphysema also existed, although this was not proved to be the case. In extreme instances the lungs bulge in all directions, both during life and after death, displacing the structures around them. Their apices protrude far above the clavicles; their bases bulge so that the diaphragm, from being arched upward, has a flattened or even (it is said) a concave upper surface. I have notes of two cases, in each of which the pericardial sac was pouched inward at its lower part, so that Dr. Moxon described the heart as resting upon and as being separated from the surface of the diaphragm by cushions of lung. Sometimes there are large bullæ or blebs containing air, of all sizes up to that of a walnut or a pigeon's egg. These are seen chiefly along the anterior borders of the lungs, but sometimes, also, along their inferior borders or near their roots. Not infrequently the ear-shaped process of the left lung shows a more marked degree of emphysema than any other part. But in some instances, even when the lungs are very highly emphysematous, no large cavities are to be seen. The tissue, if closely inspected, is found to be full of spaces of the size of fine shot or of millet seeds. One noticeable appearance is a rounding off of the free edges of the lungs, and their outer surfaces often show marks of the ribs, the intercostal spaces having yielded so as to allow the lungs to bulge outward. Emphysematous lung tissue is of a gray color, mottled with spots and lines of pigment, dry and bloodless.

The earliest change in a lung that is becoming emphysematous is, according to Rindfleisch, a dilatation of the infundibular cavities into the

sides of which the alveoli open; according to Hertz, a nearly uniform dilatation of the infundibula and also of the alveoli themselves. Gradually the alveolar walls waste until nothing is left of them but small ridges, projecting a little way into the interior of an oval or rounded space into which each infundibular cavity and its alveoli have now become resolved. After a time, the septa between these spaces, in their turn, thin away and become perforated. Thus the result is a progressive increase in the size of the spaces with a diminution of their number. An aggregation of fatty granules round the remains of the nuclei of the alveolar epithelium is commonly present, and in the "*Med.-Chir. Transactions*" for 1848, Mr. Rainey maintained that the morbid process concerned in emphysema was primarily and essentially a fatty degeneration; but there is no reason to suppose that this is the case. There is, of course, an enormous destruction of capillaries when the affection is at all extensive. Rindfleisch speaks of the vessels as collapsing until "only a narrow, ribbon-like band is left, which may be recognized as an obliterated vessel by its greater transparency amid a dark, often pigmented, parenchyma, and by its uniting with other bands like itself to form the usual anastomotic network." He goes on to say that some relatively wide communications are opened up between the pulmonary artery on the one hand and the pulmonary and the bronchial veins on the other hand. These anastomoses appear in well-injected lungs as peculiar elongated, unbranched, vascular arches of the same diameter throughout, contrasting very strikingly with a far more numerous assemblage of extremely tortuous and dilated arteries, for whose contents no such supplemental mode of escape has been provided.

Pathogeny.—With regard to the mode of origin of emphysema of the lungs there have been many different opinions. Laennec's idea was that the tubes in cases of bronchial catarrh being obstructed by swelling, or by an accumulation of mucus, the air which found its way into the alveoli during inspiration became unable to escape during expiration, inasmuch as the expiratory force was less than the inspiratory. In other words, he thought that emphysema was the result of a process, the exact converse of the ball-valve action which, as we have seen, was supposed by Dr. Gairdner to be the cause of collapse. It was objected by Louis that the ordinary seat of catarrh is the base and lower part of the lung, whereas the parts most apt to be affected by emphysema are the apex and the anterior margin. In 1851 Gairdner formulated a very definite theory of emphysema, which he regarded as arising solely during inspiration. His view was that collapse or reduction in bulk in one part of a lung is a necessary antecedent to the development of emphysema in another part. During inspiration, when the chest becomes enlarged, if each and all of the lobules cannot expand to fill it, some of them must be stretched unduly; and this constitutes emphysema. For a few years Gairdner's view was widely accepted, it being held that the affection was thus essentially "complementary" or "compensatory" either to collapse, or to retrogressive tubercular disease, or to some other contracting lesion of the lung. But, in 1856, Sir William (then Dr.) Jenner addressed to the Royal Medical and Chirurgical Society a powerful argument, proving that the development of emphysema occurred during expiration. And it is now known that this very same doctrine had already been taught in Germany, as far back as 1845, by Mendelssohn, in a work entitled "Der Mechanismus der Respiration und Circulation." Gairdner urged that it is impossible for emphysema to be produced by the act of expiration, even with a closed glottis, because the force by which the air becomes compressed within the lung opposes exactly as much resistance without as it creates pressure within. Jenner now pointed out (as Mendelssohn had done before) that certain parts of the thoracic walls are yielding, and

consequently incapable of maintaining this resistance. Both these observers indicated the apices of the lungs as being devoid of adequate protection against an expansile force from within; and we have seen that Louis had long before shown that the apices were especially apt to become emphysematous. Jenner remarked that during a fit of coughing the supra-clavicular regions may be seen to bulge, and that by placing one's hand upon them one can feel that they are distended by a considerable force. If the apices are the seat of emphysema, this bulging under violent expiration is extreme, and percussion over the bulging parts may elicit an almost tympanitic sound. He further showed that the sternum and the upper rib cartilages were to some extent yielding, and that, therefore, the alveoli of the anterior margin of each lung became affected with emphysema as well as those of the apex. Other parts which he also named as often becoming emphysematous were: the margin of the base of the lung, the part of the lung near its root below the entrance of the bronchus, and the little ridge of lung which, on the right side, projects behind the trachea. The base of the left lung generally he declared to be less firmly supported than that of the right lung, the liver being, of course, more unyielding than the stomach; and he cites Louis as having found the left lower lobe emphysematous twice as often as the right one. The correctness of Jenner's view has since been supported by observations made in certain cases of congenital malformation of the chest walls. Thus, in the case of Groux, who had a fissure of the sternum, the anterior part of the lung protruded through the fissure in the act of coughing. Ziemssen met with an example of absence of the *pectoralis minor*, and of the entire sterno-costal part of the *pectoralis major*, so that the intercostal muscles of the four upper spaces were covered only by fascia and by integument. During forced expiration these spaces bulged from 1 to 1½ mm. above the level of the ribs; when the muscles of one space were faradized, that space for the time remained flat, the others bulging as before. Further evidence is afforded by cases in which, after the cicatrization of penetrating wounds of the chest, the affected parts have ultimately become the seat of hernia of the lung, as the result of weakening of the thoracic parietes. Many such instances may be found collected in a little work by Desfosses, 1875. It may be noted, too, that horses are liable to an affection of the lungs identical with emphysema, as the result of the straining efforts which they are called on to make, during which they keep the glottis closed. As Sir William Jenner says, in vol. iv of Reynolds' "System," "No one who watches a horse draw a heavy load up a short, steep incline on a damp, cold day can doubt this. While making the effort, the horse holds its breath, having previously inflated the lungs. No sooner, however, does this animal cease its effort than the glottis is opened, and the air suddenly expressed from the lungs. The degree to which the air was compressed may be judged by the distance to which, and the sudden violence with which, the cloud of breath vapors is seen to be driven forth." In his paper in the "*Med.-Chir. Transactions*" Jenner had shown that the parts of the lungs that are emphysematous in a "broken-winded" horse are those which are so placed as to be least able to resist pressure. It is curious that a capital description of this affection of horses was given before the end of the seventeenth century by Sir John Floyer, in a treatise on asthma; the passage is cited in full in "Watson's Lectures."

The true theory of emphysema, then, is that it is the result of expiratory pressure with a closed, or partially closed, glottis. The expiratory muscles forcibly compress the air within the chest. If all parts of the thoracic parietes were equally unyielding, no harm would result. But as certain parts can and do yield, some of the compressed air is driven into the corresponding alveoli of the lungs, and gradually breaks down their structure in the

manner already described. No doubt the resistance of the chest walls in different regions fails progressively, more and more, as the affection advances. Thus the sternum and the upper rib cartilages become arched forward, a change which probably is due to the frequently repeated application of an expansile force from within the thorax. It is only as the result of long continued pressure that the diaphragm can become flattened, and that the lung can protrude inward beneath the heart, after the manner described at p. 893. Even when a part of the lung (generally the apex) is shrunken by retrogressive tubercular disease, I agree with Jenner in thinking that the development of emphysema in the tissue around is still probably due to the pressure of air driven into the alveoli during violent expiration. Many writers, however, are of opinion that to such cases, at any rate, Gairdner's theory remains applicable, the emphysema being "complementary" in the strictest sense of the term.

It must be understood that although a frequently repeated cough is one of the chief causes of emphysema, yet in man, as in horses, other actions, besides that of coughing, may induce an amount of expiratory pressure sufficient to give rise to the affection. This is the case, for example, with all violent efforts, in which the glottis is kept closed to fix the chest and to afford a firm base for the operation of muscles passing to other parts of the body. Dragging or lifting heavy weights, straining at stool, even the act of parturition, may be mentioned as possible causes of emphysema. Thus Waldenberg is cited by Hertz as having seen the affection develop itself in a medical student who, having come from a country place where he had no occasion to ascend stairs, occupied in Berlin an apartment on the fourth floor, up to which he ran without stopping several times daily. Hertz himself met with a similar case in a young shopman, whose lungs became emphysematous in about a year, without any cough or bronchial catarrh, as the result of his having to carry heavy goods up a high staircase, in haste, a great many times every day. To persons who have suffered from bronchitis, and in whom the distending process has already begun, it is a very important piece of advice that they should avoid all occupations or amusements that involve the repetition of such efforts. I can well believe that playing a wind instrument may sometimes be exceedingly injurious; for, although the glottis is not closed, the air within the chest is kept under great pressure, while it is being slowly allowed to escape.

Of late, German observers have been disposed to attribute emphysema in part to changes in the pulmonary tissue itself, independent of mechanical conditions such as those to which I have been adverting. A *senile atrophy* of the lungs, bearing a close resemblance in its characters to emphysema, is generally admitted by pathologists; it was originally described by Dechambre in 1835, from observations made at the Salpêtrière. Sir William Jenner speaks of it as "small-lunged emphysema," in contrast with the ordinary form of the disease, which he calls "large-lunged emphysema." As he says, the small size of such lungs, their lightness, and the very small space into which they may be compressed, are often most remarkable. When the thorax is opened after death they "fall in like an inflated bag of wet paper." The subjects of senile atrophy of the lungs are commonly thin, withered-looking, shriveled old persons. Their chests are very small and narrow, the lower ribs being so obliquely placed that they almost reach the crest of the ilium, and so closely packed as nearly to come in contact with one another. The lungs are so reduced in size that the extent of præcordial dullness may be increased, notwithstanding that the heart itself partakes of the general wasting. Yet there is commonly little distress of breathing, because the volume of the blood is at a minimum and because the deficiency of muscular power forbids active exercise. I must confess, however, that it has always seemed

to me that the supposed likeness of this senile atrophy of the lungs to emphysema is really due to the fact that a slight degree of emphysema, resulting from the bronchial catarrh to which aged persons are so liable, is commonly mixed up with it. Hertz, in his chapter on atrophy of the lungs in Ziemssen's "Handbuch," speaks of bronchitis as a frequent "complication;" and he also mentions that the bronchioles are very thin and "generally uniformly dilated, seldom irregularly sacculated." But bronchiectasis can hardly be otherwise than mechanical in its origin; and I think that it is reasonable to take the same view of the pulmonary rarefaction, which Hertz describes as being most marked at the apices and along the anterior edges.

That in younger persons some cause for emphysema must exist, beyond expiratory pressure upon the alveoli, is argued by Hertz from the fact that in certain families several members are found to suffer in succession, as the result of comparatively trifling affections of the air passages. Schnitzler, for example, saw three brothers, whose parents were still alive and well, but who all became the subjects of emphysema at the age of thirty, without definite cause. Walshe cites Dr. Jackson, of Boston, U. S., as having upheld the view that the disease is transmitted by inheritance; he found that "of twenty-eight emphysematous persons, eighteen had either a father, or a mother, or both, similarly affected; whereas of fifty non-emphysematous people, three only sprang from emphysematous parents." Dr. Greenhow some years ago insisted on the existence of a relation between emphysema and the "gouty diathesis." So far as concerns the mere occurrence of this affection in different members of the same family, it is important not to overlook the fact that they may all have been alike exposed to the causes of bronchial catarrh, and perhaps all alike unduly susceptible of taking cold. But Cohnheim and other recent German writers have looked for an explanation of emphysema, apart from mechanical causes, in a varying physical state of the pulmonary tissue, as regards its degree of elasticity. In this direction certain observations of Perls may be cited, of which there is a record in vol. vi of the "*Deutsches Archiv.*" By means of a pressure gauge he determined in a large number of cases the degree of force with which the lungs retracted when the pleural cavities were opened in the dead body; and he found that after death from enteric fever, or (in one case) from phosphorus poisoning, their elasticity was reduced almost to nothing. Cohnheim regards it as an established fact that in a very large proportion of cases emphysema depends upon a congenital defect of development in the elastic tissue of the lungs. But the reference which he gives to a paper by Eppinger, in the "*Prag. Vierteljahreschrift*" for 1876, does not seem to bear out this assertion, for although Eppinger found that even in slightly emphysematous lungs there was a great reduction of the network of elastic fibres in the alveoli, the smallest fibres having completely disappeared, he yet appears to have regarded this as a change occurring in the course of the development of the disease, and not as an antecedent malformation. In connection with this question a case recorded by Hertz is of great interest. It is that of a regimental cornet player, aged thirty, who had always been able to use his instrument without any difficulty, even on the march, when he was attacked with double pneumonia, which, however, subsided in a week. He afterward felt quite well, having no cough, and began again to practice with his cornet. But in the course of the next seven months he discovered that he was no longer able to take sufficient air into his lungs to maintain a long note for the proper period, and that he was short-breathed on exertion, and could not now play while marching. Hertz found on examination that the lungs were markedly emphysematous, which had not formerly been the case; and his supposition is that the

pneumonia had damaged their texture, so that they were not able to resist expiratory pressure, as they formerly had been.

Clinical Symptoms.—The recognition of emphysema is based mainly upon physical signs. The chief symptom of the disease is dyspnœa. The first thing noticed by the patient is that he is short of breath when he exerts himself, as in running up stairs; but after a time difficulty of respiration becomes a permanent condition from which he is never entirely free. As Dr. Walshe says, he feels as if his chest were never emptied of air as it naturally should be; and he is conscious of an annoying sense of inflation or distention. It is true that many emphysematous persons affirm that their dyspnœa is only occasional; but Dr. Walshe says that in all such cases which he has seen, the patient has been deceived, a moderate amount of dyspnœa having become to him a second nature—a thing unperceived and giving rise to no discomfort.

It does not necessarily happen that the affection advances; it may continue stationary, and life may be maintained until extreme old age, provided that the risk of intercurrent attacks of bronchitis can be obviated. Hertz supposes that the existence of emphysema necessarily involves an increased liability to bronchial catarrh, by leading to congestion of the mucous membrane of the air tubes; but I think it would be very difficult to give direct proof of this, seeing how very common bronchitis is as an antecedent condition.

On the other hand, in the majority of cases, emphysema becomes more and more marked every year. After a time the noisy, hurried breathing may become so short that the patient cannot utter a sentence without stopping in the middle of it. At night he has to be propped up by pillows. His distress becomes aggravated from time to time, sometimes by an exacerbation of bronchial catarrh, sometimes by the supervention of asthma, sometimes by mere pushing upward of the diaphragm, as the result of distention of the abdomen with flatus, or with undigested food. The face, the hands, and even the body generally become livid to the most extreme degree, the condition of the patient being exactly that which has been described at pp. 880, 884, 891, as resulting from bronchitis. In fact, chronic bronchitis never goes on for any length of time without being complicated with more or less of emphysema, so that it is impossible to separate the effects of these two lesions. Cough, on the other hand, may be entirely absent in cases of emphysema unless there is bronchial catarrh. But many persons whose main disease is emphysema, constantly have cough, and expectorate a frothy liquid, or pearly-gray masses of mucus.

Hæmoptysis is not generally said to be among the symptoms of this affection. Dr. Duckworth, however, in vol. xi of the "*St. Bartholomew's Hospital Reports*," declares that it is of not infrequent occurrence. My own impression has certainly been that streaks of blood in the sputa are often seen, without there being any reason to suspect the presence of any phthisical lesion. Dr. Duckworth remarks that the wasting of the pulmonary capillaries and arterioles affords a sufficient explanation. In 1869, a woman, aged forty-nine, was brought dead into Guy's Hospital, of an attack of severe hæmoptysis, and at the autopsy the only disease that could be discovered was an extreme degree of emphysema of the upper lobes of the lungs, with some excess of fibrous tissue forming the interlobular septa. The air tubes were full of clots. She was said to have suffered for three months from wheezing dyspnœa; on the morning of her death she woke up at 4.25 A.M. with "coughing and vomiting of blood through the nose and the mouth, and was suffocated in ten minutes."

Ultimately emphysema gives rise to great wasting, and to extreme enfeeblement of the muscular strength. One noteworthy circumstance,

pointed out by Walshe, is that the over distention of the chest renders the body unnaturally buoyant in water, so that the patient is astonished to find himself able to swim more easily than before, at the very time when he is growing more and more short-winded for all other kinds of exertion.

Physical Signs.—Of these, the most important are those derived from *percussion.* Even very slight degrees of the affection may be detected by carefully mapping out the area of the heart and of the liver respectively. Instead of beginning at the upper border of the fourth left costal cartilage, the cardiac dullness is discoverable only at a lower level, over the fifth or even the sixth cartilage. Instead of beginning at the upper border of the sixth right rib the hepatic dullness is discoverable only at the level of the seventh or of the eighth. If in a case of bronchitis one finds that percussion over these two organs yields normal results, one is generally safe in declaring that no appreciable amount of emphysema has as yet developed itself.

As the disease advances, the heart becomes so completely covered by lung that no cardiac dullness at all can be detected, the pulmonary resonance above meeting the tympanitic sound caused by the stomach below. At the same time the apex beat ceases to be felt in the normal position, in consequence of the downward displacement of the diaphragm carrying the heart with it; and the axis of the organ becomes altered, so that its pulsations can often be felt in the epigastrium. On the right side pulmonary resonance now extends down to the margins of the ribs. The edge of the liver may sometimes be felt in the hypochondrium. But very often this is not the case and there may be a marked reduction in the area of the hepatic dullness, so that one may be tempted to suspect that the organ is atrophied or cirrhosed when this is not really the case. The chief reason why in such circumstances the liver fails to be in contact with the parietes of the chest or of the abdomen to the normal extent is the great increase in the antero-posterior diameter of the trunk, to which I shall presently refer. Another noticeable feature of well-marked cases of emphysema is the clear character of the percussion sound over the sternum as high as its upper border; and over the bases of the lungs behind a clear percussion sound is elicited to a much lower level than normal.

Again, the quality of the percussion sound, over parts of the chest where it ought naturally to be resonant, is altered whenever the lungs are emphysematous in any considerable degree. The alteration is in the direction of hyper-resonance, and according to Dr. Walshe and Dr. Gee there is often a fall in its pitch, so that it may fairly be described as tympanitic. The change in the character of the percussion sound is often very conspicuous over the backs of the lungs, a sonorous, drum-like note being readily produced where normally there would be great difficulty in eliciting anything approaching a clear sound.

Next to percussion, *inspection* affords the most valuable indications of emphysema. I have already alluded to the arching forward of the sternum and of the upper ribs as the result of their yielding to the frequently repeated expiratory pressure which is the cause of the affection. The sternum not infrequently becomes convex in a vertical plane, with an angle, known as the *angulus Ludovici*, at the junction of the manubrium with the body. According to Freund, the rib cartilages grow in length and in breadth—a change which he actually regarded as the starting point of the pulmonary disease. The clavicles, also, are far more bent than under normal circumstances. In addition to this the curve of the dorsal vertebræ becomes greatly increased. Sometimes the back is so rounded that the scapulæ seem to be almost horizontal in position. The effect of all these alterations in the parietes of the chest is to give it a cylindrical form. It is often aptly said to be

"barrel shaped." By the cyrtometer its horizontal circumference is shown to be almost perfectly circular, as is well illustrated in a diagram given by Dr. Gee. Sometimes the rounding of the ribs and of their cartilages continues to the very bottom of the thorax, the angle at the ensiform cartilage being far more open than usual, and the hypochondriac regions permanently expanded to the fullest possible extent. One result of this is, as Hertz has pointed out, a transverse groove, which crosses the abdomen horizontally from one twelfth rib to the other; it is due to the stretching of the upper part of the transversalis abdominis muscle, which is fixed to the rib cartilages, as compared with the relaxed condition of the lower part, which has no such attachment. I have known this groove, a very conspicuous and at first sight puzzling feature of a case, when there was at the same time a considerable accumulation of fluid in the peritoneal cavity. But in other instances the lower ribs and their cartilages are flattened, or even hollowed inward; this occurs especially when the pulmonary affection began in bronchitis or whooping-cough at an early period of life, so that the bases of the lungs became collapsed. The state of the intercostal spaces in emphysema has been the subject of a good deal of discussion among writers on diseases of the lungs. That the upper ones are much widened is certain. What has been regarded as doubtful is whether they are depressed below the corresponding ribs, or at the same level, or bulging. Stokes declared that he had never seen them otherwise than depressed. But Walshe and others say that it is not uncommon for them to be prominent or bulging when the parts of the lungs beneath, being highly emphysematous, have lost their elasticity.

Further indications as to the presence of emphysema, and as to the extent to which it has advanced, are yielded by observation of the act of breathing. During inspiration, the chest in well-marked cases is seen to be almost motionless. It is, indeed, impossible for the upper ribs to rise and expand, as they normally should do, because they have permanently assumed a position far in advance of that which could ever have been reached by them in health. There is, however, a jerking movement upward of the thorax as a whole, produced mainly by contractions of the sterno-mastoidei and of the scaleni, which start into unnatural prominence and appear to be considerably hypertrophied. If the lower ribs are thrown outward as much as the upper ones, the chief agent in inspiration must be the diaphragm. But in many cases the state of affairs is reversed. The lower ribs still retain a certain degree of mobility, but the diaphragm is pushed downward so that it can do scarcely anything toward enlarging the thoracic cavity. It may then be observed that the epigastrium becomes hollowed during the act of inspiration. According to Hertz, the diaphragm sometimes actually becomes convex toward the abdomen, so that the contractions of its muscular fibres must necessarily cause its tendinous centre to move upward, and it becomes a muscle of expiration and not of inspiration.

I have already mentioned the effect of cough in causing bulging of the suprascapular and suprasternal as well as of the upper intercostal spaces. Sir William Jenner alludes to the possibility of confounding this condition at the root of the neck with a prominence of the same part due to distention of the veins. In speaking of aneurism of the aorta, I shall have to mention a still more important source of error in the sudden protrusion of an aneurismal sac during the act of coughing. A point which is, perhaps, also worthy of notice, and to which Jenner has drawn attention, is that if one is feeling the pulse of an emphysematous patient while he coughs violently, one perceives the artery to become suddenly full and tense, after which it ceases for the moment to beat.

The stethoscope gives comparatively little information in cases of emphysema, apart from the signs of the bronchitis that is so commonly associated

with it. The most marked *auscultatory* sign of the emphysema itself is an enfeeblement or absence of the vesicular murmur. Hertz further mentions, and I have repeatedly verified the fact, that the bronchial breathing which may normally be heard over the roots of the lungs behind and beneath the clavicles is often wanting in emphysematous patients. But even in those who are healthy this is not infrequently the case, as I have already remarked at p. 870. In many instances the expiration is exceedingly prolonged. Walshe says that instead of being only one-third the length of the inspiration, it may be four times as long; in other words, its relative duration may be increased twelvefold. The "growling" or "wheezing expiratory sound of emphysema" is, indeed, very commonly spoken of. But it seems doubtful whether this sign does not belong rather to a concomitant bronchitis than to the pulmonary lesion itself. Walshe points out that it ought strictly to be termed a fine expiratory sibilus. And I well remember one case in which as the tube became free this sound entirely disappeared, although the extremely feeble state of the inspiratory murmur and the hyper-resonance of the percussion sound clearly showed that emphysema still persisted. When air has escaped into the subpleural connective tissue, constituting what is known as "interlobular" or "interstitial emphysema," there is sometimes heard a friction sound very like that which occurs in pleurisy. This sign was originally noticed by Laennec. Most writers since then have been disposed to doubt the correctness of the observation; but it has been recently confirmed by Dr. Gairdner; and Dr. Hudson, in his edition of Stokes's works, says that he also met with a case in point. In the latter stages of pulmonary emphysema a tricuspid regurgitant murmur may often be detected; and even at an earlier period increased pressure on the venous side of the circulation may be indicated by an intensified pulmonary second sound.

Dilatation of the Bronchi—Bronchiectasis.—This affection was first described by Laennec. It comparatively seldom constitutes a substantive disease. In some instances it is associated with fibroid induration of the lung tissue intervening between the enlarged tubes; such cases I leave to be discussed in the account which I shall have to give of chronic pneumonia or cirrhosis of the lung, and also in the chapter on phthisis, in which complaint, however, I believe bronchiectasis to play a very much less important part than is supposed by many pathologists. The only cases that I shall now take into account are those in which the pulmonary parenchyma is either healthy or else more or less extensively emphysematous or collapsed, showing, in fact, no changes except those that we have seen to be frequent secondary results of bronchial inflammation.

Anatomy.—All writers divide bronchiectases into cylindrical and sacculated. In the *cylindrical* form of the affection the tubes may run through the substances of the lung, with but little diminution of calibre, until they end abruptly beneath the pleural surface. Their appearance is then often compared to that of the fingers of a glove; but it very rarely happens that this is the case throughout their whole length. In one instance, which occurred at Guy's Hospital in 1873, they were so enlarged as to be conspicuous at the root of the lung, "pushing the lobes apart from one another," as it is expressed in the report of the autopsy. Much more often, the medium-sized and smaller tubes are alone affected. The existence of bronchial dilatation may then be obvious on the cut surface of the lung, far too many large orifices being visible, from which pus wells up in great quantities. Or, in order to detect the enlargement of the tubes, especially if they are empty, it may be necessary to carefully open them up with scissors. One

may then find, perhaps, that only a few of them are dilated in any marked degree. Judging from my own observations, I should say that such slight forms of bronchiectasis are much more often met with in the extreme bases than in any other parts of the lungs. Not infrequently I have seen enlargement of the principal tube passing into the ear-shaped process of the left lung, when I could make out no such change elsewhere. This was when the ear-shaped process itself was emphysematous; and, indeed, these less-marked examples of bronchiectasis are very often found in association with emphysema. On the other hand, when the bronchial affection reaches an extreme degree, there is seldom a corresponding amount of rarefaction of the pulmonary tissue. If the tubes in any one part of the lung are universally dilated, running to the surface side by side, and, perhaps, as large as quill-pens or even larger, the parenchyma between them is almost necessarily reduced to a small space. In these cases it is sometimes difficult, at the first glance, to say whether the affection is or is not secondary to a fibroid change in the lung itself. Such a condition is sometimes found in the middle of the lung or even of the upper lobe, the lower lobe being free. It must be understood that cylindrical bronchiectases are by no means always absolutely uniform in diameter at different points. Sometimes they gradually widen as they approach the surface. Sometimes they have fibrous bands or trabeculæ projecting from their walls here and there and rendering their calibre very irregular.

Sacculated dilatation of the bronchial tubes, again, varies widely in character in different cases. The most typical form is one which presents appearances that I have never seen exactly described. When a section is made of the lung the cut surface appears to be covered with an immense number of shallow, smooth-walled depressions, exactly like so many minute saucers. Each of these has in its floor a very small, rounded orifice, and it is obvious that they are all sections of little bronchiectases, which probably were spherical before they were cut across, but of which the halves have become flattened by their own elasticity and by that of the adjacent pulmonary parenchyma. I have met with at least four instances of this kind. In all likelihood, if the lung in such a case could be inflated and dried, and the parenchyma then cleared away so as to expose the tubes in their continuity, they would be found changed each into a regular series of globular dilatations, so as fairly to deserve to be called moniliform. In other instances sacculated bronchiectases are more unequal in size and irregular in form; but I believe that most of the cavities of this sort are really smooth-walled vomicæ which have been formed by ulceration. I can only attribute to such an error the statement of Biermer (in vol. v of Virchow's "Handbuch") that the majority of bronchiectactic sacculi have openings only toward the trachea, being entirely closed on the distal side. Nor am I at all ready to accept his opinion that even this communication is sometimes obliterated, so that they are converted into shut cysts.

Dilated bronchial tubes often have exceedingly delicate walls, being apparently as thin as, even if not thinner than, they were before they began to increase in size. But sometimes the tissues are thickened, the lining membrane being velvety and showing the changes above described as occurring in bronchitis.

Pathology.—As to the way in which bronchiectasis arises, I do not think that there can be any doubt that it is the mechanical result of expiratory pressure, being, in fact, produced exactly by the same cause as pulmonary emphysema. To explain satisfactorily why in one case the lung tissue should yield, and in another case the tubes, would, perhaps, be impossible. But we may fairly suppose that it depends upon the degree to which the walls of the tubes have been softened by inflammation, and also, it may be, upon the

amount of elastic resistance possessed by the pulmonary tissue in each particular instance. It accords with this view that (unless I am greatly mistaken) dilatation of the tubes, unlike emphysema, is more apt to occur in the bases of the lungs than elsewhere, the bases being the most frequent seats of bronchitis.

Diagnosis.—It is only in cases in which bronchiectasis has reached rather an extreme point that it can be said to be characterized by definite *physical signs*. As a rule, I think that the chief indication of this affection is the presence of râles which appear to be too large and coarse to be formed in the tubes of the part of the lung in which they are heard, as, for instance, at the extreme base, or along the anterior edge. If, however, a number of tubes cylindrically dilated are arranged side by side, while the lung tissue between contains but little air, there may be more or less marked bronchial breathing, bronchophony, and even dullness on percussion. But such a case could not be clinically distinguished from primary chronic pneumonia, with bronchiectasis as a minor feature. Nor again does the question of the diagnosis of a saccular dilatation of a bronchial tube from a phthisical vomica, to which question stethoscopists formerly devoted themselves with much ardor, apply at all, so far as I know, to cases arising out of bronchitis alone, and independently of a lesion of a pulmonary tissue proper. One circumstance which is strongly indicative of bronchiectasis, as may be understood from what is about to be stated of the symptoms, is that the physical signs over a certain part of the lung should undergo more or less regular variations from time to time, being now well marked, and now again indistinct or even altogether absent.

As for its *symptoms*, dilatation of the tubes is, of course, in part concerned in causing cough, dyspnœa, and lividity in patients affected with it. But one can never clinically separate its share in producing these effects from that taken by the bronchitis which is necessarily associated with it, and, perhaps, also by a concomitant emphysema. The only thing that enables one to diagnose bronchiectasis with confidence is a peculiar way of expectorating which may in some cases be observed. For some hours, perhaps, there is no cough at all. During this time liquid is accumulating in the dilated parts of the air passages, the sensitiveness of which appears to be blunted, so that they do not resent its presence. Then it perhaps happens that some runs over into a tube which is still healthy. The result is a more or less violent fit of coughing, by which all the liquid that has collected is suddenly expelled, pouring out of the patient's mouth, and even through his nose, in enormous quantity, so as to half fill his spittoon. Sometimes this process is set in action by an attempt at physical examination of the chest. Sometimes it occurs, especially in the morning, when the patient rises from the recumbent posture. When this accumulation has been got rid of he usually feels much more comfortable than before, the breathing is easier, and the chest less oppressed.

Fetid or Putrid Bronchitis.—It is especially in cases in which the bronchial tubes are dilated that bronchitis becomes accompanied with the expectoration of foul-smelling sputa, and sometimes with horrible fetor of breath. Traube has, indeed, recorded one or two cases in which putrid bronchitis occurred without there having been any bronchiectasis. But, as a rule, it is only when liquids have become for a considerable time stagnant in some part of the air passages, or in a space communicating with them, that putrefactive chemical changes occur.

The characteristic symptom of putrid bronchitis, as was first pointed out by Traube, is the presence in the matters expectorated of certain soft, friable, smooth masses, of a dirty grayish-yellow color, of very fetid odor,

varying in size from a millet seed to a bean. Such bodies had been originally noticed by Dittrich in 1850 as plugging the affected tubes in fatal cases; and in Germany they are commonly called "Dittrich's" or "Traube's plugs" (Pfröpfe). Microscopically they are made up mainly of a finely granular detritus mixed with fat globules. They also often contain certain very long, narrow, acicular crystals, of which Virchow gave a description long ago, in the first volume of his "*Archiv*," as consisting of a fatty acid of a peculiar unpleasant odor. These crystals are colorless, often sharply bent or even twisted, sometimes collected together in sheaves or in thick bundles. Some of them may appear to be varicose, a condition which Traube has shown to be the result of pressure by the cover glass. According to Guttmann they contain a combination of palmitic and stearic acids, and not merely margaric acid, as used to be supposed. In 1867, Leyden and Jaffé further pointed out, in vol. ii of the "*Deutsches Arch.*," that under high powers the granular detritus is composed of vegetable organisms, some round, others rod shaped, others forming beaded chains or long filaments. Their presence appears to be the reason why iodine often gives a purple, or a violet, or even a blue tint to the whole mass, as Virchow originally observed. Chemical analysis of the sputum of putrid bronchitis in different cases has also shown that it may contain volatile fatty acids (valerianic, butyric, and acetic acids), methylamine, leucin, and tyrosin, ammonia and sulphuretted hydrogen. The authority for including methylamine and acetic acid in the list is Dr. Gregory, of Edinburgh, as reported by Dr. Laycock in the "*Med. Times and Gazette*" for 1857. It is also worthy of mention that Leyden and Jaffé succeeded in inducing in ordinary muco-purulent sputum, outside the human body, a putrefactive process closely analogous in its results to that which must be supposed to give its peculiar character to the expectoration of patients with putrid bronchitis. They suppose that the source of the vegetable organisms described by them is the common leptothrix of the buccal cavity.

In putrid bronchitis the sputum as a whole is generally very abundant. It separates in the spit jar into three layers: of these the uppermost is opaque, greenish-yellow, and frothy; the middle is a transparent, albuminous liquid like serum; the lowest is opaque and of a dirty-yellow appearance, consisting mainly of swollen pus cells and of the *débris* resulting from their destruction.

The odor of the sputum or of the patient's breath in cases of putrid bronchitis is commonly identical with that which belongs to gangrene of the lung, as one observes it in the post-mortem room under most varied conditions. Guttmann compares it with the smell that pervades a soap manufactory. But in other cases it is of an altogether different character. There is, of course, great difficulty in defining the distinction verbally, but I may cite Dr. Laycock's statement that in one of his patients the odor was like "that of the mayflower or of apple blossom with a kind of *arrière goût* of fæces." He referred its origin to volatile compounds, such as the "butyrates of ethyl." I have often observed this kind of smell, especially in cases in which dilated bronchial tubes were emptied with a gush of enormous quantities of fluid at once, after the manner described above. I do not suppose that in such cases there is generally any active process going on in the walls of the affected tubes themselves. On the other hand, in many of those cases in which the odor is like that of gangrene of the lung the development of fetor in the sputa indicates the abrupt commencement of a destructive change both in the air passages and in the pulmonary parenchyma, which rapidly brings about a fatal issue. The credit of having first pointed out the clinical features of the disease in cases of this kind belongs to Dittrich. His description is that it "commonly arises in persons of the middle period

of life who have suffered for years from bronchial catarrh, with abundant muco-purulent expectoration, and who may either have already begun to waste, or may still remain well nourished. Suddenly, and without apparent cause, the sputum becomes offensive, of a dirty-gray color; the breath also stinks, poisoning the air around. Thereupon follow severe dyspnœa, fever of typhoid character, rapid collapse, an earthy, dirty-yellow complexion, and ultimately cessation of expectoration, coma, and death. At the autopsy the walls of some of the bronchial tubes are found intensely inflamed and sloughing. There are more or less extensive areas of pneumonic consolidation, passing here and there into gangrene. Other parts of the lung tissue are œdematous, exuding a fetid liquid. The bronchial glands are greatly swollen, soft, and of a dirty-gray color. Several cases exactly in point have since been recorded by Traube. It must be noted that the occurrence of hepatization and gangrene of the substance of the lungs is by no means limited to those regions which were before the seats of bronchiectasis. Pneumonic patches may be scattered throughout every part of the organ on each side; and it seems obvious that many of them owe their origin to the inhalation of particles of putrid *débris* derived from tubes already diseased into such as have hitherto been healthy.

But the issue of putrid bronchitis is not always thus serious. Slight cases sometimes end in recovery. We may then suppose either that there has been no ulceration of the walls of any of the tubes, or that the necrotic process has been limited, and that healing has taken place after detachment of sloughs of no great size. Other cases, again, run on for months with but little change in the symptoms, and without marked impairment of the general health.

To complete my account of the effects of dilatation of the bronchial tubes I may mention that in vol. xv of the "*Deutsch. Archiv*," Gerhardt has recorded two cases in which a painful swelling of some of the joints occurred as a sequela. He was inclined to regard this rheumatoid affection as analogous to that which is met with after gonorrhœa or dysentery. To the occasional supervention of abscess of the brain as a complication of suppurative processes in the air passages or in the lungs I have referred elsewhere (*supra*, p. 562).

Of the *causes* of bronchitis generally, the chief is *exposure to cold*. In all probability cold air entering the air passages through the nose, or the mouth, sometimes acts upon them as a direct irritant. It is true that the nasal mucous membrane warms the air that traverses it, but, on the other hand, the experience of persons who venture into the open air at a low temperature, with a bronchial surface already in a morbidly sensitive condition, is conclusive as to the fact that cough is very apt to be excited, and that this is not the result of mere exposure of the skin to the cold. Still, the analogy of so many other inflammatory affections of internal structures, which contain no tubes communicating with the external atmosphere, is entirely in favor of the view that bronchitis may also be set up by the action of cold upon the surface of the body. And this is borne out by the circumstance that in many cases there is no reason to suppose that cold air has been inhaled. A patient may, for example, "take a chill" by getting wet through, by sitting in a draught, by lying on damp grass, by merely remaining motionless out of doors when perspiring profusely after exertion. As a rule, it is especially when the body, from having been heated, is cooling that danger of catching cold exists. The reason appears to be that whenever the body has more heat to dispose of than is required to maintain its due temperature the cutaneous capillaries become dilated to allow as much loss of heat as possible. This is equally true whether the heat is supplied

to the organism from without or generated in its interior (as when it is the result of muscular exertion). Accordingly, after a Turkish bath, one plunges for a few seconds into cold water, which causes contraction of the blood vessels, before one ventures to sit in a room at an ordinary temperature. Rosenthal, and afterward Riegel, have shown by direct experiment that if animals after exposure to great heat are removed and placed in air which is not warmed, they go on cooling until their temperature falls below the normal point. They, therefore, suggest than when a person catches cold, what occurs is that blood from the surface of his body, chilled by loss of its heat, is carried to deeper structures, until they also become less warm than natural. If now there be anywhere a *locus minoris resistantiæ*, it suffers and becomes inflamed. Some individuals are far more sensitive to the action of cold than others. The risks of catching cold, which such persons are constantly running, may, however, be often obviated to a great extent by *hardening* the skin, that is, by exposing it regularly to sudden changes of temperature, so as to accustom its vessels to contract promptly and vigorously. The best way of doing this is doubtless to sponge the surface with cold water, or to use a cold douche or a shower bath every morning after a tepid bath. The cold bath by itself is probably less effectual for the particular end in view, although it is all that is required for robust persons with an active circulation, in whom it is followed by a good reaction. Even in young children a warm bath, especially in the morning, may always with advantage be followed by rapid sponging with water somewhat chilled.

Many *secondary*, or (as they would formerly have been called) *predisposing*, causes of bronchitis are really conditions which favor the injurious action of cold. Thus the disease is very apt to attack young children, on account of their feeble powers of resistance. A curious point that came out in some investigations made by Geigel as to the infant death rate in Würzburg was that bronchitis was relatively less fatal to illegitimate than to legitimate children, the reason being, in all probability, that the latter are coddled up and kept warm, so as to be rendered more sensitive to cold than they otherwise would be. The prevalence of the disease in Europe is, as might be expected, least in the hot season of the year, from June to September, but it is a remarkable circumstance, to which I shall again have occasion to allude, that its proportionate frequency in different months is by no means exactly the same as that of acute pneumonia. This is true, also, of the geographical distribution of the two affections. Bronchitis increases in frequency from the equator toward the poles, but the increase is not uniform in all longitudes, varying with the climatic conditions of each particular country. What favors it most is not a low mean temperature, but the occurrence of sudden and violent changes of temperature, and above all the presence of much moisture in the air. In some parts of the tropics bronchitis is by no means uncommon at the end of the hot season. There are certain countries in which it is of very rare occurrence, especially Egypt, the western prairies of North America, the plains of India, a part of the Antilles, and California.

Next to cold, the entrance of irritant substances into the air passages during breathing is the most important cause of bronchitis. In speaking of the ætiology of phthisis I shall be obliged to discuss in detail the influence of various occupations, in which the inhalation of dust is almost inevitable upon the production of that disease. All those occupations also cause a great liability to bronchitis which may either in the course of time be followed by the subsequent development of phthisis, or run a chronic course without any such complication until it ends fatally by the supervention of an acute attack, or by dilatation of the heart and dropsy. As I shall have to

point out, pulverulent substances which happen to be colored, such as carbon or oxide of iron, often tinge the sputum deeply when they have been inhaled. But, on the other hand, it may happen that a miner, whose lungs are anthracotic, spits up a yellow muco-purulent fluid, containing no carbon whatever. This accords with the fact that the bronchial mucous membrane never itself becomes the seat of anthracosis; even the peri-bronchial tracts of fibrous tissue derive the black deposit which is found in them from the surrounding pulmonary alveoli, and not from the tubes themselves.

The inhalation of gases, especially nitrous or sulphurous, is exceedingly irritating to the air passages, and not infrequently sets up acute bronchitis in workmen whose occupation expose them to it. But, according to Hirt, the chronic form of the disease is comparatively seldom traceable to this cause. After one or two acute attacks a tolerance seems to be established, and no further ill-effects are observed. On the other hand, he speaks of the emanations from certain oil works, from tar factories, and from the pans in which brine is evaporated to make salt, as having a beneficial influence on the bronchial mucous membrane.

The *treatment* required for bronchitis varies widely in different forms and in different stages of the disease. It is not easy, in text books, to lay down rules for it.

In the milder forms of acute *bronchial catarrh*—such as are often known under the name of tracheo-bronchitis—little is necessary beyond placing the patient in an equable temperature, which should be at about 63°. Small doses of ipecacuanha may perhaps be serviceable, by favoring exudation from the inflamed mucous surface. The application of mustard poultices, or of hot flannels sprinkled with turpentine, to the throat and to the upper part of the chest often gives great relief to the sense of soreness along the trachea and behind the sternum.

Very different measures are necessary in some cases of *capillary bronchitis*. Sometimes, if suffocation appears to be rapidly impending, it is advisable, when the patient is an adult of robust constitution, to bleed from the arm freely. Antimony is often the best medicine, and for a day or two it may be given in considerable doses, so as to have a decidedly nauseant effect. Another drug which may be very successful is lobelia, the ethereal tincture of which is prescribed in half-drachm or even in drachm doses at frequent intervals. There are some cases, on the other hand, in which all depressing remedies are obviously unsuitable; and for the more dangerous among them, the administration of turpentine sometimes affords the best chance of arresting the fatal issue which is obviously near at hand. In one of the very worst cases which I ever had to treat, life appeared to be saved by alternate doses of turpentine and of champagne.

It is very important to maintain a moist state of the air round the patient. A kettle on the fire, with a long tube throwing steam out near the patient's bed, fulfils this indication better than anything else. But when it is used, one must never forget that the ceiling and the curtains necessarily become saturated with damp, and that if the temperature of the room should be allowed to fall a few degrees during the night or in the early morning, a chill will result which may probably be fatal to the patient. In some cases the inhalation of steam gives much relief, or a medicated spray may be employed, containing conium juice, or morphia, or salines, such as chlorate of potass. Large poultices are commonly placed round the whole chest, from front to back; and mustard or turpentine is applied until the surface is thoroughly reddened.

After a few days it is generally necessary to substitute for nauseant

drugs, like ipecacuanha or antimony, such remedies as carbonate of ammonia, squill and senega. In the bronchitis or broncho-pneumonia of young children, these are usually prescribed from a very early period.

In *chronic bronchitis* a great variety of medicines are useful; the difficulty is to formulate rules for selecting one rather than another of them. If the cough is dry and hard, ipecacuanha is especially serviceable. If it is distressing by its frequency, and apparent aimlessness, bromide of ammonium, or of potassium, often gives relief. In such circumstances morphia or opium may often be given with great advantage. After a few hours' sleep the patient may wake greatly refreshed and in all respects better. It is, however, always necessary to consider, before prescribing opiates in a case of bronchitis, whether one is likely to do harm by checking cough, and so preventing the tubes being emptied of their contents. If, with lividity, there is stupor, or even drowsiness, such medicines must be carefully avoided. In many cases, especially if the expectoration is viscid and abundant, sal ammoniac is very useful; it may be given in doses of gr. xv to gr. xx, with a little syrup of lemon or extract of licorice to conceal its disagreeable taste. Iodide of potassium is another salt which often does good service in chronic bronchitis. In cases in which there is excessive exudation and secretion from the surface of the mucous membrane, balsamic remedies are applicable; tolu, benzoin, Peruvian balsam, benzoic acid itself. Or, again, one may prescribe copaiba, turpentine, ammoniacum, or one of the fetid gum resins, such as assafœtida.

Of late years, several physicians, in Germany especially, have made large use of compressed air, and also of rarefied air, in the treatment of various bronchial and pulmonary affections. In the earlier attempts recourse was had to pneumatic chambers, made somewhat after the fashion of the diving bell, in which the patients sat for an hour at a time, under a pressure of $1\frac{2}{5}$ to $1\frac{3}{7}$ atmospheres. It is obviously in very exceptional circumstances only that such elaborate constructions can be available in practice; and, therefore, attention has been more recently devoted chiefly to the invention of portable forms of apparatus, by which the patient is made to inhale air of varying degrees of pressure without being himself immersed in it. Most of these instruments are upon the principle of the ordinary gasometer used to receive coal gas at gasworks; an air-containing cylinder, open below, is suspended in a cylinder, open above, so that the one can move freely up and down within the other. By pouring more or less of water into the outer cylinder, and then either pressing down the inner cylinder with weights or lifting it up to varying heights, the air inside it may be compressed or it may be rarefied to any desired extent. The object is to make the patient *inspire* compressed air, or *expire* into rarefied air. A tube from the inner cylinder is connected with a mask, which can be fitted air tight over the nose and the mouth. There is a stop cock, which is turned by the patient each time he breathes, so that the mask communicates with the cylinder either during inspiration or during expiration (as may be intended), whereas on reversing the movement it communicates with the external air. Waldenberg, who invented this machine, usually directs that compressed air should be inspired for five, ten or fifteen minutes; and then, after a pause, that expiration into rarefied air should be practiced for a similar period. The range of pressure variations employed is but small, seldom exceeding $\frac{1}{40}$ m. or even $\frac{1}{30}$ m. of an atmosphere, and being often not more than $\frac{1}{60}$ m. or even $\frac{1}{120}$ m. In most cases, two or three sittings a day are sufficient. It is obvious that so far as emphysema is concerned, the greatest degree of benefit is to be anticipated from expiration into rarefied air. The inspiration of compressed air, when the whole body is not immersed in a pneumatic chamber, might be expected to tend rather toward increasing the distention of the pulmonary alveoli.

But, on the other hand, it is said that expiration into rarefied air may, in its turn, do harm by augmenting the flow of blood to the bronchial mucous membrane, whereas inspiration of compressed air has the effect of increasing the blood pressure in the systemic vessels, and of unloading the pulmonary vessels and the right side of the heart. It is, therefore, best to alternate the two methods, as Waldenberg and others advise. And there is very strong testimony of the beneficial action of this mode of treatment, in augmenting (at least for a time) the physiological activity of emphysematous lungs, and in relieving all the symptoms of bronchial catarrh.

In some cases of chronic bronchitis, recourse may be had with advantage to certain continental spas; according to Braun, those waters are the best which contain a considerable amount of chloride of sodium as well as of carbonate of soda. Ems in Germany and Mont Dore in France may be specially mentioned.

When it is considered desirable for a bronchitic patient to spend the winter and spring away from home, the choice lies usually between climates which are soft and "sedative" (such as Torquay, Penzance, Pau, Madeira), those which are stimulant without too much risk of exposure to cold winds (of the health resorts on the Corniche Road, Mentone and San Remo appear to be, on the whole, preferable to the rest), and those which are surrounded by pine woods (such as Arcachon and Bournemouth).

PNEUMONIA

HISTORY—ANATOMY AND HISTOLOGY: STAGES, EVENTS, LOCALITY—PHYSICAL SIGNS—CLINICAL SYMPTOMS—ORIGIN AND NATURE OF ACUTE PNEUMONIA—PROGNOSIS—TREATMENT.

Nomenclature.—Although the term "peripneumonia" dates back to the days of Hippocrates, it is only since the discovery of auscultation, and as a result of the systematic study of pathological anatomy, that a definite meaning has been attached to it in relation to other thoracic diseases. By *pneumonia* (the prefix, peri, having been dropped) is now understood an inflammation of the alveolar texture of the lungs. But the disease which I am about to describe is far from including all that the morbid anatomist recognizes under the same name. Not only has that form of pneumonia which arises by extension from the terminations of the bronchial tubes to be excluded, but also many other varieties which occur as the result of infective embolism, of the penetration of foreign bodies into the air passages, and of other morbid conditions. It is a question, too, whether the pneumonia which follows injuries to the chest, that which complicates affections of the heart, and that which so often forms the immediate cause of death in persons suffering from almost any chronic or acute disease may not each require to be placed under separate heads. In strictness, some addition to the name is necessary to distinguish from other kinds of pneumonia that with which we are now concerned. Consequently the Germans have adopted the designation of "croupous pneumonia," because the exudation mainly consists of a fibrinous material like that which characterizes croup. To English ears, however, this at present has an awkward sound. And it is not likely to cause any real confusion if I still often speak of "pneumonia" without any such qualifying epithet; for whenever a patient (except, indeed, in childhood) is said to be suffering from "pneumonia," one always understands that croupous pneumonia is meant. In this country it was at one time usual to call the disease "lobar" pneumonia, by way of distinction from "lobular pneumonia," or the bronchopneumonia so common in children. But to this, again, there are the objections that neither is the whole of a lobe always affected, nor is the morbid process always limited to one lobe.

Morbid Anatomy.—The pneumonic process consists of a series of changes by which the spongy pulmonary tissue is rapidly converted into a solid mass, returning afterward, in cases that recover, to its normal condition. Systematic writers describe several distinct stages.

The first is the stage of "congestion" or "engorgement." During this the affected part of the lung is massive, heavy, of a reddish-brown color. It pits under the pressure of the finger, and a reddish, frothy serum oozes from its cut surface; when the pressure is increased, its substance breaks down much more readily than that of healthy lung. Microscopically, the most obvious appearance is the dilated and tortuous state of the capillaries of the alveolar walls; minute punctiform hemorrhages are also to be observed,

especially in the connective tissue between the lobules and beneath the pleura.

Next comes the stage of "red hepatization," so called because the texture of the lung is solid, like liver. It now sinks in water, it does not crepitate when pressed between the finger and the thumb, it is very soft and lacerable, and little or no fluid can be squeezed from it. Its cut surface has a dull and lustreless appearance, and it is distinctly granular. The granules are composed of a solid inflammatory exudation, which completely fills up both alveoli and infundibula. Rindfleisch gives drawings of masses of it obtained by scraping the cut surface; they form complete casts of the interior of the spaces in which they were moulded. The red color of the lung at this period is probably due partly to the large quantity of blood in its capillaries, partly to the fact that great numbers of red discs are extravasated and mixed up with the exudation. The exudation itself consists mainly of coagulated fibrin, in the meshes of which are also seen more or less numerous leucocytes.

The third stage is that of "gray hepatization." This is characterized not only by a change in the color of the affected parts of the lung substance, which now pass through reddish gray to gray or even whitish yellow, but by becoming even softer than before, by their being less markedly granular on section, and by their beginning to emit on pressure a more or less turbid fluid, which may be opaque, white and puriform. The extreme forms of gray hepatization are, in fact, described by some pathologists as constituting a fourth stage, which they term "purulent infiltration." Histologically there is a wide difference between the characters of "red" and those of "gray hepatization." In the gray stage no fibrinous coagulum is visible; the substance which fills the alveoli now appears to consist merely of a mass of crowded leucocytes. The extravasated red discs, too, are no longer to be seen. Rindfleisch speaks of them as becoming decolorized; perhaps it is fair to suppose that they are absorbed by the rapidly multiplying leucocytes, as suggested at p. 67. To account for the change of color, it must also be assumed that the increased amount of exudation compresses the pulmonary capillaries and drives the blood out of them. But Rindfleisch remarks that it would be a great mistake to imagine that this occurs during life to the same extent as after death. He finds that in the dead body it is always easy to inject the vessels, and he, therefore, concludes that the heart, so long as it is beating, must be able to keep up a more or less active circulation through them. In other words, it is probable that the gray appearance is, strictly speaking, a cadaveric change. A very important histological distinction between the two kinds of hepatization, however, is afforded by the state of the alveolar walls. In the "red" stage they are unaltered, except that their capillaries are distended; in the "gray" stage they are infiltrated with leucocytes, which fill up every interstice.

Events.—The formation of an *abscess* in the lung tissue, as the result of true pneumonia, is admitted by all writers to be very rare. So far as my own observations have gone, I am quite disposed to agree with those who doubt whether it ever occurs. Cases have, indeed, been recorded: but the question is whether an accurate pathology might not have led to a different interpretation of them. I shall discuss elsewhere the termination of this disease in *gangrene*, which is also infrequent, but which is undoubtedly sometimes seen.

But pneumonia often ends in *resolution*. And in such cases an interesting question presents itself, namely, whether it is possible for the disease to pass through all its three stages and yet afterward to undergo recovery. Now, it is certain that subsidence not infrequently occurs at quite an early period, before there is reason to suppose that any part of the lung

has passed beyond the stage of engorgement. But it is equally certain that many other cases end favorably after there has been clear evidence from physical signs that the pulmonary tissue has become "consolidated," or (in other words) has reached the stage of red hepatization. The doubt is whether the occurrence of gray hepatization or of purulent infiltration is compatible with recovery. In their work on Pathological Anatomy, Wilks and Moxon express a rather decided opinion that in most cases the disease does not progress much beyond the "red" stage before resolution begins. An opposite view seems to be generally entertained, chiefly in consequence of theoretical conceptions as to the nature of the process by which resolution is effected. There are obviously two ways in which the pulmonary alveoli can be emptied of the exudation that fills them; one is by its escaping into the air passages and being expectorated; the other by its being reabsorbed into the blood. Now, Rindfleisch maintains that most of it takes the former course; but I think that every clinical physician will agree with Jürgensen (in Ziemssen's "Handbuch") that he is mistaken, inasmuch as in many cases sputum is altogether wanting at this period of the disease. However, before absorption can occur, it is generally supposed that the exudation must liquefy and undergo a change more or less analogous to that of fatty degeneration. And the assumption has been that this is equivalent to the conversion of red hepatization into gray. But that such an assumption is erroneous is apparent from the description given above of the histology of the more advanced stage of the disease, which we have seen to be attended not merely with softening of the inflammatory products, but also with a greatly increased infiltration of leucocytes. And I am disposed to agree with those who think that in all probability, when this stage is reached, recovery is no longer possible. After the subsidence of pneumonia, if the patient should die, at no long interval, from some other disease, the lung is found to have nearly regained its healthy appearance, but to be slightly redder and tougher than natural. I have notes of two such cases observed at Guy's Hospital, but unfortunately not of any microscopical investigations as to the exact state of the pulmonary tissue. Rindfleisch speaks of a "loss of elasticity" as resulting from pneumonia, and as continuing a long time after recovery has taken place.

It is a very important question whether, instead of subsiding, croupous pneumonia ever leads to permanent changes in the lung, to the development of fibrous tissue in it, constituting what is termed cirrhosis or chronic pneumonia, or to a destructive process, ending in the formation of cavities more or less like those that are seen in phthisis. Now, as regards cirrhosis, though some observers (including Wilks) are opposed to the belief that it ever arises in this way, I shall hereafter cite cases which seem conclusively to prove that such a result does sometimes occur. But as for the supposed termination of the disease in phthisis, I must confess that I have always held to the opinion that the cases that have been so interpreted were phthisical from the beginning.

Complications.—I have just alluded to the occurrence of pleurisy as a complication of pneumonia. The fact is that whenever the inflammation reaches the surface of the lung the corresponding part of the pleura always becomes covered with lymph. Accordingly, some physicians always speak of the disease as "pleuro-pneumonia," at least in those cases in which they discover physical evidence of pleurisy during life. But this seems to be an unnecessary refinement, and indeed to be rather misleading, since on post-mortem examination lymph is found upon the pleura, even when there had been no signs of its presence. Probably it is best to reserve the term pleuro-pneumonia for cases in which the pleurisy leads to fluid effusion, and so becomes of clinical importance. Not infrequently there is likewise pericarditis, this

having either arisen by extension from the pleura, or having developed itself simultaneously in the serous membranes covering the heart and the lung, as well as in the lung itself. Sometimes the peritoneum, especially the upper part of it, becomes coated with lymph. Sometimes the mediastinal tissues are extensively infiltrated with a gelatinous exudation. The bronchial glands are constantly found to be greatly enlarged, pinkish-gray, and soft; and sometimes the sub-pleural lymphatics corresponding with the seat of the pneumonia are seen to be distended with inflammatory products, giving a marbled appearance to the surface.

As an occasional addition to the pulmonary lesion must be mentioned acute meningitis; how it arises is not very clear. A less infrequent complication is ulceration of the larynx, the ulcers being placed over the arytenoid cartilages, just where ulcers are apt to occur in so many other circumstances. Probably it is to the pyrexia which accompanies pneumonia that one should refer some other morbid changes that are met with in the post-mortem room, such as a moderate degree of enlargement of the spleen, catarrh of the intestine, and catarrh of the kidneys.

Like almost every other disease of the pulmonary alveoli, pneumonia attacks parts of the lungs only, and never the whole of them. Nor does it generally affect even an entire lobe at once, but almost always begins at some one spot, from which it rapidly spreads to others. All observers are agreed that the *right* lung is more often the seat of pneumonia than the left, the proportion being about as five to three. Sometimes both lungs are attacked together or in succession. On either side the *lower lobe* is affected far more frequently than any other part; Jürgensen says that it escapes altogether only in one case out of four. Very often the inflammation begins at the extreme base, and extends gradually upward from day to day. But sometimes it spreads downward from the summit of the lower lobe, or upward and downward from its middle, or backward from the anterior border. In the upper lobe it may either pass from the apex downward or from some other point in a different direction. Sometimes its distribution remains strictly limited by the lobar septa; sometimes its spreading edge forms a horizontal line, ignoring them altogether.

Signs.—It is by physical examination alone that the seat and extent of pneumonia can from day to day be determined with any degree of accuracy, although the other symptoms commonly enable one to form a confident opinion both as to the nature of the disease and as to the part of the lung in which it is most likely to be found. The earliest signs are generally recognized by auscultation. According to Stokes, there may first be a peculiar harshness of the vesicular murmur. But, as a rule, the first thing to be detected is an entirely new or adventitious sound, which is called "crepitation" or sometimes "fine crepitation." This is exactly like the sound produced by rubbing a lock of hair between the fingers close to one's ear. By Laennec, who discovered it, it was supposed to be pathognomonic of pneumonia. But it is now known to occur likewise in œdema of the lungs, and perhaps in catarrh of the smallest bronchial tubes; and a sound very like it, if not absolutely the same, may often be heard for successive inspirations in the bases of the lungs of a person who has been lying on the back with some febrile disease, if he is made to sit up and breathe deeply, so as to fill those parts which have been for some time disused. In the last case there can be no doubt that the cause of the sound is the opening up of portions of the tissue that had become collapsed; and almost all observers are now agreed that in pneumonia it has a similar origin, being due to the inspiratory separation of the walls of alveoli and bronchioles which, being swollen, had come into contact in expiration. Crepitation is, indeed, heard only during inspiration, and sometimes only just at the end

of deep inspiration, as after coughing. One must, therefore, carefully search for it before concluding that it is not present. But, on the other hand, the fact must be admitted that in many cases of pneumonia this sign is not at any time discovered. Whether it is ever really altogether absent in such cases is, perhaps, doubtful, because it is in its nature transitory, and so may have passed off before an examination of the chest was made. Sometimes, however, it remains audible during almost the whole length of the disease; not, indeed, at the same spot, for where there is complete consolidation it almost necessarily disappears; but in one spot after another, as they are successively attacked by the inflammatory process.

As the state of engorgement passes on to that of red hepatization, there are developed other signs, the chief of which may be briefly summed up under the heads of dullness on percussion, bronchial breathing, and bronchophony with increased tactile vibration. The degree of dullness varies much in different cases. There is never the absolute tonelessness that is met with in cases of fluid effusion into the pleura; nor is the sense of resistance to the finger so great. Sometimes, however, the percussion sound undergoes very curious modifications, the explanation of which is by no means obvious. Thus, a cracked-pot sound is in some cases elicited; this is attributed by Dr. Gee to the presence of "islets of unsolidified lung embedded in the surface of the hepatized tissue." In other cases the sound has a more or less markedly tympanitic quality, or is even a sonorous note like that which belongs to a pneumothorax. The only way of accounting for this that I see is by supposing that the chest wall is enabled to vibrate more freely by the removal of the negative pressure exerted by healthy lung upon its under surface. Sometimes the spot where the tympanitic sound is heard is not directly over the consolidated part of the lung but in its neighborhood, over pulmonary tissue which may be imagined to be relaxed as the result of the increased bulk of the consolidated part. Thus hepatization of the upper lobe of the lung behind may lead to the occurrence of a tympanitic percussion note beneath the clavicle on the same side. The bronchial breathing that accompanies the second stage of pneumonia may exhibit every variety of quality up to the amphoric. But sometimes no such sounds are audible, and the explanation doubtless is that the tubes passing to the consolidated part happen to be filled with fibrinous plugs. Bronchophony generally runs *pari passu* with bronchial breathing and presents like differences of degree. Dr. Gee, however, remarks that in infants a bronchophonic cry is often the only auscultatory sign that can be obtained. As for increase of vocal fremitus, this, although sometimes well marked, is by no means constantly present. Probably its occurrence depends upon the state of the tubes leading to the consolidated part. If they are full, even of fluid secretion, the transmission downward of the coarser vibrations contained in the voice is interrupted. It is only when the left side is the one on which the fremitus is greater that this sign can be regarded as of clinical importance, because on the right side it is very often greater in healthy persons.

It remains to be mentioned that when pneumonia affects only the central part of the lung, or reaches no part of the surface except that which is in contact with the diaphragm, physical signs are altogether absent. What is more common is for the disease to begin deeply in the interior, but to become superficial a few days later, so that all doubt as to the nature of the case is soon at an end.

During the stage of resolution, the auscultatory signs of hepatization disappear, and they are commonly replaced by râles, which may vary widely in character. Sometimes a crepitation is now heard which may be scarcely less fine than that of the early period of the disease; this is spoken of as "redux crepitation." In other cases the sounds are so large, and at

the same time so highly consonating in quality, that one might imagine the patient to be at an advanced period of phthisis, with the lung breaking up in all directions. Yet, after a few days, these sounds, in their turn, cease to be audible. A considerable time generally passes before the vesicular murmur becomes as loud and as distinct as it normally should be. The percussion resonance, also, may long remain deficient, but chiefly in cases of which a marked feature has been concomitant pleurisy.

Clinical Course.—An attack of pneumonia commonly sets in suddenly with a well-marked rigor. Sometimes, however, there is not more than a sensation of chilliness, to which the patient may attach but little importance. Sometimes the disease is ushered in by a convulsive seizure, or by vomiting, which may be once or twice repeated. Occasionally its onset is insidious, there being no symptoms to mark the exact time at which it begins. The development of the pyrexia is very rapid. Jürgensen cites a number of observations which show that the temperature may reach 104°, or even a higher point, within three or four hours from the commencement of the shivering. At the same time the pulse is quickened and it becomes full and bounding in character. There are the usual complaints of malaise, headache, pains in the limbs, anorexia, as in every other febrile disease. As the rigor passes off the cheeks acquire a crimson flush. A point on which Addison used to lay great stress is that, as tested by the hand, there is in pneumonia a pungent heat of the skin, which is observed in hardly any other affection. I have shown, at p. 58, how this has since been confirmed by the observations of Schülein.

So far there is nothing in the symptoms to show that the thoracic organs are the seat of the patient's illness. And formerly it was not uncommon for the diagnosis to be given as "typhus" or "continued fever," and for the autopsy to show for the first time the real nature of the case. Or if, as sometimes happens, there was violent delirium from the very commencement, the patient becoming comatose and dying in twenty-four or thirty-six hours, the disease was set down to "meningitis" or "encephalitis." But, in reality, a careful observer will seldom, if ever, fail to notice indications that will lead him to thoroughly examine the lungs, and consequently to form a correct judgment. One point of great importance, upon which Dr. Walshe has especially insisted, is the change in the pulse-respiration ratio, *i. e.*, in the relative frequency of the movements of the heart and of the lungs. Healthy persons breathe once for every four or four and a half pulsations of the heart. In febrile diseases in general, both pulse and respiration are more frequent, but the relation between them is not much, if at all, disturbed. But in pneumonia the increased frequency of the respiration is out of all proportion to that of the pulse, the ratio being as one to three or one to two, or higher still; for the patient may breathe sixty or eighty times a minute. Dr. Walshe has observed the number of respirations in the minute actually greater than that of the heart-beats. Jürgensen says that the cases in which this occurs are those of old people with slowly-acting hearts and atheromatous vessels. A curious circumstance, to which Dr. Walshe has especially drawn attention, is that the amount of subjected distress produced by such rapidity of breathing varies extremely in different cases; some patients breathing thirty, forty, or even sixty times a minute are wholly unconscious of any dyspnœa. Another indication that should draw attention to the respiratory organs as the probable seat of the disease is, that the nostrils work during inspiration, and that the *sterno-mastoidei* and the *trapezii* muscles are tense and prominent. It may also be observed that the flushed cheeks and the lips have a slightly purplish tint.

But in many cases there are present from an early period, or from

the very beginning, symptoms which point clearly to the real nature of the disease, and even show in what part of the chest it is to be found. One such symptom is *pain*, which is commonly situated near the nipple or toward the axilla, and which may be the first thing to suggest to the patient that he is otherwise than well. This pain is identical with the *point de côte* which will be described as occurring in pleurisy. And in all probability, in cases of pneumonia, it is really due to the coincident pleurisy, as Addison long ago taught. It is generally urged, as a reason for adopting this opinion, that in many cases of pneumonia there is no pain whatever. But that obviously proves nothing, unless it can be shown that in such cases there is also no pleurisy; and I am not aware that this has been attempted. Indeed, pleurisy itself is sometimes painless. Sometimes the pain does not appear until the patient has been ill some hours, or not until the second day, or even later. It is exceedingly severe, and being increased by every movement, it leads the patient to endeavor to fix the ribs by pressure with his hand, and also causes him to curve his spine toward that side, so as to bring the ribs as close to one another as possible. Sometimes the act of drinking produces so much suffering that the patient will endure the great thirst produced by the disease rather than attempt to swallow. The *cough*, which is another early symptom, is also modified by the pain, which interrupts it and, as far as possible, cuts it short. The distress caused by the cough is greatly increased by the fact that the *expectoration* in pneumonia, though very scanty, is extraordinarily tenacious and viscid, so as to be got rid of with extreme difficulty. Even when it has reached the lips, it often clings to them, so that it can only be removed by the handkerchief. And it adheres equally firmly to the spit jar, which may be inverted without any of it escaping. Pneumonic sputum has also a peculiar color, due to the circumstance that the frothy mucus of which it mainly consists is intimately mixed with blood that has, perhaps, undergone slight chemical changes during the time that has elapsed since its extravasation from the vessels. This color varies in its shade in different cases. It is usually bright orange, tawny, or like the rust of iron, so that to speak of "rusty" expectoration is common. But sometimes the tint is a paler apple yellow, and sometimes it is the bright scarlet tint of unaltered blood. Spitting of pure blood in such quantity as to deserve the name of hæmoptysis is uncommon. Remak, some years ago, pointed out that in pneumonic sputum there can sometimes be detected with the microscope branching fibrinous casts of the smallest bronchial tubes. I am not aware that the observation has hitherto been made practically useful, though in a doubtful case it might certainly serve to show what was the real nature of the disease. For it is a mistake to suppose that the occurrence of viscid, rusty sputum is in itself pathognomonic of pneumonia. I have now and then seen similar sputum in other cases, though I can hardly say of what nature these were. But some patients with pneumonia have no cough, and therefore no expectoration, throughout the whole illness. In some cases the sputum is thin, watery, and of a brownish-red color, so that it is compared with prune juice. Such sputum is seen chiefly at advanced stages of the disease and when it is taking an unfavorable course. But it may be present at an early period, and in cases that ultimately do well. Probably, as Dr. Wilson Fox suggests, it really comes from a part of the lung affected with œdema.

Having thus developed itself, pneumonia commonly runs on for some days with but little variation in its symptoms. As more and more of the pulmonary tissue undergoes hepatization, the physical signs become more and more marked, but otherwise the case often seems to show no proportionate increase in its severity. The type of the *pyrexia* is usually continuous,

with the usual diurnal oscillations. Wunderlich, however, points out that it is not uncommon for an irregular brusque elevation of temperature to occur, which is followed by a no less sudden fall through as many as 7° or even 9° F. to the normal point or below it, and that again in a few hours by a fresh elevation. I have observed more than one case in which such strange deviations in the regular course of the fever took place again and again, and, in fact, constituted the greater part of the temperature chart, without its being possible to find any explanation of them. After the first few days the *skin* is, in many cases, moist, and there may be profuse sweating. The *pulse* may gain in frequency from day to day, but sometimes it remains for several successive days at 90 or 100, the only change in it being that it becomes gradually smaller and softer, and even dicrotic in character. In adults there is always ground for alarm if it rises to 120, but in young children it may reach 130 to 140 without there being any danger; in old people it commonly stands at a much lower point. A symptom, which usually makes its appearance between the second and the fifth day, is an eruption of *herpes* upon the lips, and sometimes at the anus, or even upon the limbs. It is said to occur in from two-fifths to one-half of all cases of pneumonia; and what is very strange is, that it is far more common in cases that do well than in those that end fatally. This has long been a traditional opinion, and it seems to have been established by the observations of Geissler, who found ("*Arch. d. Heilkunde*," 1861) that out of 421 cases the mortality among those in which there was no herpes was at the rate of 29.3 per cent., whereas among those with herpes it was only at that of 9.3 per cent. The *tongue* is, of course, furred in pneumonia; as the disease advances, it may become dry and brown and covered with sordes. The bowels are generally constipated, but sometimes there is diarrhœa. An icteric tinge of the conjunctivæ is not uncommonly present, and sometimes there is well-marked *jaundice* of the skin. Formerly it was taught that this occurred only when the base of the right lung was the seat of the disease, and that it was due to the extension of inflammation through the diaphragm to the liver substance. But the truth is, that sometimes the pneumonia affects the upper lobe of the right lung or some part of the left lung. And it would seem that the explanation must be that the jaundice is due either to venous congestion of the liver transmitted from the lung through the right side of the heart, or to a catarrh of the common bile duct accidentally present as a complication. The *urine* in pneumonia is scanty, of high specific gravity, high colored, strongly acid, depositing urates in abundance. The amount of *chlorides* excreted by the kidneys undergoes a great diminution; they may even be altogether absent. At one time it was thought that this was of considerable diagnostic importance. But the same thing occurs in almost all other febrile diseases likewise, the explanation being, apparently, that the salts in question are retained in the body, for it cannot be supposed that the supply of them in the food is lowered to such a point as to account for so great a difference. Not infrequently the urine contains *albumen* in moderate quantity.

Many patients with pneumonia retain their consciousness throughout the whole of their illness, or merely wander a little at night during their broken sleep. But in other patients *delirium* is a very prominent symptom, and it may be of a furious maniacal type. This is said to be more often the case where the upper than when the lower lobe is the seat of the disease. In persons who have been intemperate, pneumonia is often attended with symptoms exactly like those of delirium tremens, which affection may, indeed, be said to be present as a complication. Another thing that seems to determine the occurrence of severe cerebral symptoms in pneumonia is an inherited neurotic tendency; in one of the most terrible

cases I ever saw, that of a man whose last hours were passed in a paroxysm of raging madness, I was told that there was hardly one of the brothers and sisters of the patient who had not suffered from one form or another of nervous disease.

Prognosis.—The frequency of a fatal termination in pneumonia is, on the whole, far less than might have been anticipated. But to state with accuracy what is the average death rate in this disease is at present impossible. One difficulty is that it varies enormously, according as primary cases are more or less numerous in proportion to those which are secondary to some other affection, and which are often obviously hopeless from the very first. Then, again, the danger of pneumonia is widely different at different ages —children almost always recover (it must be remembered that we are now concerned only with "croupous" pneumonia). Again, in young healthy adults, of temperate habits, recovery is the rule; Jürgensen gives the death rate in such cases as from 3 to 6 per cent. But to old people, or to those who (though not old) are worn out by overwork, dissipation or drunkenness, the disease is exceedingly fatal; though from time to time one sees a patient recover even when the circumstances have appeared most adverse. In an individual case, if the symptoms are severe danger is indicated; or, again, if the local lesions are extensive, and especially if both lungs are affected. But experience has taught me that it is never safe to speak confidently of the prospect of recovery of a patient with pneumonia, however favorable its course may be during the first few days. Suddenly a change for the worse is apt to occur; the pulse, which may have been of moderate frequency hitherto, runs up rapidly, and in a few hours the end may come. In all probability the cause of the fatal issue in such cases is the supervention of an inflammatory œdema in parts of the lungs that had previously been unaffected by the disease. Other cases go steadily downward from their very commencement. However this may be, the symptoms which precede death are generally the same. The breathing becomes more and more rapid, and it is shallow and superficial, the patient being no longer conscious of the necessity for filling his lungs, as is shown by his sinking down in bed away from the pillows by which he had been propped up. The face is intensely livid and pale, so that it may be described as slate colored. The skin is covered with a cold sweat. The pulse becomes weak, irregular, and finally imperceptible. But in certain cases death is sudden, the heart failing during some effort, as when the patient raises himself up in bed. Lastly, it now and then happens that the occurrence of the crisis (which will be described in the next paragraph) is followed by great prostration and collapse, which end fatally.

In cases of pneumonia that end in recovery, the subsidence of the pyrexia and of the other symptoms commonly takes place abruptly, *by crisis*. And in about sixty-five per cent., when the exact duration of the disease can be reckoned from there having been an initial rigor, or convulsive seizure, or attack of vomiting, the crisis is found to occur between the fifth and the eighth days. In some cases it is even earlier; in others it is delayed until some time in the course of the second week. Sometimes fever continues longer even than this; but I agree with Jürgensen in thinking that whenever defervescence fails to occur by the fourteenth day there is ground for suspicion that the case is either not one of croupous pneumonia in a strict sense, or else that some complication or other is present. It almost always happens that the crisis begins in the evening or night, scarcely ever in the forenoon. The fall of the temperature is usually rapid, being completed in about sixteen hours, or sometimes even in five or six hours; but in some cases from twenty-four to thirty-six hours pass before the thermometer reaches 98.5°. For the next two or three days it often stands below the

normal point; the lowest reading observed by Jürgensen during this time was 95.5° (in the rectum). Sometimes, however, the defervescence in pneumonia takes place gradually, or *by lysis*, occupying two or three days. In cases which end by crisis, it is surprising how much better the patient feels as soon as the pyrexia has subsided. His appetite quickly returns, he sleeps well, his skin perspires comfortably. Both the pulse and the respiration decline *pari passu* with the temperature. Yet the physical signs may at first fail to indicate any improvement in the state of the affected lung, and several days may pass before the consolidation can be said to have cleared up to any great extent.

Pathology.—With regard to the nature of pneumonia, and its relation to other affections, there are two opinions. The most obvious view is to look upon it as an inflammation attended with pyrexia, like other severe inflammations. Against this, however, many objections may be urged. Some of them are based upon what is known as to the ætiology of the disease. "Catching cold" is commonly supposed to be the principal exciting cause of pneumonia, as it undoubtedly is of pleurisy and of bronchitis. There is no physician who cannot recall to his memory numerous instances in which patients were attacked immediately after getting wet through, or after lying on damp grass, or after some other very definite exposure to cold; there being no doubt as to the part played by such circumstances in bringing on the illness. But writers who have tabulated their cases, with the object of determining the frequency of the operation of cold as a cause of the disease, have failed to trace it in any but a comparatively small proportion of them. Among 205 cases collected by Grisolle, 45 were, indeed, supposed to be due to cold; but among 186 cases of Ziemssen's only 10, and among 212 cases of Griesinger's only 4 could be clearly attributed to this cause. (These figures I take from Jürgensen's article in Ziemssen's "Handbuch.") Again, in the relations of pneumonia and of bronchitis respectively to different climates and to different seasons of the year, there appear to be certain differences that would hardly have been anticipated on the view that the two diseases have the same ætiology. Pneumonia does not, like bronchitis, increase in frequency with the latitude, from the equator toward the poles. And as for its occurrence at various seasons of the year, observations made at Vienna have shown that whereas in that city the prevalence of bronchitis reaches its maximum in March and then gradually declines through the rest of the spring and summer, the prevalence of pneumonia increases steadily from February to May, after which it falls rapidly. This last part must not, indeed, be taken as applying to other places besides Vienna, still less to Europe generally; for Jürgensen points out that there is a broad difference between continental and insular climates as regards the months in which pneumonia is most apt to occur; in the former it is between March and May, in the latter between December and February. But if the year be divided into two halves, the one from December to May, the other from June to November, then it is found that in whatever part of Europe, two-thirds of the cases of the pneumonia fall into the first half, one-third into the second. Another point which tends to confirm the opinion that pneumonia is not generally directly due to cold is that sailors and others whose occupations expose them to bad weather do not seem especially prone to be attacked. Again, Jürgensen maintains that the disease is not capable of being set up by local injuries to the chest, wounds of the lungs, foreign bodies in the bronchi, or any like causes. Of course, he does not deny that any of them may give rise to inflammation of the pulmonary tissue, but his contention is that this has not the characters of true "croupous" pneumonia. He also declares that it is impossible to induce that affection experimentally in animals. Sommerbrodt had stated that he saw it follow the injection of

a solution of perchloride of iron into the air passages; Jürgensen repeated the experiment, but says that the result was not the same.

Different opinions have been expressed as to whether pneumonia is more apt to occur in persons who are strong and healthy, or in those who are weak and delicate. There is no difficulty in finding instances in support of either view, but although one cannot help being profoundly impressed by the cases that one now and then sees, of vigorous young men carried suddenly off by this disease, I believe that they are, after all, exceptions. Jürgensen insists on its frequency in prisoners, and in those who are worn down by wasting maladies, such as cancer or diabetes. It is met with at all ages and in both sexes, in males rather more often than in females.

I cannot say that the facts above stated in regard to the ætiology of pneumonia appear to me to go very far toward disproving the common opinion that it is essentially a local inflammation accompanied by a symptomatic pyrexia. Jürgensen, however, attaches importance to them as tending to show that it properly belongs to the group of infective diseases, and is due to a specific exciting cause. His chief other reasons for this view are (1) that throughout its course there is no constant relation between the local and the febrile symptoms, and (2) that its course is "typical." Contagious it certainly is not, but indications are not altogether wanting of it sometimes prevailing epidemically. Instances have been recorded of its attacking in turn different inmates of the same house, and in certain years it has been far more frequent than usual.* But the same things may be said of many other affections, such as jaundice, the local character of which remains undisputed.

The truth seems to be that the question of the real pathology of pneumonia is involved in a much wider one, to which at present no positive answer can be given. We have seen, in the chapter on Inflammation, that many modern observers believe that no form of spreading inflammation is due merely to the reaction of the organism against a local injury. If this be the case, some specific exciting cause, such as Jürgensen assumes for pneumonia, must be supposed to be present in a vast number of other inflammatory diseases. Probably many years will pass before the true relations of all of them are fully understood. In the meantime it would not be really advancing in the right direction to detach pneumonia from other thoracic affections, with some of which it may after all prove to be closely allied, or to force it into a close relation with the group of acute specific diseases as now understood, from which it certainly differs in many important particulars.†

Recurrence.—A person who has once suffered from pneumonia is afterward more likely to be attacked by the disease than if he had not already had it. Instances have been recorded in which it has recurred eight, ten, and even twenty-eight times. Relapses are not frequent, if by this term we understand the repetition of the morbid process before the patient has completely recovered from a former attack; when such a thing happens it probably ought to be regarded rather as an independent illness, due to the renewed operation of the exciting cause upon a system now

* The most detailed observations that I am acquainted with, as tending to establish the epidemic occurrence of pneumonia, are those recorded in a volume published in 1883, by Jürgensen, with regard to the prevalence of the disease from 1873 to 1881, in the village of Lustnau, near Tübingen.

† [In addition to the above arguments in favor of the specific non-traumatic character of pneumonia, the discovery of an apparently peculiar micrococcus by Friedländer ("*Virchow's Archiv*," vol. 87) must be mentioned. The theory which Jürgensen advocates was held by Oppolzer, and in this country has often been put forth, among others by Sir Andrew Clark, who even maintains that true pneumonia is not inflammation at all. See, on this and other points, Dr. Sturges's excellent monograph.—Ed.]

exhausted by pyrexia. Sometimes the same part of the lung is affected on successive occasions, sometimes not.

The *treatment* of pneumonia has been the subject of more discussion, and of greater differences of opinion, than that of almost any other disease. Forty years ago it was the universal practice to bleed freely and to administer calomel, antimony, and opium. In place of this "antiphlogistic" method the administration of wine or brandy in large quantity was afterward commonly adopted, mainly under the influence of the late Dr. Todd. But for several years past the conviction has prevailed that, as a rule, no active interference with the natural course of the disease is needed.* The patient must be kept in bed, in a well-ventilated room at an equable temperature, and he must not be allowed to talk. His diet should consist of milk, beef tea and gruel. For medicine it is generally considered sufficient to prescribe salines, with ipecacuanha and other expectorants. The pain, however, when it is severe and interferes with the freedom of the breathing, must be kept under control; a mustard plaster or a blister will sometimes very quickly relieve it, or laudanum may be applied externally upon a hot flannel; or it may be necessary to use morphia hypodermically. Sleep, too, must be secured by chloral, or opium or morphia, if there is wakefulness, but when lividity and other signs of failure of the respiration appear, much caution is required in the administration of such remedies. If indications of cardiac weakness show themselves, stimulants should be given, sometimes in large quantities. Patients advanced in years commonly need brandy from the very commencement of the disease.

Of late, however, it has been asserted by some German physicians that better results can be obtained by vigorous antipyretic treatment, like that which has been described in the chapter on enteric fever. At Basle cold baths have been used whenever the temperature rose a little above 102°; Jürgensen advises that 104° should be the point at which the baths should be systematically employed, for patients whose temperature ranges at from 101° to 103° he merely orders a tepid bath in the morning, so as to increase the normal remission during the early part of the day. He also administers quinine in doses of thirty grains at intervals of forty-eight hours. The theoretical basis on which this practice is made to rest is that the great danger of pneumonia is failure of the heart as the result of the combined action of pyrexia and of disturbance of the pulmonary circulation caused by the pneumonia. In proof of its efficacy, Jürgensen adduces a tabulated statement of the fatal cases that have occurred in his practice, showing that scarcely any of his patients died except such as had some dangerous complication. This observer makes a point of giving wine as a stimulant to the heart before each cold bath, and when signs of cardiac failure appear in the course of the disease he gives alcohol very freely, as well as camphor and musk.

So far as I know, the antipyretic treatment of pneumonia has not hitherto been tried, on a large scale at any rate, in this country. I have hitherto felt satisfied with the results of the usual "expectant" method, which in the large majority of cases certainly could not be bettered.

Cirrhosis of the Lung—Chronic Pneumonia.—The term "cirrhosis of the lung" was first used in 1838, by Sir Dominic Corrigan, for an affection of the pulmonary tissue consisting in a replacement of its alveolar structure by a fibroid material, histologically analogous to that which in the liver causes the disease known as cirrhosis of that organ. Unfortunately, however, the mere presence of such a fibroid material in the lung is very far

* [The principal share in establishing this reform in practice on a sound basis of observation must be attributed to the late Dr. Hughes Bennett, of Edinburgh.—Ed.]

from being characteristic of any one morbid process. When I come to discuss the subject of phthisis, I shall express my own belief that in all but an insignificant minority of cases what has of late been termed "fibroid phthisis" is nothing but an advanced stage, or a very chronic form, of a disease that is really tubercular. I even incline to the opinion that this is true of those destructive affections of the lung which are set up by the inhalation of dust. Still, there can be no doubt that exceptional instances are now and then met with, to which such an explanation is altogether inapplicable. One peculiarity that often characterizes them is that whereas the whole of one lung may be shrunken into a hard, gray mass, showing no trace of its proper structure, the opposite lung is perfectly free from any similar morbid process. I need not say that in phthisis long before one lung is completely destroyed the other always becomes involved in the disease. Another feature is that when one part of a lung is affected before the rest it is usually the lower lobe. I have notes of five cases which were observed in the post-mortem room of Guy's Hospital, and in each of which the bases of both lungs were found to have undergone cirrhosis. In not a single instance was there any indication of the presence of tubercle, nor were any caseous nodules or masses present.

The *pathology* of this affection is still a matter of uncertainty; possibly it has different modes of origin in different cases. Sometimes it seems to have begun in an attack of pneumonia. This is a question which I have already touched upon at p. 911, where I cited the opinion of Dr. Wilks that "croupous" inflammation of the lung, if it does not prove fatal, always undergoes resolution, and never leads to a chronic form of consolidation and induration of the pulmonary tissue. But, after all, the evidence on which this opinion was based must have been of a negative kind, consisting in the observation of a number of cases, in none of which such a result was noted. In hospital practice it can obviously happen but very rarely that a patient should come under the eye of the same physician during an acute illness, and again many months or even years afterward, when a chronic affection of the lung proves fatal, so as to afford opportunity for exact pathological investigation. However, medical literature contains several carefully recorded cases in which the origin of cirrhosis in pneumonia appears to have been clearly established. I may cite two from Dr. Bastian's collection of thirty cases, tabulated in vol. ii of Reynolds' "System." One is a case of Charcot's. The patient, a man aged sixty-one, was admitted on March 30th, 1850. He had been attacked five days before with rigors and pain in the side, and had rusty sputa. There were all the signs of pneumonia affecting the whole of the right lung. These continued with but little alteration until his death on July 19th. At the autopsy the right lung was of a grayish-blue color on section, as hard as cartilage, shrunken to two-thirds its natural size, and enveloped in an immensely thickened fibrous mass. The other was recorded by Dr. Mayne, of Dublin. It is that of a man, aged fifty-four, who in July, 1855, after a hard day's work, was seized with rigors and all the symptoms of pleuro-pneumonia. The acute disease subsided, but he never afterward regained his health and strength. In October, 1856, he was attacked with fresh febrile symptoms, and he died at the end of the year. The lung on post-mortem examination was found affected with well-marked cirrhosis. Recently Dürr has recorded in a volume published by Jürgensen, at Tübingen, two further cases, which occurred in very young children, but in each of which the primary attack appeared to be one of croupous pneumonia. Probably it often happens that the pneumonia which leads to cirrhosis, instead of belonging to the croupous form of the disease, is rather of the catarrhal form, occurring secondarily to measles or to whooping-cough. I may incidentally observe that I am partic-

ularly disposed to refer to this cause one variety of the affection, in which, instead of the tissue of any part of the lung being all converted into a fibrous substance, it is marbled or intersected by bands crossing one another so as to split it up into areas of various sizes and shapes. Such an appearance, which is not uncommonly seen in the post-mortem room, is not known to possess any clinical significance. I think it is generally supposed to represent an early stage of cirrhosis, such as might have advanced, if the patient had lived, to total destruction of the pulmonary texture. But it has seemed to me to be rather a residue, or relic, complete in itself of the former acute process.

In most cases of cirrhosis, the pleura over the affected part of the lung is adherent to the parietal layer, and the two together form a dense white mass, of cartilaginous hardness, and from a quarter of an inch to an inch in thickness. The only way to remove the lung from the chest at the autopsy is to cut it out with the knife. There is also a similar thickening of the pleural layers separating the different lobes. This state of the serous membrane has led to the notion that the affection may, perhaps, have begun as in an attack of pleurisy, and that the lung substance may have been invaded from the surface. But I am rather disposed to agree with Dr. Wilson Fox in doubting whether it is possible for the whole of the interior of a lung to become cirrhosed in such a manner by extension along the interlobular septa. A case showing how easily erroneous conclusions may be drawn, even from post-mortem observations, occurred at Guy's Hospital in 1877. A man, aged twenty-seven, died of disease of the left side of the chest, with a history of having had inflammation of the lungs at the age of fifteen, and some thoracic affection even before that, in early childhood. There was found to be a localized empyema in front, and the pleura elsewhere was thickened in places to the extent of an inch. The lung was very small, but its tissue was generally healthy, except that it was intersected by fibrous bands. It would have been natural to infer that the organ had been invaded from without. But the apex of the other lung was affected in a precisely similar way, although the serous membrane covering it was in a normal state.

Dilatation of the bronchial tubes in the affected lung or part of a lung, is present in most cases of cirrhosis. So intimate, indeed, has been supposed to be the connection between these two morbid conditions, that Jürgensen (in Ziemssen's "Handbuch") discusses them together in the same chapter. This, however, is certainly a mistake. As I have already remarked at p. 900, various forms of bronchiectasis may occur without there being any change in the pulmonary tissue, unless it is, perhaps, emphysema. And in no fewer than six of Dr. Bastian's thirty cases of cirrhosis, it is expressly stated that the tubes were of the natural size. As to the relation between bronchial dilatation and the lung affection, when they coexist, there are differences of opinion. Some think that a chronic inflammatory process may start from the tubes and lead to a gradual fibrous thickening of the alveolar walls with obliteration of their cavities, just as in other cases an acute gangrenous pneumonia is well known to spread from them. But the very definite localization of the morbid process, the completeness of the destruction of the pulmonary texture, and the fact that the pleura becomes so greatly thickened, seem to me to form strong arguments against this view. By Corrigan it was suggested that the occurrence of bronchiectasis was secondary to the cirrhosis; he imagined that the contraction of the adventitious fibrous material in the lung dragged upon the walls of the tubes, so as to widen their channels. Dr. Wilson Fox, however, seems to me to be more likely to be right in thinking that if the dilatation of the tubes really follows the lung affection, it is caused by the expiratory force

of cough, exactly as it is in other circumstances; at an early stage of the disease it is fair to suppose that all the affected structures may be soft and yielding.

On the whole, I am disposed to conclude that there is but little probability in favor of either of the two views that would refer the origin of cirrhosis to a starting point beyond the walls of the pulmonary alveoli themselves. Many of those cases which cannot be traced definitely to an antecedent acute attack of lung inflammation, may, after all, have arisen during an illness that occurred in childhood, so far back as to have been forgotten. And it seems by no means impossible that in others the morbid process may be chronic from its commencement, a true primary chronic pneumonia.

Incidence.—Cirrhosis seems to be most apt to occur during early adult life and middle age; nearly two-thirds of Dr. Bastian's cases proved fatal between the ages of fifteen and forty. There were more males than females among the patients, in the proportion of four to one.

Symptoms.—Clinically the symptoms of cirrhosis vary according to its extent. If it affects only a small portion of one lung, or even of both lungs, the patient becomes a chronic pulmonary invalid, with cough and dyspnœa; the expectoration is purulent, sometimes stained with blood, sometimes dirty gray in color and offensive; the fingers become clubbed; and there is more or less marked emaciation. The physical signs are those of consolidation of the pulmonary tissue, with the addition of more or less abundant râles, which may be consonating or even metallic in character. Such cases are not at all uncommon in hospital practice. But after staying a certain number of weeks in the ward, the end is that the patient is discharged unbenefited, or at best with some relief to the symptoms from the treatment adopted. In each of the five cases of cirrhosis of the bases of both lungs, above referred to as having been observed in the post-mortem room at Guy's Hospital, the cause of death was either quite unconnected with the pulmonary affection, or else it was an attack of acute pneumonia, or lardaceous disease of the kidneys, the result of the protracted suppuration.

Very different is the course of cirrhosis when it involves the whole of one lung. In that case the affected side of the chest falls in, so that it measures in each direction much less than the other side. There is often considerable difficulty in distinguishing the disease from a mere chronic pleurisy with retraction, or even from a malignant tumor producing a like condition. According to Dr. Walshe, there is not in cirrhosis the same degree of twisting of the ribs on their axes as in pleurisy, nor is the shoulder lowered so much, nor is the inferior angle of the scapula tilted so far outward. Great assistance in the diagnosis may be afforded by the discovery of râles on auscultation, and by the presence of an abundant purulent, and perhaps offensive, sputum. From malignant tumor cirrhosis is commonly distinguished by the state of the mediastinum. This, in the disease now under consideration, is dragged out to the furthest possible extent. If the right lung is cirrhosed, the heart is seen beating at the right nipple, if the left, its visible pulsation may extend upward nearly to the left clavicle. In either case the opposite lung undergoes an extreme degree of enlargement, so that the whole sternal region, and even the space beneath the costal cartilages on the affected side, becomes resonant on percussion, and transmits to the stethoscope a loud vesicular murmur. After a time, however, this overdistended and perhaps hypertrophied lung fails to carry on the respiratory function efficiently. The right side of the heart becomes dilated, a tricupsid regurgitant murmur develops itself at the ensiform cartilage, the patient suffers from permanent orthopnœa, the liver becomes nutmegged, and ascites and œdema of the lower limbs set in. The case, in fact, assumes all the characters of one of heart disease with dropsy, and so terminates fatally.

About the *treatment* of cirrhosis of the lung, all that need be said is that for the relief of the different symptoms such remedies must be used as are recommended for the like symptoms in other diseases, such as phthisis and bronchitis, which are more commonly met with in practice.

Gangrene of the Lung.—This affection, to which allusions have already been made, forms no exception to a statement made at p. 75 with regard to gangrene in general, namely, that the death of any part of the human body is always either the result of an intense inflammation, or else of an injury which, if a little less severe, would have set up inflammation, but which by its violence kills the tissues outright before there is time for inflammation to occur.

Of pulmonary gangrene without antecedent pneumonia it would, indeed, be difficult to find unequivocal examples. But in the post-mortem room cases are sometimes seen in which there has been rapid and extensive sloughing of a portion of a lung, and in which no zone of hepatized tissue separates the gangrenous part from that which is healthy or merely œdmatous. In such cases the fact of there having been inflammation is, at any rate, unproven. Generally, however, the sloughing mass lies within a more or less broad area of consolidated lung substance, of which it had evidently at one time formed a part. Should the disease have proved fatal at an early stage, the one may slide off gradually into the other; should it have reached a more advanced stage, there may be a well-marked line of demarcation, or extrusion may even have occurred, with the formation of a cavity. It does not seem to me that it is in accordance with actual experience in the dead house to distinguish two separate forms of pulmonary gangrene, the one "circumscribed" the other "diffuse," as is done by almost all writers from the time of Laennec, who was the first to recognize it as a special affection. With regard to the anatomical characters of gangrene of the lung, little more need be said than that the diseased part is of a dirty greenish-brown or black color, and so soft as to be sometimes almost diffluent. It is often horribly fetid, but I think I have seen cases in which there has been no marked odor, probably from death having occurred before putrefaction had had time to set in. Microscopically, I believe that the pulmonary structure is sometimes hardly to be recognized, the alveolar walls having broken down into a mere granular *débris*.

As I have observed at p. 909 in describing the disease, which is from a clinical point of view the chief kind of pneumonia, and which is almost the only one that can be regarded as a definite member of the nosology, that it rarely leads to gangrene, except in very old and feeble persons, in drunkards, and in those who are exhausted by some other acute or chronic malady, as (for example) by diabetes. But I can recall to mind one or two instances in which what had appeared to be an ordinary attack of "croupous" inflammation of the lungs in a healthy subject has, when the acute stage subsided, been followed by symptoms which seemed to indicate that at least some of the hepatized tissues had undergone sloughing. Dr. Walshe records just such a case—that of a man who was slowly recovering from an attack of pneumonia of the right lower lobe when hæmoptysis set in, and was followed by the copious expectoration of a frothy, intensely fetid fluid, while at the same time physical signs like those which indicate the formation of a cavity made their appearance. Ultimately this patient left the hospital in fair general health, and with no physical signs except such as commonly denote "hard consolidation." Another pulmonary disease, in the course of which gangrene may occur, is phthisis. Dr. Walshe speaks of having seen some six cases in which the special fetor appeared incidentally in connection with tuberculous vomicæ already formed. To one

of these cases I shall have occasion to refer later on. Far more frequently, gangrene of the lung arises out of an inflammatory process of septic character. There are various ways in which such a process may be set up. Sometimes it is by direct extension from neighboring parts, as when an ulcerating cancerous growth in the œsophagus eats its way into the lung, or when perforation occurs from a suppurating hydatid or simple abscess of the liver, or from an abscess starting from an ulcer of the stomach, or even from a putrescent empyema. Sometimes the infection is brought by the blood vessels, as when septic emboli become lodged in branches of the pulmonary artery, having been derived from a cerebral sinus diseased as the result of some ear affection, or from a systemic vein in the neighborhood of a part involved in an "unhealthy" inflammation, or even from the right side of the heart in a case of ulcerative endocarditis. Almost all these causes of pulmonary gangrene will be found mentioned elsewhere. In yet other cases, the disease starts from the bronchial tubes. A foreign body, as a piece of bone impacted in one of the bronchi, is very apt to set up a sloughing pneumonia; or it may even be caused by the entrance into the air passages of pulpy or liquid food, as in patients who have chronic laryngeal disease, or in those who are comatose from apoplexy, or in lunatics who have to be fed by force. A like result may be produced by matters from the stomach drawn into the lungs during the act of vomiting, especially in persons rendered insensible by anæsthetics; I have seen death brought about in this way in cases of hernia or of intestinal obstruction, in which there had been a copious discharge upward of the contents of the small intestine. Again, putrid materials that pass into the air passages may be originally derived from the mouth, as in cases of gangrene of the cheek or of the tonsils, of diphtheria of the fauces, or of sloughing cancer of the tongue; Volkmann has even suggested that sometimes severe disease of the ear leads to pulmonary gangrene as the result of the dropping of morbid secretions down through the Eustachian tubes into the pharynx, and not (as is more usual) through the occurrence of thrombosis and embolism. Lastly, there are cases in which a sloughing pneumonia is due, not to the entrance into the bronchi of matters from without, but to the decomposition of retained secretion or inflammatory exudation, which had been found within their channels. I shall have to discuss elsewhere the question whether this is not the real cause of the occurrence of gangrene of the lung in patients affected with aneurism of the aorta, or with solid mediastinal tumor. It certainly is so in those who have bronchiectasis, as I have explained in speaking of "fetid bronchitis." (*Supra*, p. 902.)

This brings me to a question with regard to which even the most recent writers seem to me to have failed to obtain clear views. In fetid bronchitis, as we have seen, the patient's breath and his expectoration may have either the true odor of gangrene, dependent upon the decomposition of dead tissue, or a peculiar nauseous, acrid odor, which is sometimes not unlike that of fecal matter, and which appears to be due to the presence in it of free fatty acids.

In the latter case the sputum presents the further peculiarity of separating into three layers, in the lowest of which are found certain masses, or "plugs," consisting of exudation that had accumulated in the dilated tubes, and undergone chemical and other changes there. Now, it seems to be assumed that the same description applies also to other forms of sloughing of the lung. But I cannot doubt that this is a complete mistake. Unless there is an antecedent bronchiectasis, the peculiar "plugs" cannot be formed, nor is there any reason why fatty acids should be set free. And, as far as I have seen, the odor in all other cases is simply that of gangrene—an indescribable fetor, but one which is always of the same character,

though it varies greatly in intensity, being sometimes only just perceptible, sometimes so strong as to poison the whole air of a room. As a rule, the patient's breath has the same smell as the expectoration, especially after coughing. And it now and then happens that the breath is characteristically offensive for some days, while the sputum remains odorless. But there are a good many cases in which, during life, no fetor is discoverable, whether in the breath or in the expectoration, or even in which the presence of gangrene is not suspected until it is seen at the autopsy. Hertz (in Ziemssen's "Handbuch") accounts for this fact by assuming that the tubes passing from the sloughing parts are obstructed by secretion. But, judging from my own experience in the post-mortem room, I should say that the absence of fetor before death occurs especially in cases in which the gangrene is acute, which would be commonly described as belonging to the "diffuse" variety of the affection, and in which, therefore, it is most unlikely that obstruction of tubes should be present.

The fetid sputum of gangrene of the lung is commonly of a dirty gray or greenish color, sometimes it is brown or almost black, from the presence of altered blood. The microscope does not often lead to the detection in it of recognizable fragments of pulmonary tissue, although such fragments are so often easily to be found in ordinary cases of phthisis. Actual hæmoptysis is said to occur comparatively seldom in adults, but frequently in children. Fatal hemorrhage from this cause, due to the erosion of a large vessel, is an event of great rarity. When the surface of the lung is affected, the pulmonary pleura usually gives way, leading to the formation of pneumothorax, which is quickly followed by septic pleurisy. If there should happen to be local closure of the serous cavity by adhesions, it is said that a subcutaneous emphysema may develop itself, or that an abscess may form, which may open externally after burrowing to a greater or less distance. Another occasional effect of the presence of a patch of gangrene in the lung is said to be the dropping of putrid matters into tubes belonging to other parts of the organ, so as to set up sloughing in them also. In this way, according to Hertz, it often happens that the "diffuse" form of the affection arises out of the "circumscribed" form.

The only *physical signs* that can be said to belong to gangrene of the lung are such as serve to indicate the formation of a cavity in the organ at a spot where the tissue previously was either healthy or simply consolidated. But it can be only in very rare cases that such signs are to be definitely made out. They would, of course, include cavernous or amphoric breathing, consonating or even metallic râles, and loud bronchophony. It is important to remember that where there is a possibility of the presence of phthisis, the mere detection of a cavity proves nothing as to the exact seat of the sloughing process, unless it is known that no vomica existed at the same spot before the fetid expectoration began.

The general *symptoms* that accompany gangrene of the lung are often very severe, but it does not seem to me that they point to the presence of this affection so definitely as might be supposed from the statements made by most writers on the subject. It is said, for example, that the pulse is small, feeble, and very frequent, and that the pyrexia quickly passes into an adynamic form, with great prostration of the vital powers. That absorption of putrid matters into the blood from the lung should produce such results is, indeed, to be expected, but when the characteristic fetor is absent, it surely is not possible for any one, from the intensity of the general symptom alone, to suspect that sloughing of the lung is taking place. In pneumonia, the patient commonly falls into a similar condition before death, even when the affection has not advanced beyond the stage of gray hepatization. And of many other diseases that may lead to the occurrence of

pulmonary gangrene, the same thing may be said apart altogether from their being ever attended with any lung complication. Nor does it appear to me that when the sloughing part is very limited in extent, the nature of the morbid process is commonly to be inferred from the fact that the constitutional symptoms are disproportionately severe. The truth rather is that in such cases the patient's general condition often remains for several weeks much better than might have been anticipated. Hertz, indeed, speaks of anorexia and gastric disorder, and even diarrhœa, as being caused by the swallowing of some of the offensive material expectorated from the lung, but I suspect that this would be rather difficult of proof.

It is only when the gangrene is limited to a small part of the lung that recovery is possible. How minute a slough may cause fetor is well shown by one of the cases of phthisis complicated with gangrene observed by Dr. Walshe; in that instance the expectoration of a pea-like mass brought the fetor to an end. Unfortunately, he does not say for how long a time it had been present. When a case of gangrene is about to end favorably, the separation of the dead tissue is doubtless followed by the formation of a lining membrane to the cavity left by it, and, perhaps, this may ultimately shrink and become converted into a fibrous cicatrix.

In the *treatment* of pulmonary gangrene it is an important point to diminish the fetor as far as possible, and this applies also to cases of fetid bronchitis, the treatment of which affection I left over from the last chapter, to be dealt with now. The most effectual means of attaining the object aimed at is by inhalations of oil of turpentine, carbolic acid, *oleum cadinum*, or eucalyptol. Turpentine inhalations were, I believe, first used by Skoda about thirty years ago. His plan was to pour a teaspoonful or two of oil of turpentine upon the surface of some boiling water, and to let the patient draw the vapor into the lungs. A much better method is to use a Siegel's spray apparatus, so as to atomize a liquid containing from five to two parts of carbolic acid in 100 parts of water or of a solution of common salt. The inhalations may be repeated two or three times a day. Care must be taken that there is not enough absorbed to set up headache or giddiness, or to give the urine a brown or black color. The *oleum cadinum*, or the eucalyptol, may be directly inspired from a sponge placed in an "orinasal respirator." The effect of such inhalations is sometimes very striking, especially in cases of fetid bronchitis.

It is needless to say that the strength of the patient must be maintained by good and abundant food, that the administration of stimulants in large doses is sometimes necessary, and that ammonia, camphor, ether, quinine, and the tincture of perchloride of iron, may each in turn do good service.

PLEURISY.

PHYSICAL SIGNS—OF DRY PLEURISY—OF PLEURITIC EFFUSION—SYMPTOMS—COURSE, PROGNOSIS, AND EVENT—EMPYEMA—TREATMENT—PARACENTESIS—DIFFICULTIES OF DIAGNOSIS—COMPLICATIONS—ÆTIOLOGY AND RELATION TO TUBERCLE.

Pleurisy or pleuritis was mentioned by Hippocrates, and was described by other ancient Greek writers, who undoubtedly were referring to cases of the same disease to which we now apply that name. Nevertheless, it is only during the present century that its real nature has been known—an inflammation of the two surfaces of the serous membrane which lines the space surrounding the lung, while that organ itself is unaffected, unless pneumonia is also present. For, until percussion and auscultation were discovered, there was no possibility of drawing valid distinctions between the two diseases in clinical practice.

Anatomically, inflammation of the pleura resembles the inflammations of other serous membranes very closely. The earliest morbid appearance is generally said to be an injection of the smaller blood vessels and perhaps the formation of ecchymoses, and no doubt hyperæmia is really present at the commencement of very severe pleurisy. But, as a matter of fact, one often finds patches of recent lymph upon the surface of a lung without any reddening, when an inflammation of no great intensity has set in shortly before death, in a patient suffering from some other disease; and ecchymoses by themselves indicate, not an early stage of pleurisy, but either that there has been obstruction of some part of the air passages, causing a great impediment to the entrance of air into the lung, or else that there has been septicæmic or pyæmic infection of the blood. In many instances the effusion of lymph upon the opposed surfaces of the serous membrane occurs only at the very commencement of an attack of pleurisy, of which it may be said to constitute a "plastic stage" or "period." But in others it forms the whole of the disease. The case is then said to be one of *dry* pleurisy. When the inflammation afterward subsides, the morbid material sometimes undergoes complete absorption, leaving the pleura in its natural condition, or slightly dull and opaque. But very often the two surfaces have in the meantime adhered together, and remain henceforth connected by separate bands or by a uniform layer formed of connective or fibrous tissue, which may have a free supply of blood vessels.

Signs of Dry Pleurisy.—There is an auscultatory sign which, when it can be heard, is of itself almost conclusive as to the presence of lymph. This is the "friction sound" or "pleuritic rub," due to the movement upon one another of the two roughened serous surfaces. One cannot but be interested to know that Hippocrates described the pleura as "creaking like leather," for such a phrase is often exactly applicable to the sound which is conveyed to the ear by the stethoscope. Yet Laennec, though he must have often heard this sound, failed to understand its meaning, and left to Reynaud the credit of rightly interpreting it. Laennec's idea about it was that it indicated emphysema, especially what he described as interlobular emphysema. And we shall presently see that there are still some observers who think that a precisely similar sound may sometimes be due to that cause, or,

again, to miliary tuberculosis of the pulmonary surface. On the other hand, Dr. Walshe has expressed the opinion that a friction sound of the most marked character may be heard when there is no lymph whatever, if the serous membrane is rough from "simple vascularity." But this conclusion appears to me to be hardly warranted by the case from which he drew it. That case was one in which death occurred sixteen days after a rub had been heard, whereupon fluid was found in the pleural space, but no plastic exudation. But how is one to know that lymph may not have existed previously, and have undergone disintegration, or absorption by leucocytes?

It is difficult to describe in words the characters of a *pleuritic rub:* one must hear it to appreciate them. But I may say that in its most typical form it consists of an irregular succession of short, harsh sounds, which give one exactly the impression of something catching or dragging against an obstruction and then slipping, but only to catch or drag once more. The patient himself is often conscious of a rough grating sensation each time he breathes; and one may be able to *feel* the rub quite plainly by placing one's hand over the affected part of his chest. Sometimes a rub accompanies both inspiration and expiration. Sometimes it is to be detected only at the very end of a deep inspiration, when the lungs are just becoming expanded to the greatest possible degree. It may be heard within twelve hours of the commencement of the disease, and in cases of dry pleurisy it may persist for days or even weeks with but little alteration. But it much more often disappears after a short time, because fluid effusion is formed which keeps the two surfaces apart. Or, if the inflammation should be spreading, it may, after it has ceased to be audible at one spot, be for the first time discoverable at another. A rub is not often present over a large area at once. The part of the chest at which one is most likely to hear it is in the axilla, or below the nipple, or round to the back below the scapula. The reason is not only that pleurisy more frequently affects the surface of the lower lobe than of the upper, but also that the descent of the diaphragm causes an actual movement of the pulmonary upon the costal pleura, which is wanting elsewhere. Sometimes, however, a rub can be heard over the front as high as the clavicle. In some cases, I believe, a sound which is really due to pleural friction possesses characters so indefinite that one cannot distinguish it from moist or dry sounds seated in the bronchial tubes. This has long been taught at Guy's Hospital. In his well-known paper on the "Difficulties and Fallacies attending Physical Diagnosis," Addison cited a case of Dr. Barlow's, in which lymph upon the adjacent surfaces of the liver and of the parietal peritoneum caused a "crepitus, which closely resembled a mucous rattle" ("Collected Works," p. 87). It is hardly possible to obtain positive proof of the fact, so far as regards the pleura itself, because, even if an autopsy shows lymph upon that serous membrane, one cannot be quite certain that an adjacent tube may not during life have contained fluid secretion.

The only other physical signs of the plastic stage of pleurisy are a certain degree of impairment of movement of the affected side of the chest and a corresponding enfeeblement of the respiratory murmur.

Signs of Pleuritic Effusion.—In most cases of pleurisy, however, liquid is effused into the serous cavity, often in large quantity and with great rapidity. Two or three quarts are not uncommonly found, and Watson cites a case in which Crampton, of Dublin, drew off from the left pleura as much as fourteen imperial pints. The liquid is sometimes translucent and of a yellowish color, perhaps containing shreds and floating masses of fibrin. It has then an alkaline reaction; accord-

ing to Fräntzel, who writes on pleurisy in Ziemssen's "Handbuch," its specific gravity may vary within wide limits, from 1.005 to 1.030. But in other instances it is more or less opaque, and on standing throws down a layer of greenish-yellow pus. Or it may be altogether purulent, in which case its reaction to test paper is acid. This constitutes what is termed an empyema.* As a rule, no doubt, the formation of pus in the pleural cavity is a gradual process, the liquid being at first serous or sero-purulent, and becoming more and more opaque as the leucocytes in it increase in numbers. And in such cases both the parietal and the pulmonary surfaces may still remain coated with more or less thick layers of fibrin. But I believe that when the inflammation is from the very first of an exceptionally violent character—as, for instance, when it is set up by the entrance of putrid matter from without—a primary suppuration may occur and the serous membrane itself may remain as smooth and shining as does the peritoneum under similar circumstances.

Sometimes a pleuritic effusion is of a deep brown or purple color, from admixture of blood. This may be due to the fact that the patient has scorbutus; or it may depend upon the presence of tubercles in the serous membrane. According to Fräntzel, "a hemorrhagic tubercular pleurisy" is less rare in persons advanced in years than in those who are younger; but the only example of it that I have met was in a man, aged thirty-five. The writer to whom I have just referred also hints at cases, comparable with hemorrhagic pachymeningitis, in which, after connective tissue has already been formed as the result of a pleurisy, fresh inflammation is lighted up attended with extravasation of blood; but he does not say that he has actually met with such an instance. Not long ago I removed a quarter of a pint of liquid of a dark-brown color from a patient who had pleurisy as a sequela of scarlet fever. He rapidly recovered.

Liquid pleuritic effusion usually gravitates into the most dependent part of the serous cavity, whatever may have been its starting point. Thus, at the commencement of the disease, lymph may have covered the front and side of the lung, but when serum or sero-purulent fluid is poured out, it falls into the back and lower part of the chest if the patient is in bed, or it accumulates above the diaphragm if he is not recumbent. But this rule is liable to exceptions, when portions of the lung have previously become fixed to the chest wall as the result of a former attack of pleurisy, or even by the rapid development of adhesions at the commencement of the illness which is still in progress. Thus a considerable quantity of liquid may accumulate somewhere toward the upper part of the pleural sac, or between the lung and the pericardium, or between two lobes of the lung itself, without there being any in the usual position at the base. And even when the whole of the serous membrane is affected, the seat of a serous or purulent effusion may be more or less irregularly circumscribed. Between 1873 and 1876, I saw four instances of this in the post-mortem room. In one case, there was a broad adhesion to the lateral region of the chest and another to the diaphragm, so that the liquid filled the upper part of the pleural cavity while crepitant lung tissue still existed in the lower part. In another case, there was liquid at the base behind, and also above the root of the lung, with an intervening zone where the lung

* In England the meaning attached to the word *empyema* is, I believe, always that of a collection of pus in the pleural cavity. But on the Continent, in spite of etymology, collections of serous fluid, and even of blood, are included under the same name (see Littré and Robin's "Dictionary"). It has also been used as a synonym for thoracentesis, so that when the pus escapes through an intercostal space, and has to be let out with the knife, an *empyema necessitatis* is said to arise.

was firmly adherent. But I think that the limitation of pleuritic effusion by adhesions is seldom so complete as to lead to the formation of two or more collections of fluid entirely cut off from one another; generally speaking, they communicate freely, as can be shown at an autopsy by passing a bent probe behind the bands of adhesion from space to space.

A necessary consequence of the presence of liquid in the pleural sac is that the lung becomes reduced in size. It is common to speak of the organ as being *compressed* by effusion, but this is a very inadequate way of stating the case, as must now be shown. Every physiologist will admit that the elasticity of the pulmonary tissue must lead it at once to recede as soon as there is anything to take its place, until it has become collapsed to at least the same extent as when air is admitted into the serous cavity after death. But, further, Lichtheim has proved by certain experiments, to which I have referred when speaking of bronchitis, that the elasticity of the lung does not become exhausted until the alveoli are completely emptied of all their gaseous contents. The reason why a lung is not rendered altogether airless when the pleural sac is laid open in the dead body seems to be mainly that the walls of the tubes presently fall together and offer a resistance to the further escape of air, which the elasticity of the pulmonary tissue is unable to overcome. And during life an additional force is in operation to empty the alveoli of air, namely, absorption by the blood current circulating in their walls. Consequently it is incorrect to say that a lung undergoes compression by pleural fluid until it is absolutely airless, only; beyond this point it undoubtedly may become compressed until all the blood is driven out of its substance. I think it is advisable to mark this distinction by separate names, and a lung which is bloodless as well as airless may be said to be *carnified*, whereas a lung which is merely airless may be spoken of as *collapsed*. Both terms have long been in use, but not with the precise shades of meaning here assigned to them. A carnified lung has a very peculiar appearance; it has a kind of slaty-gray color and is best described as being mouse colored. Its cut surface is smooth, very hard, and dry, showing the flattened orifices of air tubes and vessels closely packed one against another. Sometimes, however, the bronchial tubes in it contain pus, and if its substance should happen to be affected with pneumonia or œdema, its characters are necessarily modified. The position occupied by a lung entirely compressed by liquid effusion is, as a rule, determined by its root; it becomes flattened against the mediastinum and backward toward the spine, and if covered by a mass of false membranes its very presence may be altogether overlooked. I believe that the notion of patients living on after having "entirely lost one lung," which is now so often applied to those who have phthisis, had its origin in autopsies made in cases of pleurisy of long standing. But when the organ has previously been firmly fixed by adhesions, it cannot thus be driven inward, and it may lie in the summit of the pleural space or be irregularly pushed to one side or even downward. The most common deviation from the rule is, doubtless, when the upper part of the lung is affected with tubercular disease, and there is consequently a solid mass occupying a corresponding extent of the pleural cavity.

When pleural effusion is insufficient in quantity to cause the lung, as a whole, to become empty of air, and when, therefore, *compression* of the organ is a long way from having begun, the effect on the pulmonary tissue is very different from what is generally supposed. I think that every one would anticipate that the elasticity of the organ would lead to a gradual and uniform shrinking of its substance, so that all parts of it should contain less air than before, without any part becoming completely airless. But the exact contrary is, in fact, the case. Dr. Moxon long ago pointed out to me,

when he was Demonstrator of Pathology at Guy's Hospital, that the presence of even a few ounces of liquid in any part of the pleural cavity causes a total collapse of the pulmonary tissue which ought to occupy that space. I have since repeatedly verified his observation ; and Cohnheim, at p. 190 of vol. ii of his "Vorlesungen," makes a precisely similar statement. One often sees a small, triangular area of collapsed lung substance at the posterior inferior corner of the organ or a thin strip of it running up along its posterior margin. Nay, a mere enlargement of the heart, without there being any pleural effusion, may give rise to complete airlessness of the inner surface of the left lung ; and distention of the abdomen, thrusting up the diaphragm, may cause a similar affection of the inferior surface either on one side or on both. The explanation of these remarkable facts can hardly be understood except in connection with the mode of origin of collapse of the pulmonary tissue, which has been discussed in the chapter on bronchitis (*supra*, pp. 885, 886). It depends upon the general principle, that whenever a part of the lung fails to be acted upon by the inspiratory forces, it becomes airless, notwithstanding that the tubes which serve it remain patent.

The production of local collapse of the lung substance as the result of the effusion of moderate quantities of liquid into the pleural cavity has important clinical bearings. It accounts for a circumstance which has long attracted the attention of clinical physicians, namely, that temporary changes of posture on the part of the patient often fail to alter the position of the liquid within the chest, so far as one can tell by percussion. I remember to have heard the statement made by a great teacher of medicine that whereas in a chronic pleurisy the effusion could be made to "gravitate" to a different part of the pleural cavity, this could not be done in acute pleurisy, because it was held in the meshes of fibrinous exudation. But for my own part, I think it is quite the exception to be able to elicit evidence of gravitation, even in cases in which paracentesis is directly afterward followed by a very free flow of liquid through the trocar, and Niemeyer says so too. Even if one can alter the level of dullness a little by making the patient sit up (when he has been lying down) the alteration does not, I think, amount to more than a finger's breadth or two. Now, if a certain part of the lung is rendered altogether airless by pleuritic effusion, it is easy to see how the fluid may (as it were) be *held up* in a fixed position in opposition to the force of gravity.

Even when the quantity of liquid is not very large, one can often, in thin patients, make out that the intercostal spaces are less depressed and offer more resistance to the finger than on the sound side, and when the pleural cavity is full of effusion the ribs may be obviously wider apart, and the spaces between them may bulge or (occasionally) yield fluctuation. This, however, is but seldom observed. On measurement one generally finds, if there is much liquid, that the affected side is enlarged, and sometimes the difference between the two halves of the chest is considerable. In determining this, Dr. Gee's cyrtometer is very useful. The play of the ribs in respiration is greatly impaired, much more so than during the plastic stage of the disease. Moreover, in consequence of the extent to which the sternum is carried forward, the movement, even of the unaffected side, during breathing may be much diminished.

Long before the lung has undergone complete compression in a case of pleurisy, other adjacent structures feel the pressure of the effused fluid upon them. The mediastinum is pushed over to the opposite side, the elasticity of the unaffected lung, no doubt, assisting to displace it. Thus, if the pleurisy be on the right side, the apex beat of the heart is felt and seen during life to be situated further to the left than usual; it may lie some distance outside the left nipple. If the disease be on the left side, the heart may

pulsate in the epigastrium, between the sternum and the right nipple, or even to the right of the nipple, while in the normal position of the apex beat no sign of the presence of the organ can be detected. Some observers have supposed that in such cases the heart swings over as a pendulum might, and that its long axis is now directed downward and to the right, so that the part which beats against the chest wall is still the point of the left ventricle. However, I have not in the post-mortem room seen anything which would support such an opinion, and I believe that the displacement of the organ is attended with little change in the inclination of its axis, and that the impulse is now given by some part of the right cavities. But the cardiac sounds are sometimes altered in character under such circumstances. Dr. Hope heard a systolic murmur over the aorta, which disappeared when the pleuritic fluid subsequently underwent absorption. Dr. Walshe met with a case in which each sound of the heart was more or less masked by a blowing murmur for several successive days, while effusion into the left pleural cavity was at its height. He thinks that the diastolic murmur must certainly have depended upon malposition of the heart, probably through tension of the aorta. It is of some importance to know what amount of liquid is required in order to produce a perceptible cardiac displacement. Fräntzel says that effusion scarcely ever reaches up to the third rib without affecting the position of the apex beat to a greater or less extent, and that even smaller amounts of liquid often suffice. He also observes that in children the heart is more easily thrown out of place than in older persons. Another point mentioned by him is that when there have been previous adhesions between the pericardium and the left lung, pleurisy on the left side may cause the heart to be carried backward, away from the chest wall, so that no impulse whatever can be felt or seen. But I think that whenever the amount of effusion is moderate, the normal apex beat may be absent without there being any discoverable impulse elsewhere, the reason probably being that the sternum conceals it. In all cases of this kind the stethoscope must be used, with the object of determining the spot at which the cardiac sounds appear the loudest.

The diaphragm is pushed downward whenever the amount of pleuritic effusion is at all considerable. The displacement of the liver or of the spleen may be recognized by percussion, or one or the other may be felt projecting below the ribs. Fräntzel remarks that in the female the diaphragm resists pressure less than in the male, except when the abdomen is occupied by a pregnant uterus or when the intestines are over filled with gas. According to this writer, when the distention of the pleura is extreme, it is sometimes possible to detect an elongated, tense, fluctuating swelling, which protrudes below the costal cartilages, and which is nothing else than the front part of the diaphragm itself.

In the clinical recognition of pleuritic effusion physical examination of the patient plays the most important part. I have already mentioned several points which must always be carefully sought for and noted; these are enlargement of the affected side, impairment of its movements, an altered state of the intercostal spaces, and displacement of thoracic and abdominal organs. I have still to speak of the results of percussion and of auscultation. Now, of those two methods the former is in this stage of the disease by far the more valuable, as was long ago maintained by Piorry, in opposition to Laennec.

Dullness on *percussion* is, in fact, the main sign of pleuritic effusion. The percussion sound becomes altered long before there is any evidence of pressure upon adjacent viscera. A circumscribed collection of serum and pus may, of course, cause dullness of any part of the chest, but when fluid lies free in the pleural space, the dullness is to be made out first at the base

behind. One must not, however, suppose that small quantities of fluid ought always to be discovered by this means. Wintrich long ago declared that eight or ten ounces could scarcely be detected with certainty. I myself have repeatedly known a considerably larger amount escape recognition; but I think that a great deal depends upon the habitual posture of the patient. If he is sitting upright in bed, the diaphragmatic surface of the lung, rather than its posterior surface, becomes compressed, and the percussion sound may at first be scarcely altered. If he is lying down, the fluid is more or less spread out over the back, and is much more readily discovered. Perhaps, after all, Wintrich's dictum applies not so much to simple pleurisy as to the secondary effusions which occur in persons suffering from other complaints. For, in an ordinary uncomplicated case, I certainly believe that if the percussion sound down to the very bottom of the lung is perfectly resonant, one may in general assert that effusion, if present at all, is in such quantity only as to be clinically unimportant.

As I have remarked at p. 913, the dullness caused by fluid in the pleura differs from that which would (as a rule) be produced by consolidation of the lung in being more complete or absolute, and in the far greater sense of resistance which it conveys to the finger. Another important distinction, referred to at p. 876, is impairment or loss of vocal fremitus. The area over which these signs are to be detected, of course, varies with the amount of effusion. If this is considerable, the whole of the back and sides of the chest may be devoid of resonance, and may yield no vocal fremitus whatever. But now comes a remarkable circumstance. In such cases it usually happens that over the front of the chest—below the clavicle, or downward nearly to the nipple—the percussion note is sub-tympanitic. Skoda first pointed out this fact, which will probably always remain associated with his name. The reason of it, however, is still doubtful. German writers are content to ascribe it to "relaxation of the pulmonary tissue," but I have failed to find evidence that this is adequate to account for it. Dr. Walshe thinks that it depends upon the presence of air in the multitude of minute tubes within the lung, so that a condition essential to its production is that these tubes should not have undergone compression as well as the lung substance. I have already, at p. 865, given what appears to me to be a satisfactory explanation of it in the diminution of the vibrating area formed by the thoracic walls, as suggested by Dr. Bristowe. When the whole of the pleural cavity is filled with fluid, there is absolute dullness in front up to the clavicle as well as behind. The dullness also passes over across the sternum to the costal cartilages on the opposite side.

Auscultation is of less assistance than percussion in the detection of pleuritic effusion. In many cases one finds that the breath sound on the affected side of the chest is enfeebled, indistinct, or altogether wanting. More often, perhaps, tubular breathing is audible over at least a part of the compressed lung, and in some exceptional cases this can be heard so very extensively that one might well hesitate to believe that air is not entering freely into every lobe. I remember to have been told of a case which happened when physical diagnosis was still understood by but few medical men, and in which a surgeon, asked to perform paracentesis for pleural effusion, because he heard a loud sound over one side of the chest, plunged a trocar into the opposite side, with the result that he at once killed the patient.

Nor is there any fixed rule with regard to *auscultation of the voice*. Very often one merely finds that it is conveyed to the surface somewhat more feebly than on the healthy side of the chest, but sometimes there is well-marked bronchophony, and this may occasionally be extremely loud. Or a

peculiar modification of the voice may be transmitted to the ear, constituting what was called by Laennec "ægophony," from its resemblance to the bleating of a goat. It has also been compared to the voice of Punch, and in its most typical form it has a tremulous, squeaking character, which is very curious. Dr. Stone's explanation of it has been given at pp. 875, 876. With regard to the frequency of this sign, widely-opposed statements have been made by writers, in consequence of their differing as to the definition of the term. Almost all the best observers are agreed that what may be called *pure* ægophony is very uncommon, and that it rarely lasts more than a few days. But between it and bronchophony there are all degrees of transition, and if one is to speak of the voice as ægophonic in every case in which it reaches the surface of the chest with more or less of a twang, one will, I believe, find that there are very few instances of pleuritic effusion in which the change in its character is altogether absent. In one particular region this sign is observed far more frequently than anywhere else, namely, about the inferior angle of the scapula, and round toward the axilla. But sometimes it is heard in front, near the nipple, or even upward, close to the clavicle. Its production is believed to depend upon the presence of a rather thin layer of liquid between the lung and the parietes. Consequently, it generally disappears as the effusion increases, unless there are adhesions which keep the lung fixed at a certain distance from the surface of the chest. But Dr. Walshe says that in some exceptional cases, which are "not always explicable by the existence of adhesions," ægophony persists in spite of abundant accumulation of fluid, and I shall have to state presently that according to some of the most experienced of modern auscultators it is sometimes present when there is no fluid effusion at all. Dr. Walshe points out that one source of fallacy lies in the possibility of overlooking the fact that the ordinary voice of the patient is shrill and tremulous, as it so often is in women of advanced age. He also remarks that bronchophony may acquire an ægophonic character if a person speaks with the nostrils closed.

General Symptoms.—The symptoms of pleurisy vary widely in severity in different cases. They are sometimes so marked as directly to suggest to the least experienced observer the nature of the case with which he has to deal; they are sometimes almost, if not quite, absent.

Foremost among them is pain in the side, the *point de côte* of French writers. This is often very violent, and of a sharp tearing or cutting or stabbing character. It is increased both by movement and by pressure. The patient, therefore, breathes in a shallow, jerky manner, for he fears to draw a deep inspiration. His cough, if he coughs at all, is short and half suppressed, for the same reason, and he abstains as much as possible from laughing or sneezing. He lies during the early part of his illness on his back and on the unaffected side, and he shrinks from percussion and even from pressure with the finger upon the intercostal spaces of the inflamed side. Sometimes the pain is augmented by each ensuing exacerbation of pyrexia. As to its immediate cause, there have been discussions which have not led to any very definite result. Some have thought that it might depend upon an extension of inflammation from the parietal pleura so as to involve the neurilemma of adjacent intercostal nerves. Cruveilhier attributed it to the friction of the opposed serous surfaces upon one another, and in this way he accounted for the fact that its seat is so often limited to the lower part of the chest—about the nipple, or between the fifth and the eighth ribs—since there is so much more movement of the visceral upon the costal pleura there than higher up. But the pain is sometimes felt in the shoulder, in the armpit, beneath the clavicle, or along the sternum. In

some cases, too, it is referred to the terminal branches of the intercostal nerves: to the hypochondrium, so that a mistaken diagnosis of hepatitis has sometimes been given; to the loins, so that the case has been called one of lumbago; to the neighborhood of the umbilicus, so that peritonitis has been suspected. I have myself had a patient whose sole complaint was of pain in the crista ilii; I feel sure that if it had not happened that a short while before my attention had been specially directed to this question, I should have failed to discover that he had pleurisy, although on applying my stethoscope I at once heard a rub. Laennec, and afterward Gerhardt, declared that the pain of pleurisy was sometimes seated upon the opposite side of the chest to that which was inflamed, but this has been disputed by other observers. In some cases pain is altogether absent, and a large quantity of effusion may then accumulate in the pleural cavity without its presence being thought of. Fräntzel says that this is especially apt to occur in children, in very old people, and in lunatics. He remarks that the pain of pleurisy is by no means particularly intense in persons of nervous, irritable temperament, who might be expected to suffer more than others from such a disease. It often happens that this symptom subsides or entirely disappears toward the end of the first week, or even after two or three days.

Next to pain, *dyspnœa* is the most striking symptom of pleurisy. I have already referred to the short jerking character of the breathing; but it is also increased in frequency, especially when the patient exerts himself, as in lifting anything or in going up stairs. Sometimes the scaleni and the other muscles of forced inspiration are brought into action; and the nostrils dilate each time air is drawn into the chest. As effusion accumulates, the patient begins to find that he can lie over toward the affected side more comfortably than in any other position, because the weight of the fluid is then removed from the mediastinum. He often has orthopnœa, for the diaphragm works more freely while the upright posture is maintained. Sometimes there is lividity of the cheeks and lips. It is worthy of notice that the dyspnœa of pleurisy is generally more marked in robust plethoric patients than in those who are anæmic and wasted. But as Andral long ago pointed out, it sometimes happens that this disease scarcely at all interferes with a person's comfort. He had a patient who went on with his work as a carter, in spite of an enormous effusion into his pleura; and Sir Thomas Watson speaks of a butcher who, under similar circumstances, could not be persuaded that he was otherwise than well, and fit to leave the hospital.

Cough is seldom entirely absent in pleurisy; and there appears to be no doubt that it may occur independently of any affection of the lung or of the bronchial tubes. Sometimes it can be excited by percussion or pressure over the painful intercostal spaces, or by changes of posture. Kohts also says that during the operation of paracentesis he has seen cough produced by movements of the trocar, disturbing the serous membrane. In his experiments on animals he found that it was caused by irritation of the parietal, but not of the pulmonary, pleura. The cough or pleurisy is not usually attended with expectoration; but sometimes the patient gets rid of a small pellet of mucus or of a little muco-purulent material, in which case one must suppose that a slight bronchial catarrh is also present.

The *pyrexia* of pleurisy is generally moderate. The disease comparatively seldom sets in with a violent rigor, and Fräntzel suggests that, when such a rigor occurs, it should be set down rather to a concomitant pneumonia than to the pleurisy itself. But slight chills returning day after day are observed in many cases, especially when the patient remains out of bed during the day. The temperature commonly ranges at about 101° or 102°,

but in the most severe forms of the disease it may reach 104°, or even a still higher point. In persons suffering from cancer, or from a chronic affection of the kidneys, there may be no pyrexia at all. The pulse is accelerated in proportion to the intensity of the local inflammation and of the consequent disturbance of the system generally Fräntzel insists upon the importance of watching the characters of the pulse with care. For, he says, as effusion goes on, although the temperature may fall, the pulse not only becomes more rapid, but its volume and tension diminish, in consequence of the obstruction to the flow of blood through the pulmonary vessels. This affords a valuable indication of the degree of danger to the patient's life; and during paracentesis the physician may be able to feel the pulse becoming fuller, and at the same time slower, under his finger.

A very material question is whether one can determine by symptoms whether the effusion in a case of pleurisy is serous or purulent. One of the chief indications of the formation of pus is the continuance of high evening temperatures after the first two or three weeks; the pyrexia in many instances assumes a regular hectic type. Œdema of the subcutaneous tissue of the affected side of the chest has long been mentioned as a sign of empyema, but it is often absent where suppuration is going on; and Fräntzel cites a case observed by Traube, in which it was present, but in which a fibrino-serous liquid was withdrawn by paracentesis. The best way of detecting the œdema is to pinch up a fold of skin, and to compare its thickness with that of a similar fold on the opposite half of the body. Fräntzel and Peter have each found, in certain cases of pleurisy, that the temperature of the skin was persistently higher by about a degree Fahr. on the diseased than upon the healthy side of the chest. In almost every instance the effusion was purulent.

Course and Event.—The course of pleurisy varies widely in different cases. In the most severe form of all, which is very rare, but of which Fräntzel says that he has seen three examples, the patient quickly falls into a typhoid state, with delirium, stupor, and a dry, fissured tongue; the dyspnœa and lividity increase, so as to threaten his life by the end of the first week; and even if paracentesis is performed, once or often, the effusion collects again so rapidly that the fatal issue is scarcely retarded.

Even when the disease seems to be attended with no alarming symptoms, one must never regard it as free from danger, if the amount of effusion is large. Death sometimes occurs quite suddenly and unexpectedly. In 1874 this happened to a patient in Guy's Hospital with double pleurisy, who a short time before had been seen by the sister of the ward quietly asleep. For some days previously this patient had had much dyspnœa, and it seems not unlikely that the immediate cause of death was exhaustion of the respiratory centre, after the manner described at p. 25. But in another case, which ended fatally after an hour's extreme distress of breathing, on the evening after admission, it was observed that the pulse ceased before the respiration. In that instance the effusion was on the right side; but there was œdema of the left lung, and it is probable that this really killed the patient. For the occurrence of fatal syncope, when the left pleura is the one affected, a special explanation has been suggested by Bartels, of Kiel, in the "*Deutsches Archiv*" for 1868. It depends upon the anatomical fact (which has been verified after death on several occasions by him and by Fräntzel) that when the heart is pushed far over to the right, the mouth of the inferior vena cava becomes bent almost at a right angle, just above the quadrilateral aperture in the diaphragm, the wall of the auricle forming a fold which covers a large part of that aperture. This is supposed to interfere with the due supply of blood to the heart, especially if at any moment

the diaphragm is suddenly curved upward in a fit of coughing, or if a sudden muscular effort is made. Trousseau had previously attributed the occurrence of sudden death in cases of pleuritic effusion to "twisting of the aorta and large vessels," as a result of displacement of the heart; but there does not seem to be any reason for supposing that he had the inferior vena cava definitely in his mind.

But pleurisy is not always attended with such risks. The inflammation need not go beyond the exudation of lymph, and after a time it may subside, leaving adhesions which fix the lung to the chest wall for the rest of life. Whether dry pleurisy necessarily ends in closure of the affected part of the pleural space, or whether it may not sometimes pass off without permanent damage to the serous membrane, is a question to which it would probably be very difficult to give a positive answer. What is well known to every pathologist is the fact that an adherent pleura is so often found when there has been no history of any chest affection. Dr. Gee gives a tracing, made by the aid of the cyrtometer, which shows that in a child the chest may be markedly contracted on one side, as the result of a universal closure of the pleural space, without there having been any symptoms to suggest the presence of such a morbid condition; the patient died after an operation. Indeed, I am not aware of any evidence that the adhesions left by a dry pleurisy ever affect the health in any way. Some auscultators have thought that such adhesions might account for the occurrence of creaking sounds over the lower part of the lung on inspiration, especially when such sounds are constantly heard over a period of weeks or months.

The *duration* of an attack of dry pleurisy is sometimes exceedingly brief. I was once asked to visit a student who had been seized the same day with violent pain in the side, and who told me that he knew he had pleurisy, because he had had the disease before. I heard a very well-marked rub on auscultation, and told him that I should come to see him on the following day. But when on the morrow I walked into his room he assured me that he was well again; and on listening I could detect nothing abnormal.

When pleurisy gives rise to effusion it sometimes happens that the amount of liquid remains small, so that one can express a confident opinion that absorption will after a time occur, and that the compressed pulmonary tissue will expand and perfectly resume its functions. And even if the quantity should be very large, there is always a possibility that the patient's ultimate recovery may be complete, although one is not justified in predicting it. Percussion usually affords the earliest indication of the subsidence of pleural effusion. The dullness becomes less extensive and less absolute, not only in front, but also over the upper part of the lung behind. A little later, the displaced organs may return to their proper situations, and the side may fall back to its proper dimensions. A feeble vesicular murmur may then be heard where none had been discoverable before. But with regard to this, there is a source of fallacy which must be borne in mind. At a certain stage of pleurisy, even while the affected lung still remains completely flattened and airless (as subsequently appears from an autopsy), the inspiratory sound from the opposite lung is very apt to be carried across the spine in such a way as to suggest that air enters both sides of the chest. Why this should be the case when the disease is advanced, and not at its commencement, I do not altogether understand; perhaps it is because the respiratory movements are at first so shallow. Another physical sign which commonly attends the absorption period of pleural effusion is what is termed a "redux rub." This exactly resembles the friction sound of the earliest stage of the disease, differing (if at all)

only in being still louder and in being heard over a more extensive area. It often remains audible for several days or even weeks together. I have known it to be accompanied by a return of pain in the side without there being any reason to suspect a recrudescence of the inflammatory process.

Even when the attack has so far subsided that the patient is discharged from treatment, and, perhaps, resumes his occupation, it often happens that the side still remains more or less dull on percussion, and that the breath sounds over the affected part are much feebler than natural.

But there are other cases in which pleural effusion, if left to itself, instead of undergoing absorption, perforates the serous membrane and makes its way out of the body. The liquid is then always purulent; the disease is an *empyema*. Sometimes it escapes through the lung and into the bronchial tubes. If this should occur while the patient is asleep, or if he should be in an exceedingly feeble state, so as not to be able to expectorate, he may be instantly suffocated. But surprisingly large quantities of pus are sometimes ejected, with much less distress than might have been anticipated. If the opening leads directly into the bronchial tubes, air passes into the pleural cavity to take the place of the liquid, and a "pyopneumothorax" is established, the effects of which will be discussed further on. But in many cases no such result follows. This appeared so remarkable to some of the older physicians that they invented, for the purpose of explaining it, the theory that pus was capable of undergoing absorption from the pleura and of being afterward excreted from the bronchial mucous membrane. The true way of accounting for it seems to be one which was first suggested by Traube in 1872 ("Ges. Abhandlungen," vol. iii, p. 44). He pointed out that if the pleura alone is eaten through, the alveolar texture of the compressed lung may allow pus to be forced through it by violent coughing, while it yet fails to afford a passage to air in the opposite direction, especially as there is little or no movement of that side of the chest during inspiration. In one case he found post-mortem that the visceral layer of the pleura had been destroyed over a surface an inch broad by two and a half inches long, but that the pulmonary tissue was simply laid bare, there being no discoverable opening into a bronchial tube. Such cases often end in recovery, as Hippocrates long ago remarked.

In other cases, but far less often, an empyema makes its way outward through the parietal pleura. An intercostal space is then usually perforated, and a soft, elastic swelling, of greater or less size, forms beneath the skin, which ultimately becomes reddened, ulcerates through, and allows an enormous quantity of pus to escape. At the present day one seldom has an opportunity of observing this result of pleurisy, because scarcely any practitioner now fails to diagnose a large pleuritic effusion and to treat it surgically. The point at which perforation is most apt to occur has recently (in the "*Lancet*" for 1882) been accurately defined by Mr. Marshall as being in the fifth space, below the nipple; here there is a weak spot in the chest wall, covered only by the internal intercostal muscle and a thin layer belonging to the great pectoral and the external oblique muscles. But its situation may be elsewhere, and is sometimes as high as the second space. On the other hand, the diaphragm may be perforated by an empyema. It does not in this case always happen that an acute, diffused peritonitis is set up, which would, of course, prove rapidly fatal. In 1865 a man died in Guy's Hospital of an empyema, which had been tapped eleven days previously. A hole, which had a diameter of a quarter of an inch, was found in the fleshy substance of the diaphragm, and below it was a large circumscribed abscess.

Again, the pus may make its way backward and point in the loin. In 1858 a boy nine years old was in the hospital for pleurisy, and was discharged convalescent. Afterward he came back with a swelling which, as it pulsated

and was situated in the left lumbar region, was at first suspected of being an aneurism. However, it proved to be an abscess and was punctured. Two months later the boy died of tubercular meningitis, and an autopsy showed that the left lung was still contracted, and that a sinuous channel, six inches long, extended down from the pleural space behind the diaphragm to the external opening. Cases have been recorded in which the pus from an empyema has burrowed until it actually reached the popliteal space.

Except in those cases, already alluded to, in which the pus escapes through the pulmonary tissue, the spontaneous discharge of an empyema is almost always followed by a protracted illness, and very generally ends in the death of the patient. The entrance of air into the serous space often renders the fluids contained in it putrid, and this leads to irritative fever and to more or less rapid emaciation and exhaustion. Otherwise, a pleural fistula may go on discharging for five, ten and fifteen years, or even longer, until lardaceous changes develop themselves in the abdominal viscera and cause death by renal dropsy. The only chance of recovery in cases of this kind seems to be that the whole cavity should be gradually obliterated by the abundant formation of granulation tissue, and by the contraction of the dense fibrous material which becomes developed from it. This indurated substance sometimes reaches the thickness of an inch. At the same time, all the structures which surround the pleura become dragged inward so as to reduce it within the smallest possible limits. The ribs fall in and may almost come into contact with one another; they remain motionless during inspiration, or (as was once observed by Dr. Gee) their anterior parts may actually recede and move backward each time that the healthy half of the chest expands and draws the sternum forward. The dorsal spine becomes curved, so as to present a concavity toward the affected side. The shoulder sinks, the diaphragm is dragged upward, with the abdominal viscera beneath it; the mediastinal structures are pulled over, the heart especially being often brought so widely into contact with the inner surfaces of the ribs that its impulse can be seen and felt over a far more extensive area than under normal circumstances.

Treatment.—It is evident, from what has been stated in the last few paragraphs, that the treatment of pleurisy is a very important matter. There are cases which, left to themselves, run a course as favorable as could possibly be desired. There are others which, even if they do not end fatally, leave the patient crippled and deformed, worn out and emaciated by the drain of a constant purulent discharge. The problem is to modify the progress of these latter cases so as to make it resemble that of the former ones.

Except in the mild, adhesive form of the disease, a person affected with pleurisy should be kept strictly in bed during the early stage. For the relief of pain, the application of a blister has always appeared to me more serviceable than anything else. I believe it may be used with advantage at any period, even when the inflammation is advancing. Cupping, however, may sometimes be first employed, if the patient is robust. Among drugs, those most commonly prescribed are purgatives, diuretics (such as digitalis, acetate of potass, nitrate of potass), and absorbents (especially iodide of potassium internally and diluted mercurial ointment externally). I am convinced that I have several times seen marked results from the application of mercurial ointment, a rub which had been heard day after day for weeks ceasing almost as soon as it was used, or fluid effusion clearing away after a somewhat longer period. The diet should be light and spare. I do not remember to have observed any striking success from the plan of allowing the patient very little to drink, which has been advocated by Niemeyer, and which I often saw practiced at Guy's by Sir William Gull. However,

Fräntzel remarks that a very rapid absorption of fluids from the chest has been sometimes noticed when the patient has been attacked by cholera.

Whenever the physical signs indicate that there is considerable liquid effusion into the pleural cavity, the question of removing it by the operation of thoracocentesis must be carefully considered. This procedure, it is interesting to know, dates back to Hippocrates. But, of course, nothing that could be written about it before the time of Laennec is of any practical value now. And although Trousseau advocated it very strongly as far back as 1843, scarcely any physicians, until within the last twenty years, understood its importance or the desirability of frequently resorting to it.

If percussion shows that one side of the chest is full of fluid, tapping should be performed at once, on account of the danger of sudden death which attends this condition, as pointed out at p. 937. One should never wait even until the following day; and it is important to remember that the absence of obvious distress of breathing affords no proof that the operation may safely be postponed. Whether the temperature is high or not makes no difference whatever.

If percussion shows that the quantity of fluid is moderate, it is often advisable to wait for a few days before performing paracentesis. The presence of pyrexia is, at an early stage of pleurisy, a reason for delay, since it may fairly be hoped that, by the end of two or three weeks from the commencement of the attack, the temperature will fall. When one decides to put off the operation, one must, of course, watch the patient very closely, observing whether the amount of effusion undergoes increase or diminution as time goes on. Unless it begins to diminish in the course of a fortnight, no further postponement of tapping is, I think, generally permissible. For, even though the quantity of liquid should remain quite unaltered, the probability that the lung will quickly expand and regain its functions after paracentesis must obviously become less and less, in proportion to the length of time during which it has become compressed, and during which the layers of lymph that may have bound it down have been allowed to organize themselves and to contract. Another point of great importance is that the withdrawal of a part of the fluid by operation seems often to facilitate the absorption of the rest of it. The supposition is that the sub-pleural lymph channels are mechanically pressed upon by the liquid and that the flow through them is interfered with. At an advanced stage of the disease the fact that the temperature is high is, I think, an additional reason for tapping the chest without further delay, especially if there is fever of a hectic type. For, as Kussmaul originally pointed out in the "*Deutsch Arch.*" for 1868, the removal of pus from the pleural cavity, especially if fetid, is often at once followed by the cessation of pyrexia. Of this I saw a most striking instance, in 1881, in a man who had pleurisy as a sequel of enteric fever. His temperature rose every afternoon to between 102° and 103°, falling in the night and morning, I had made one unsuccessful attempt to draw off the effused fluid, the reason of my failure being that I used a very fine aspirator needle, because the area of percussion dullness was not in the usual position behind, but at the side of the chest, just outside the situation of the pericardium, so that I felt some hesitation in acting upon my diagnosis. However, six days later, when I visited him, I found him in a most critical condition, with great anxiety of face and with extreme distress of breathing. As the only chance of saving his life I had a somewhat larger trocar plunged into the chest at exactly the same spot as before. Aspiration was then performed, but at first no fluid appeared. However, I took the instrument and pushed it inward, feeling, as I did this, that the end of it encountered and seemed to pass through a resisting membrane. A quantity of rather viscid, blood-stained liquid at once escaped,

and of this four and a half ounces were withdrawn. The patient was instantly relieved, and from that time went on to recovery without a bad symptom. His temperature, which was rising at the time of the operation and had reached 100.8°, fell from that very moment; three hours later it was normal, and during the next three days it only once reached 99.4°.

When it is decided to tap the chest, some physicians have recommended that a preliminary puncture should be made, by means of an empty hypodermic syringe, into the chamber of which some of the pleuritic liquid may be drawn. But it often happens that this procedure leads to no result, even though the diagnosis may be quite correct, and I think that the use of such an instrument is not to be advised, at least when the condition of the patient is so serious as to make the removal of the effusion a matter of importance. For either liquid enters the syringe or it does not. In the former case a larger trocar is at once employed, and the patient might as well have been saved the slight pain of the preliminary puncture. In the latter case one is very much hampered in taking any further step, which, perhaps, is nevertheless essential to his safety.

The aspirator is greatly to be preferred to an ordinary trocar for paracentesis of the chest, for through a trocar fluid will only escape from the pleural cavity when the pressure there is greater than the atmospheric pressure. Several pints may be present, and yet sometimes there may be only a momentary flow of it during the act of expiration, or when the patient happens to cough. Moreover, there is great danger of air being sucked back into the serous space by a deep inspiration. Some of those who advocated the operation a few years ago maintained that the introduction of air is a matter of but little importance, on the ground (which cannot be contested) that it has often occurred without ill effects. But at the present day few surgeons would deny that it must involve the risk of giving a septic character to the inflammatory process. However, one can without difficulty prevent it by surrounding the mouth of the trocar by a piece of moistened gold-beater's skin, which acts as a valve; or, as has been usual in England, one can fit on an elastic tube and make it dip beneath the surface of water containing carbolic acid, in a basin, on the floor.

Even when the aspirator is used, there is often a good deal of difficulty in getting out any considerable quantity of the liquid. Pieces of lymph are drawn against the inner orifice of the tube, or its channel may be occluded by viscid or curdy pus. Sometimes one can restore the flow by moving it in various directions. If this fails it may be necessary to withdraw the instrument and to make a second puncture at a different spot. It should, I think, be a rule, to which no exceptions are allowed, that one should never employ for tapping the chest the hollow needles which are commonly sold with the aspiratory apparatus. One expects the lung to be expanded as the fluid is withdrawn, and there must be a very great risk of its being wounded if there be a sharp point in the way. A perfectly safe instrument may now be obtained without difficulty. It consists of a trocar and cannula, the cannula having a lateral opening, to which the tube of the aspirator is fitted; there is also a stopcock which can be turned at the moment when the trocar, having punctured the chest, is being withdrawn. Another advantage of this instrument is, that should the cannula become obstructed, a blunt probe, fitting loosely to the cannula, can be passed in, so as to clear it out, without having to detach the aspirator tube.

The best spot for puncturing the chest is generally said to be about two inches outside the edge of the pectoralis major, and just above the edge of the sixth rib on the left side or of the fifth rib on the right side. At Guy's Hospital I think it has been usual to select a lower intercostal space, and a

point much nearer to the posterior fold of the axilla. Dr. Bowditch recommends that the instrument should be introduced between the ninth and the eleventh ribs. The reason for keeping close to the upper edge of a rib is that one is then not likely to wound the intercostal artery. In 1855 this accident happened during an operation performed at Guy's; the patient (who had phthisis) became faint at the time and died the same evening; a pound of clotted blood was found in the base of the chest. Fräntzel speaks of the use of a "capillary" trocar as obviating all risk of such an occurrence, but I have never heard of another instance of it, and from what I have seen of the difficulty of extracting the fluid in many cases I should not be disposed to recommend that too small an instrument should be employed.

It is not desirable in performing paracentesis to attempt to empty the pleural cavity. Fräntzel says that not more than about two pints and a half should be withdrawn at once. In many cases, during the operation or immediately afterward, paroxysms of cough occur which are best relieved by a hypodermic injection of morphia. As Fräntzel remarks, they are doubtless set up by the re-entry of air into the tubes of the lung which had been compressed. That they are not due to the contact of the cannula with the surface of the lung is, he says, shown by their being sometimes produced when the quantity of fluid is still so great that such contact cannot have taken place. Moreover he has repeatedly felt the orifice of the instrument rest against the pulmonary pleura, without any cough resulting. Since he has used the aspirator and drawn off pleuritic effusion very slowly, he has very seldom observed these severe fits of coughing. Once, in a phthisical patient, he has known thoracocentesis to be followed by fatal hæmoptysis after an interval of eight hours; this was due to the rupture of an aneurism in a vomica. It is easy to see that the increased activity of circulation in the pulmonary vessels, which must be one result of the operation, is very likely to lead to the giving way of any weak spot in their walls. A much more frequent occurrence is œdema of the pulmonary tissue on the affected side. This seems to be the cause of a phenomenon which attracted much attention in Paris about ten years ago—the expectoration after thoracocentesis of large quantities of a frothy liquid containing much albumen. The patient to whom this accident happens may have experienced the usual relief from the operation; but after an interval of from ten minutes to an hour his breathing becomes distressed, he begins to cough, and he may turn livid, and die in a quarter of an hour. Twenty-one instances of it were collected by Terrillon in a monograph published in 1873; most of them, however, ended in recovery, and some were comparatively slight. The close resemblance between the fluid discharged from the air passages in such cases and that withdrawn by the trocar so short a time before seems to have led some distinguished French observers to suppose that the expectorated albumen was dependent upon the presence of a communication between the cavity of the pleura and the interior of the lung, their notion being either that the lung was wounded during the thoracocentesis or that a perforation existed previously, which became opened out as the pressure was removed from the surface of the organ. But, as Terrillon had no difficulty in showing, such opinions are quite untenable, and the only reasonable explanation is that there is suddenly produced an active hyperæmia which leads to œdema of the pulmonary tissue.

Among the objections that have been urged against the performance of thoracocentesis, except in cases of absolute necessity, one which had the support of Dr. Stokes, of Dublin, and of Sir Thomas Watson, is that it may lead to the conversion into pus of an effusion originally sero-fibrinous. And although this suggestion is altogether rejected by Trousseau, I doubt

whether he is right. Nothing is more likely than that the vessels in an inflamed pleura are often weakened and dilated to such an extent that a sudden and great removal of pressure from them may cause increased exudation. And it is certain that in many cases in which a clear fluid is withdrawn at a first operation, pus appears on the second or third occasion. Thus, although it accords with the natural tendency of the disease that as it advances the proportion of leucocytes in the exudation should become greater, one cannot but admit that this process may sometimes be hastened by the performance of an operation. Fräntzel speaks of a burning pain in the seat of puncture and of an indefinite sense of pressure on the affected side as being generally present and as lasting in some cases for a day or two. He also says that thoracocentesis is usually followed by a slight elevation of temperature, and that until the second, third, or fourth day a more or less considerable increase of effusion may be observed, after which a somewhat rapid process of absorption sets in, attended with diuresis. How such statements are to be reconciled with the fact that an existing pyrexia is often suddenly cut short by the operation I am not able to say. My experience has been that most patients experience marked relief from the operation, and are in every way more comfortable after it than before.

I have already alluded to the circumstance that in certain cases the fluid quickly accumulates again after paracentesis, so that the patient's condition becomes as bad as before, and that the operation has to be repeated. When this happens two or three times in succession, perhaps at intervals of only a few days, Fräntzel advises that one should desist from further interference, as the downward course of the case will but be precipitated by it.

If the fluid withdrawn from the chest by paracentesis is purulent, the further treatment requires to some extent to be modified. At the time the best course is generally to close the puncture with lint and plaster, for sometimes what remains of the effusion afterward undergoes absorption. Cheesy masses, and even thick, hard deposits of calcareous matter are now and then found after death lying between adherent pleural surfaces; and I think there can be no doubt that such residues have usually, if not always, had their origin in an empyema. In other cases a collection of liquid pus, enclosed in a dense capsule, has been discovered in the pleural cavity, when the fatal illness was of an altogether different nature. Dr. Moxon alludes to such an instance, in which there was a history of pleurisy three years before, and in which physical signs of fluid in the chest had been observed during the intervening period. Thus it is not a matter of course that one should give a very grave prognosis whenever one has tapped an empyema. But in most cases the effusion quickly reaccumulates, a second operation is required, and after this a third. It is right to allow at least two chances for the subsidence of the disease; and, as Fräntzel remarks, there is the further advantage that, each time one removes fluid, adhesions may possibly form between the opposed surfaces of the pleura so as to narrow the cavity. But when a third puncture becomes necessary, it is generally advisable to alter one's procedure, and to provide a channel by which for the future the cavity may go on draining itself, so that the best possible opportunity may be afforded for its gradual closure by granulations. In children I have sometimes attained admirable results by making an aperture just large enough to admit one end of a long elastic tube, of which the other end is carried beneath the surface of carbolized liquid in a jar placed beneath the bed. The elastic skin of a child grasps the tube firmly, and does not ulcerate. The negative pressure of the column of liquid acting hydrostatically seems gradually to raise the com-

pressed lung. Within a surprisingly short time the flow of pus may cease, and a permanent cure may be obtained. I much regret that I have no notes of several cases which were treated in this way some years ago at the Evelina Hospital. With older patients I have never had similar success. The plan usually adopted at Guy's Hospital is to make rather a free incision, under the carbolic spray, into one of the lower intercostal spaces, to introduce a drainage tube, and to allow the pus to escape into an antiseptic dressing. Injections of iodine, or of permanganate of potass, are often used to wash out the cavity at intervals of twenty-four or forty-eight hours.

A somewhat different method is recommended by Fräntzel. Having, at the time of the operation, got rid of as much as possible of the pus, he pushes a long catheter downward toward the spine, and slowly injects through it distilled water, at a temperature of 100°, until the space is full. He then draws off the water by another catheter with an exhausting syringe, and he repeats this procedure three or four times until what returns is quite pure. Masses of fibrin, sometimes as large as the palm of the hand, generally appear in the wound while this is being done, and are carefully removed. I may incidentally remark that this appears to me to be one of the great advantages of Fräntzel's plan, for in one case I saw much trouble caused at a later period by such masses, which had become putrid. Finally, he fixes in the aperture a flat silver cannula, with a broad plate fitting upon the surface of the chest, of such a size that two catheters can be passed through it side by side; outside this are placed antiseptic dressings; and over them a bag of ice. Each day afterward the pleural cavity is twice washed out by means of the catheters, of which one is introduced with great care to the furthest possible point, so as to prevent any accumulation of pus from taking place. Another detail which he deems very important is that the patient should lie in such a position that the wound is at a higher level than any part of the pleural space, so that the fluid may gravitate into every part of it. After two days a solution of ½ per cent. of common salt is substituted for the distilled water; and later still a very dilute solution of iodine or of carbolic acid. He says that of eleven patients treated in this manner five were completely cured; five died, but most of them from causes which had little or nothing to do with the operation; one was under observation when he wrote, and was doing well.

The operation of washing out the chest is not altogether free from danger. In 1876 Dr. Cayley read before the Clinical Society a case which had occurred to him, and in which, while a solution of iodine was being injected, the patient suddenly became pale, unconscious, and convulsed; the temperature rose to 107°, and death followed in sixteen hours. He cites three cases recorded by French observers, in each of which like symptoms appeared, though one of them ended favorably. In 1874, at Guy's Hospital, a girl, aged sixteen, died in precisely the same way. She had had a drainage tube inserted into the right chest for an empyema five weeks before, and was going on well. One day she was sitting up, and her chest was being washed out with carbolic acid, when she suddenly ceased to breathe, and, although artificial respiration was set in action, remained unconscious, with muscular twitchings, until death. Nothing has been found, on post-mortem examination, to account for such accidents. A suggestion that thrombi in the pulmonary veins may have been dislodged and have formed emboli in the heart or in the cerebral arteries seems to have been shown to be without foundation. It is noteworthy that in every one of the cases the chest had been washed out many times before without any ill effects occurring; the only difference being that in two instances a somewhat larger quantity of fluid was being injected than usual. Fräntzel hints that it is important that the stream should not be directed toward the

pericardium. In the girl who died at Guy's Hospital it was noted that there was a very slight basal pericarditis, and that the septum between the ventricles of the heart was ecchymosed.

The process by which the sac of a discharging empyema becomes gradually obliterated seems to consist in the formation of granulation tissue and in the union of the two opposed surfaces; in most cases this union probably begins at the root of the lung, and spreads from one point to another until it reaches the external orifice. Fräntzel suggests, and I think with probability, that the compressed lung is re-inflated with air from the opposite lung during the act of coughing, or as the result of simple expiratory efforts with closed glottis.

But in many cases the cure of an empyema remains incomplete. The cavity may have shrunk to very narrow dimensions, the chest may have regained a fair amount of resonance over a large part of its surface, air may enter the lungs pretty freely, but there is a fistulous opening from which small quantities of pus continually drain away. In such cases, and generally when other treatment has proved unsuccessful, it has recently been the practice to excise portions of one or more ribs, so as to allow the side of the chest to fall in and meet the lung. This operation seems to have been first performed by Dr. Peitavy. In the "*Birmingham Medical Review*" for 1880, Dr. William Thomas has recorded several cases which were so treated, and in almost all of them the result seems to have been highly satisfactory, the wound healing in a few weeks, and the lung rising completely. The rib, too, was restored by a new growth of bone. It is to be observed, however, that the patients were all children under eight years old. In 1877 Mr. Howse excised portions of three ribs from a child, aged six, a patient of Dr. Taylor, in the Evelina Hospital, whose case may be found in vol. xiii of the Clinical Society's "*Transactions*." An interesting point was that the seventh and the eighth ribs were found at the time of the operation to be nearly united together by bridges of bone, which had formed round the track of a drainage-tube that had been lying for a considerable time between those bones. Some improvement followed, but the cavity of the empyema did not close, and ultimately the child died with lardaceous organs.

Diagnosis.—As a rule, the recognition of pleurisy is easy, being based directly upon the characteristic physical signs. But some medical men are far too ready to set down to this disease cases in which there is no symptom except a pain in the side, perhaps of a neuralgic or myalgic character. In hospital practice I have learned to distrust the statements of patients, when they tell me that they have been under treatment elsewhere for "pleurisy." It has sometimes happened that an eruption of shingles has escaped notice, not having been looked for; and costal periostitis and abscess of the chest wall are other affections the possible presence of which must not be forgotten. The only case in which it is allowable to diagnose pleurisy without positive evidence from percussion or auscultation is when violent pain in the lower part of the chest is accompanied by pyrexia. A fair supposition, then, is that there is inflammation of the upper surface of the diaphragm and of the corresponding surface of the lung.

Even when one thinks that one hears a pleuritic rub, there is sometimes need of caution. Dr. Gairdner has recorded an instance in which a sound which he describes as having a shuffling character, attended with a tactile sensation as of a jerking movement, produced by something rubbing up and down against the walls of the chest, proved to be due to emphysema of the lung. And Guttmann cites a case of Jürgensen's, in which a similar effect was produced by tubercles projecting above the surface of the pulmonary pleura.

A much more common mistake is that of attributing to pericarditis a friction sound which is really pleuritic. This point, however, will hereafter be fully discussed.

Pleuritic effusion has sometimes been diagnosed when the disease (if on the right side) has been a *hydatid* in the liver, or a hepatic *abscess*, or a hypophrenic abscess, or when (if on the left side) it has been an abscess connected with the spleen. Again, it is remarkable that all the examples of very large chronic *pericardial effusion* which have occurred at Guy's Hospital have been set down to pleurisy. But it is probable that one might always be put on one's guard by carefully mapping out the area of percussion dullness, and by noting exactly how far it extends in front, at the side, and behind. To say that the disease might not be a circumscribed empyema would, indeed, be impossible; but at least one would be saved from imagining that the fluid lay free in the pleural cavity. I may take this opportunity of remarking that, when there is a very large effusion of pus into the left side of the chest, pulsation synchronous with the heart can sometimes be felt in the intercostal spaces near the nipple or above it and toward the clavicle, so that the presence of an *aneurism* may be suspected. A case of this kind was recorded three centuries ago by Baillon; its real nature was cleared up by the bursting of the swelling with discharge of pus from it. In our own time Dr. Walshe has studied "pulsating empyema," as he terms it; and Traube threw out the suggestion that the existence of pericardial (in addition to the pleural) effusion might, perhaps, aid in giving it its peculiar character by facilitating the movement of the heart from left to right during the systole, and so increasing the force of its impact against the structures adjacent.

Again, all the physical signs may point to the presence of a large pleuritic effusion occupying the lower and back part of the serous cavity, and yet the diagnosis may not be absolutely free from possible sources of error. Traube has related a case of acute thoracic disease in which he imagined that there were both hepatization of the left lung and exudation upon its surface, but in which an autopsy showed that the serous cavity had been closed by former adhesions; the great diminution of tactile vibration in this instance was attributed to plugging of the smaller bronchial tubes by lymph. Most English observers followed Laennec so far as to think that the detection of ægophony is conclusive evidence that there is at least some fluid effusion into the pleura. Fräntzel, however, declares, as the result of careful observation directed to this question for some years, that such an opinion is incorrect, as had, indeed, long ago been asserted by Skoda.

But in chronic cases a more serious error may be committed—that of mistaking for pleuritic effusion a mass of malignant growth. Every physician of experience must either have made this mistake himself or, at least, have seen it made by others. One should, therefore, never give an opinion without having thought of such a possibility; the points to be especially noticed are whether the area of dullness corresponds in shape with that caused by a distended pleura, and whether tactile vibration is or is not still to be felt in certain positions. It must be borne in mind that a new growth situated in the mediastinum or in the lung is often accompanied by effusion into the pleura, so that a very strict differential diagnosis may be, after all, sometimes less accurate than a more doubtful opinion. Fräntzel relates a converse case, in which a large hemorrhagic effusion was for a time supposed to be a solid tumor.

Even when the presence of liquid in the pleural cavity is positively and correctly diagnosed, one must not assume, as a matter of course, that there is pleuritic effusion. A simple dropsy of the serous cavity, for which the technical name is *hydrothorax*, may give rise to precisely similar physical

signs, except that, being probably never altogether unilateral, it is not likely to displace the heart. Again, pure blood may fill the pleura, constituting what is termed *hæmatothorax*. Apart from surgical injuries, the chief cause of such an affection is the rupture of an aneurism of the aorta. Sir Thomas Watson mentions a case in which caries of a rib led to destruction of the wall of the intercostal artery and to distention of that side of the chest with blood, a large part of which was clotted in concentric layers.

Complications.—Pleurisy is generally attended with more or less swelling of, and exudation into, the subserous connective tissue. In a case which occurred at Guy's Hospital in 1872, the surface of the lung was covered with reticulated lines, due to the presence of pus in the lymph channels beneath the visceral layer of the pleura. In another case, in 1869, pus was found outside the parietal layer of the pleura, infiltrating the intercostal muscles; there was also in the mediastinum a diffused abscess of the size of a plum. In a third case, in 1873, the mediastinal tissues were infiltrated with a mass of puriform lymph, three-quarters of an inch thick. All three were examples of double pleurisy, accompanied by pericarditis. Cases differ very much as regards liability to the spread of inflammation from the pleura to adjacent serous membranes. So far as I know, the pleurisy which so constantly arises in phthisis never extends to the pericardium. But the more intense and violent forms of the disease are exceedingly apt to set up pericarditis. Moreover, there are cases in which both pleuræ, the pericardium, and the peritoneum seem to become simultaneously inflamed, or at least in which it cannot be determined that any one of them was affected earlier than the rest. As a rule, such cases present very acute symptoms and run a rapid course. But this is not invariable. In 1876, a girl, aged twenty-three, was admitted into Guy's Hospital with what was supposed to be enteric fever. After a fortnight fluid effusion was detected in the left pleura, and with the aspirator forty-two ounces were drawn off. Her febrile symptoms continued; she became emaciated and died. As may well be understood, it was thought toward the last that she was suffering from some obscure tubercular disease. However, on making an autopsy I could discover no tubercles anywhere. But, besides the pleuritic effusion on the left side which had been diagnosed, there was lymph over the whole of the right pleura; the pericardium was adherent by a recent plastic exudation; the liver and the spleen were fixed to the diaphragm by a similar material; and the lower part of the abdominal cavity contained a small amount of purulent fluid. It may be that the disease was of rheumatic origin, for the girl was said to have had a painful affection of her joints about two months before; but there was no endocarditis, the presence of which would, I think, have been almost conclusive as to the correctness of this hypothesis. The case appears to me to be of great interest, not only pathologically, but also on account of its obscurity during life. For I have repeatedly found the thoracic serous cavities and the upper part of the peritoneal space all closed by old adhesions, when there had been no history of any chest affection; and it would now seem that such a result may arise from an illness which clinically might be taken for fever. It has been laid down that, whenever the two pleuræ are attacked with inflammation simultaneously or in succession, one ought to suspect that there is some underlying condition, such as Bright's disease or latent tuberculosis. But I think that a good many exceptions to this rule are met with in practice.

Ætiology.—I have left to the last the causes of pleurisy; because the symptoms, the diagnosis, and the treatment of the disease can be studied

without reference to them, whereas they sometimes affect in a very important manner its ultimate issue.

Foremost among them is *cold*. This has been so commonly set down as giving rise to all forms of internal inflammation, and often with so little reason, that one cannot be surprised that many physicians should be reluctant to recognize its operation in any case whatever. But I quite agree with Fräntzel that the clinical evidence in proof of the direct dependence of pleurisy upon cold is of the most striking character, and such as cannot possibly be explained away. He cites, for example, cases of persons who have been attacked after having exposed one side of the body to a draught in changing their clothes while heated, or after having got one side wet through in a driving rain. Other cases have immediately resulted from sitting near an open window or by a badly-fitting door, especially during convalescence from some acute illness or (it is said) after a course of mercurial treatment.

Injuries to the chest often give rise to pleurisy. Not only does this occur when the ribs have been broken, but also when there is no evidence of any damage to the parietes of the thorax.

Certain general diseases are very apt to be attended by pleurisy as a complication. This is the case, for example, with acute *rheumatism;* but I am not aware that inflammation of the pleura, when it is of rheumatic origin, is ever a primary and an independent malady, as not infrequently happens with pericarditis or endocarditis. Scarlet fever, again, must be mentioned as a rather frequent cause. On the other hand, Fräntzel lays down the rule that enteric fever never becomes complicated with pleurisy during its early stage, when the morning temperature is not below 102°. He says that he has been saved from many mistakes by bearing this fact in mind; and I myself have recently met with an instance in which a young lady, who had obvious signs of pleuritic effusion, was erroneously supposed to be also passing through an attack of fever, so that her condition caused a great deal of speculation and anxiety which (if Fräntzel is right) were altogether uncalled for. Among visceral affections, none is so commonly accompanied by pleurisy as Bright's *disease of the kidneys;* indeed, one should always make it a rule to test the urine for albumen.

Lastly, a secondary pleurisy is often due to the direct extension of inflammation from some adjacent structure. To the pathologist there is probably no condition which is so familiar as this; but the affection is in many cases found to be quite recent and early at the time of death, so that it has no clinical significance. Among the less obvious starting points for a severe or even fatal pleurisy may be mentioned abscesses in the armpit, operations upon the breast, suppuration of the cervical connective tissue after tracheotomy, caries of the ribs, mediastinal abscesses, cancer of the œsophagus, and caries of the dorsal vertebræ. Or its origin may be below the diaphragm, the lymph channels discovered by v. Recklinghausen probably conveying the inflammatory process from one serous cavity to another. Thus, I have seen cases in which pleurisy appeared to be the immediate cause of death, and in which it was due to extension from a puerperal peritonitis, or from a peritonitis following ovariotomy, or arising from disease of the rectum. Again, I have met with instances in which a very acute inflammation of the pleura, with fetor of the pus, started from the upper end of a psoas abscess, or from a localized abscess behind the stomach, due to a perforating gastric ulcer.

But by far the most common causes of a secondary pleurisy are affections of the lungs. It is unnecessary for me to insist on the fact that exudation upon the serous surface occurs in every case of acute *pneumonia*, and in almost every case of heart disease in which pulmonary infarcts are formed, for then the pleurisy is rarely of clinical importance. On the

other hand, in pyæmia (especially when resulting from thrombosis of cerebral sinuses, itself consequent upon disease of the temporal bone) pleurisy is sometimes the most conspicuous feature of the case, and may be mistaken for the primary disease and the cause of all the patient's symptoms. Still more important is it to bear in mind that what seems to be an uncomplicated and simple attack of pleurisy may really be dependent upon an extensive pneumonia, of which there may be little or no clinical evidence. When pleural effusion has once taken place, it may be impossible to discover by physical signs the presence of hepatization of the corresponding part of the lung. It often happens that the characters of the sputa afford the only clue to the real nature of the case, or a correct diagnosis may depend entirely upon one's having seen the patient at an earlier period before the fluid was poured out into the serous cavity. Again, it is not improbable that a very limited patch of pneumonia, involving the surface of the lung, may sometimes be the starting point of a diffused and severe pleurisy.

It is in regard to *phthisis* that this question is of the greatest importance, on account of its bearing on prognosis. One is frequently seeing patients who, having favorably passed through an attack of pleurisy, are shortly afterward seized with hæmoptysis, or develop signs of tubercular disease of the lungs. Sometimes, no doubt, the presence of such disease can be detected even while the serous inflammation is in progress, if one is careful to examine the upper lobes thoroughly, and I always make a point of doing so before I express an opinion as to the probable issue of a case of pleurisy. But in many cases all the clinical evidence points to the conclusion that the pulmonary affection has been of later development. Those who adopt Buhl's infective theory of tubercle can then, of course, maintain that it is really secondary, having been due to the absorption of caseous matter into the blood (*cf.*, p. 89).

A somewhat different point is whether pleuritic effusion, while it continues to compress one of the lungs, favors a fresh development or a further growth of tubercles in that organ, or whether it may not rather be adverse to such an occurrence, even though it may increase the susceptibility of the other lung, which has to perform extra work. I have notes of six cases bearing on this question. In one instance there were no tubercles except in the opposite lung; in another, the tubercles were much less numerous on the side of the pleurisy; in two others, it was observed that on that side they were all of old date and inactive. On the other hand, there was one case in which they were more abundant in the compressed lung than in the opposite one, and once a lung entirely airless was actually full of tubercles (some of which were gray and others caseous) in its lower lobe, where their presence, the apex being healthy, is an occurrence so exceptional that one could hardly doubt that the pleurisy had determined their formation. Precisely the same question has been raised in regard to the effect of the supervention of pneumothorax on phthisical disease of the lung.

According to Dr. Stokes, hectic fever sometimes subsides when perforation of the pleura occurs, and I shall have to mention at least one case of pneumothorax in which the patient recovered, notwithstanding that he had previously had symptoms of phthisis.

PNEUMOTHORAX.

ORIGIN AND PATHOLOGY—PHYSICAL SIGNS—DIAGNOSIS—SYMPTOMS—TREATMENT—PROGNOSIS.

Some of the older pathological anatomists, including Morgagni and Merkel, appear to have made mention of the fact that air sometimes accumulates in the cavity of the pleura; but the term Pneumothorax was first used in 1803 by Itard, a pupil of Bayle. It was, however, left to Laennec to give a full description of this affection, and to indicate various ways in which air may pass from without into the serous cavity. Since his time most writers have admitted that in exceptional cases gases may be formed there as the result of chemical decomposition of liquid effusion, or perhaps by direct secretion from the lining membrane. Such notions, however, accord ill with the general doctrines that are now held by almost every one. And as neither of these supposed causes of pneumothorax has in its favor the slightest clinical evidence, we may now, guided by the experience of more than half a century, reject them altogether, and assume that air is never found in the interior of the pleural space except as the result of a breach in the continuity of its surface, placing it in more or less direct communication with the external atmosphere.

Seeing how delicate are the structures which separate the pulmonary alveoli from the surrounding serous space, one cannot be surprised that in the immense majority of cases pneumothorax is a consequence of perforation of the visceral layer of the pleura, allowing air to escape from the lung. Very often this arises from direct violence. Broken ribs are exceedingly apt to wound the lung, and in persons who are run over or severely crushed the organ may be torn without there being any fracture of bone or laceration of the costal pleura. So, again, pneumothorax may be produced by powerful muscular efforts, even when the lung has not been previously diseased. Thus, Fräntzel relates, in Ziemssen's "Handbuch," a case in a lad, aged nineteen, who was exerting all his strength to push a heavy cask, when he felt something give way in his chest, and became suddenly short of breath and powerless. As he recovered entirely within six weeks, without any lung affection having been discoverable, it may probably be assumed that none existed. A similar accident sometimes happens during the violent straining which attends the paroxysms of whooping-cough. The fact that the bullæ in emphysema of the lungs often have the thinnest conceivable walls might naturally lead one to anticipate that pneumothorax should occasionally arise from their rupture. But this must be exceedingly rare, and I believe that writers admit it only when the so-called interlobular emphysema is present. As a general rule, the entrance of air into the pleural space is the result of some local inflammatory affection of the lung, leading to ulceration or sloughing of the pulmonary pleura. Sometimes, though but seldom, the disease is a simple acute pneumonia running on to gangrene. Much more often it is a sloughing infarctus, dependent upon infective emboli, such

as are carried to the lungs in cases of ear disease, or in a great variety of surgical affections. Sometimes it is a "bronchitis fœtida," with sacculated dilatations of the tubes. It may even be an abscess starting from the glands at the root of the lung, and making its way in succession into a bronchus and into the pleural space. But such cases are after all exceptional. By far the most common cause of pneumothorax is the giving way of a superficial vomica in phthisis. Walshe estimates that nine out of ten cases arise in this way, and Fräntzel carries the proportion still higher, putting it at fourteen to one. It would occur very much oftener than it does but for the adhesive pleurisy, which generally advances *pari passu* with chronic pulmonary disease, slowly sealing up the serous cavity. Indeed, even when there is no evidence of phthisis either before or afterward, it is a question whether the spontaneous development of pneumothorax, independently of any violent muscular effort, should not generally be attributed to the rupture of a small tuberculous cavity, by which (as sometimes happens) neither physical signs nor symptoms have been produced. A case in point was related by Prof. Vogel, of Dorpat, in vol. ii of the "*Deutsches Archiv.*" A woman, aged twenty-nine, became suddenly the subject of pneumothorax one morning at nine o'clock. All that could be made out as to its possible causes was that some months previously she had had a slight, loose cough, and more recently a little pricking pain in the region of the liver; when the attack began she was engaged in turning over her baby's mattress, and just before she had been lifting its bath, which was rather heavy. Vogel himself was inclined to think that she had had latent tubercular disease.

Another way in which pneumothorax arises is by perforation of the visceral pleura from without, as when an empyema discharges itself through the air passages. In medical practice this cause comes next to phthisis in order of frequency. Perhaps it is also possible for pleurisy to give rise to pneumothorax at an earlier stage, if the inflammation is sufficiently intense to lead to sloughing of the visceral layer of the serous membrane. Thus, in 1869, a man, aged forty-two, was brought into Guy's Hospital with a very severe chest affection, under which he was said to have been laboring for a fortnight; he died half an hour after his admission. He was found to have acute pericarditis, mediastinal inflammation, and early pleurisy on the left side. But the principal seat of disease was the right pleural cavity. This contained fetid gas, and four and a half pints of dirty, purulent fluid. In the upper lobe of the lung there were two openings, and through these air had doubtless entered. But the pulmonary pleura was gangrenous over an area of two square inches, and the substance of the lung beneath it to a depth of half an inch, the affected part being bounded by a yellow border. That the pleurisy was of exceptional severity was also evident from the fact that there was suppuration outside its parietal layer, involving the intercostal muscles.

There still remain cases in which the air is not derived from the lung at all, but directly from the outside of the chest, or from some part of the alimentary canal. As a consequence of perforation of the thoracic walls, pneumothorax is scarcely ever seen by physicians except when an empyema has broken through spontaneously, or has been let out by operation. And when the pus points of its own accord, the channel by which it reaches the surface is commonly oblique and indirect, so that air fails to find its way along it. Dr. Moxon has drawn attention to the possible occurrence of double pneumothorax as the cause of death after tracheotomy, subcutaneous emphysema extending down from the wound so as to fill the mediastinal connective tissue with air, which then bursts into the serous cavities. One such case occurred in a woman, aged thirty-three, who died in less than twenty-four hours after the operation. Emphysema had spread over the

neck, chest, and arms as far as the fingers. Both lungs were found collapsed and almost airless.

The part of the alimentary canal which is most often the starting point of pneumothorax is the œsophagus; a malignant growth may eat its way into the serous cavity, or the ulceration due to a foreign body may have a like result. But sometimes a gastric ulcer, after setting up a circumscribed hypophrenic abscess, has led to perforation of the diaphragm; and a hydatid cyst of the liver has been known to open communications in two opposite directions, with the bowel below and with the pleural space above.

The recognition of pneumothorax is not always a perfectly simple matter, even in the dead body. In making an autopsy, at the moment when the knife is first plunged into the thorax, the air can sometimes be heard to rush out; or, if a puncture is made with a trocar, it may escape in a jet, so as to blow out a lighted match. But this occurs only when its pressure is greater than that of the atmosphere, which is by no means generally the case. In all probability the existence of air in the pleural space is very often overlooked in ordinary post-mortem examinations, especially in the bodies of phthisical patients, in whom, from their having extensive adhesions, the collapse of the lung has been only partial. The best way of making sure whether there is pneumothorax or not is to puncture the chest under water, which may be done either by dissecting off the tissues from the ribs so as to form a pouch that can be filled with water, or by pouring water into the abdomen and then perforating the diaphragm with a trocar. Or, if pleuritic effusion is present, it may be sufficient to shake the body before opening the chest, after which, if there is any air, the liquid will be found to be frothy.

When the pneumothorax arises from perforation of the visceral pleura, the aperture by which the air entered is sometimes plainly visible; it may be as large as a threepenny piece. Much more often it is covered by recently formed lymph, and the only way of detecting it is to inflate the lung with bellows through the trachea. Or it may have become completely closed by adhesions during the interval that has elapsed between the occurrence of the pneumothorax and the death of the patient, so that there may be no possibility of discovering its position. It is most commonly situated upon the lateral surface of the lung, in the upper lobe near its lower border or in the lower lobe near its upper border. The chemical nature of air withdrawn from the pleural space was investigated by Dr. John Davy many years ago,* and analyses have since been made by other chemists; it has always been found to consist mainly of nitrogen, and the amount of carbonic acid in it has generally been greater than that of the oxygen; sulphuretted hydrogen has been present when the other contents of the cavity were putrid. Dr. Walshe labors to explain the difference from atmospheric air by pointing out that it traversed the lung before reaching the pleura. But it can hardly be said to have passed through pulmonary tissue, and, moreover, its composition is far more altered than that of normally expired air. Obviously, therefore, it must have undergone change while in the serous space, either as the result of the action upon it of liquid effusion, or in consequence of the absorbent energy of the pleural membrane, which is very considerable, as we shall presently see.

When death occurs within a few hours after the development of pneumothorax, the cavity of the pleura is, of course, found empty, there having been no time for the occurrence of effusion. But in other cases, at least such as are seen by physicians, an empyema is, as a rule, formed within a few days. Dr. Walshe has even discovered signs of liquid effusion within twenty-four hours. The most striking instance to the contrary

* [*"Phil. Trans.,"* 1823: and in his collected "Researches," vol. ii, p. 249.—Ed.]

with which I am acquainted is afforded by Vogel's case, already referred to at p. 952. He repeatedly examined his patient during the month after she was attacked, and could never detect the slightest indication of pleurisy. Even when effusion does take place it is not always purulent. Fräntzel speaks of sero-fibrinous exudations as of not infrequent occurrence. In one patient the pleural space gradually became full of fluid without any pyrexia developing itself, until there was no longer any pneumothorax; a puncture showed that the fluid was sero-fibrinous. In another case the same thing happened notwithstanding that a large opening through the lung kept up during a period of three months a free communication between the serous cavity and the external atmosphere. So far as the air itself is concerned, one must indeed suppose that its power of setting up inflammation depends upon the accidental presence in it of "germs." But, as we have seen, pneumothorax is, in a very large majority of cases, due to the rupture of a phthisical vomica into the serous space. The contents of the vomica must generally escape with the air, and they may well be regarded as the cause of the pleurisy which follows. When the original lung affection is a sloughing infarct, or when the pleura is perforated by a malignant œsophageal growth, or by a hypophrenic abscess communicating with the stomach, the consequent inflammation is, of course, peculiarly severe and rapid in its course. On the other hand, in surgical practice, when a healthy lung is wounded by fractured ribs, I believe that pleurisy not seldom remains absent. Probably very much depends upon whether or not the aperture in the lung becomes quickly closed again. For the risk of the entrance of "germs" must be greatly diminished if no air is admitted beyond that which immediately fills the serous cavity. It is clearly impossible for subcutaneous emphysema to be produced by fracture of the ribs without there being also pneumothorax, unless the pleural space at the seat of injury happens to have been closed by former adhesions. But in cases of this kind I have often failed to detect any signs of the presence of air in the serous cavity when a day or two had passed before I had an opportunity of examining the patient. I can, therefore, hardly doubt that air must often very rapidly disappear from the pleural space by absorption. This conclusion is quite in accordance with the results of experiments on animals. Cohnheim says that in rabbits it is not possible by injection of air into the pleura to cause compression of the lung, so as to study the effects of that condition, because the air is so quickly absorbed. Obviously these remarks apply also to those exceptional instances of pneumothorax occurring in medical practice in which a lung, previously healthy, is ruptured during a straining effort, or in a paroxysm of whooping-cough.

Dr. Walshe states that of eighty-seven cases of tuberculous perforation of the lung collected by him from various sources, fifty-five affected the left and only thirty-two the right pleura. But among twenty-six cases of pneumothorax which I extracted from the post-mortem records of Guy's Hospital without selection, the number on each side of the chest was exactly equal.

Physical Signs.—Clinically, it depends upon a variety of circumstances whether pneumothorax is easy or difficult of diagnosis. The recognition of this affection must always be based directly upon the results of a physical examination of the chest, although we shall hereafter see that the patient's symptoms, and the way in which they develop themselves, often enable one to form a shrewd guess as to the real nature of the case. As regards physical signs it should, I think, be a fundamental rule that pneumothorax is to be suspected whenever, over a large part of the chest, but on one side only, a marked deficiency or absence of vesicular murmur is associated with an alteration of percussion sound in the direction of hyper-resonance or of

tympanitic quality. The limitation of the signs to one side of the chest suffices to exclude the possibility of their being due to pulmonary emphysema, which from the time of Laennec has been given in text books as the disease chiefly needing distinction from pneumothorax, although in practice the two are not in the least likely to be confounded. Enfeeblement or absence of vesicular murmur is a very important indication of the presence of air in the pleural space; and sometimes the sound which accompanies the breathing gives one a distinct impression of being conveyed from a distant part of the chest. But in many cases there is marked cavernous or amphoric breathing. Sometimes, no doubt, this is due directly to the passage of air backward and forward into the serous cavity; but it is often present when the aperture is closed and when, as Dr. Gee remarks, it must in some way acquire its peculiar quality by "transmission through the pneumothorax." The voice may be either less audible than on the healthy side, or it may be conveyed so as to produce bronchophony or even pectoriloquy. As a rule, vocal fremitus is either absent or greatly diminished.

I have spoken above of the percussion sound as being "altered in the direction of hyper-resonance or of tympanitic quality." When air escapes into a healthy pleural sac, the sound is, as a rule, purely tympanitic. But, if the air should accumulate so as to cause extreme distention, it may, in the words of Dr. Walshe, at length become "muffled, toneless, almost dull," like that of a drum tightened to the highest possible point, and with all escape of air from its cavity prevented. Much more frequently, the reason why the percussion sound in pneumothorax is imperfectly tympanitic is that the pleura itself is thickened; in all probability not only do the chest walls themselves fail to vibrate, but they are even incapable of transmitting the blow made in percussion to the air within, so as to throw it into anything like free vibration. In such cases one may obtain any one of the modifications of percussion sound mentioned at p. 866, osteal, tracheal, tubular, or subtympanitic.

Hitherto I have refrained from mentioning certain physical signs of pneumothorax, which when they are present point very strongly to this affection. They are therefore apt to impress inexperienced auscultators with an undue sense of their importance, but must after all be regarded as accidental rather than as essential indications of it. They may be grouped together as "metallic" phenomena. By Laennec most of them were included under the name of "metallic tinkling," a sound which he compared with that "produced in a metal cup, or in one made of glass or of porcelain, by gently striking it with a pin, or by dropping into it a grain of sand." He described it as being heard when the patient either breathed, or spoke, or coughed. There was afterward much discussion as to the origin of this sound, but writers seem now to be generally agreed that the main cause of it is the bursting of bubbles of fluid in a large space, which is filled with air and has a smooth surface. In other words, metallic tinkling is a moist sound, or râle, modified by the vibrations of the walls of a cavity of great size, and by those of the air contained in the cavity. One way in which the bubbling necessary to give rise to such a sound may be produced was noticed by Laennec himself; namely, by the dropping of liquid from the upper into the lower part of the pleural space when it contains air as well as pus. Thus metallic tinkling may be due to the patient's changing from the recumbent to the sitting posture; but one is hardly likely to hear this unless one has the stethoscope applied to his chest before he begins to move. Again there is no difficulty in understanding how the sign may be produced by coughing, as well as by drawing in the breath, especially if there be a free communication between the space in which it is found and a bronchus. But I must confess that it does not seem to me

clear that it can arise as a mere result of speaking, apart from the agitation of the contents of the cavity, which may, perhaps, result from the act of expiration. I should rather think that what is heard under such circumstances is an *echo* of the voice, which acquires a metallic quality from the conditions under which it is produced. So, also, the heart sounds, and even the sound produced by percussion of the chest, may be reverberated with a similar character. To all such phenomena it would, I think, be better to give the name of "metallic echo;" reserving that of "metallic tinkling" for sounds which in their origin resemble râles. Coughing, as is obvious, may either be attended with echo, or with tinkling. A particular kind of metallic echo has been specially described by Trousseau under the name of *bruit d'airain*. Among all "metallic" phenomena it has the advantage that it is, much more than any other of them, under the control of the observer. Metallic tinkling is well known to be exceedingly capricious, accompanying certain respiratory movements, and being absent with others, according as bubbles happen or do not happen to burst. Even a metallic echo of the patient's voice may probably fail to be heard unless he speaks distinctly and with a particular degree of loudness. But in regard to the *bruit d'airain* one can not only determine the time at which the echo shall appear, but modify the sound which is to produce it, until one obtains the best possible result. The method of eliciting it was originally given in the "*Gazette des Hôpitaux*" for 1859. It consists in applying one ear to the back of the patient's chest, while a third person strikes the front of the chest, either with a plessor upon a plessimeter, or else with one coin upon another. The metallic echo which results is sometimes extraordinarily well marked, and I suppose that there is hardly any case of pneumothorax in which it is altogether absent. Traube, however, has pointed out that one may sometimes fail to obtain a metallic echo by percussion during life, and yet have no difficulty in eliciting it from the dead body of the same patient. He attributes this fact to lowering of the tension of the air in the pleural space, as the result of post-mortem cooling of the tissues.

Another sign of pneumothorax which is of considerable practical and of extreme historical interest is that which is termed "succussion splash." It was well known to Hippocrates, so that it is sometimes spoken of as "Hippocratic succussion." To obtain it, one may shake the patient's body while one has one's head pressed against his chest. But sometimes it can be heard at a little distance off, and the patient himself may be conscious of it every time he makes any abrupt movement, as in stepping down stairs, or in riding on horseback. It is literally nothing else than the splashing of pleuritic effusion against the sides of the serous cavity, and, of course, it is never audible unless there is liquid present as well as air. In such cases the signs of pleuritic effusion are, of course, to be observed, as well as those of pneumothorax; it may also be noted that alterations of the level of dullness when the patient changes his posture are generally very conspicuous, whereas in uncomplicated pleurisy (as I have already remarked at p. 932) they can seldom be made out satisfactorily.

Lastly, in most instances pneumothorax is attended with lateral displacement of the heart. Dr. Douglas Powell has pointed out, in vol. lix of the "*Med.-Chir. Trans.*," that the mere elasticity of the opposite lung drags the mediastinum over whenever air has free entrance into one pleural space without there being of necessity any excess of pressure above that of the atmosphere. He remarks, however, that in some cases of phthisis consolidation of the lung on the one side opposite to the pneumothorax prevents the mediastinum from being thus displaced, and I think that a like effect must also be produced by consolidation and adhesion of any considerable part of the lung on the side of the pleural affection, or, again, by the rigidity

and thickening of the serous membrane, which so often occur in cases of empyema before perforation takes place. Thus one must not expect to find the heart beating in an abnormal position in those instances of chronic chest disease in which it is sometimes so difficult a question to determine whether pneumothorax has or has not developed itself at an advanced stage. Yet, even in such cases, it is quite possible for the pressure of the air in the pleural cavity to become considerably increased. The way in which this is brought about is now well understood to be by the action of a piece of lymph lying over the aperture; this plays the part of a valve and allows air to enter the cavity during inspiration, but hinders its escape during expiration. Cohnheim, indeed, declares that for air confined in the pleural space to retain for any length of time a high pressure after closure of the opening by which it entered is impossible, on account of the rapidity with which it undergoes absorption. But however this may be, it is certain that among seventeen cases collected by Dr. Powell there were twelve in which after death the pressure was found to be above the atmospheric pressure, the difference amounting in these cases to that of a column of from five and a half to seven inches of water. When the adjacent organs are capable of yielding to it, one cannot be surprised that the elastic force exerted by air in the pleural cavity should displace them even more than they are displaced by liquid effusion. Thus Dr. Gee speaks of the diaphragm as being pushed down so that the upper surface of the liver lies altogether below the level of the anterior costal margin, percussion yielding a tympanitic sound within the ordinary confines of the abdomen above the liver dullness. The intercostal spaces, too, may be flattened, or even bulging, and the affected side of the chest may be greatly enlarged, and almost or quite motionless.

Diagnosis.—If, now, it be asked what affections there are of which the physical signs may be mistaken for pneumothorax, or *vice versâ*, there is but little to answer. I have already remarked that when distention of the pleura with air is very extreme, the percussion sound may become muffled and toneless; but I do not know whether this ever reaches such a point that the case might be supposed to be one of liquid effusion. In vol. xi of the "*St. Bartholomew's Hospital Reports*," Mr. Butlin has recorded an example of rupture of the diaphragm, with escape of the distended stomach and colon into the left pleural cavity: it was the result of a severe crush between the buffers of two railway coal wagons, and was diagnosed during life as a traumatic pneumothorax. But in general the only cases which are attended with doubt are those in which, if air is present in the pleura at all, it is confined to a limited portion of the serous space. Thus at the upper part of the chest it might very likely be impossible to diagnose a localized pneumothorax from an exceedingly large vomica; but I should doubt whether a localized pneumothorax ever occurs in that position, and in all probability the cases that have been admitted as open to question have always been really examples of vomicæ attended with unusual signs, such as metallic tinkling or Hippocratic succussion splash. On the other hand, at the base of the chest, a cavity within the lung of sufficient size to be mistaken for a pneumothorax is a thing almost, if not quite, unknown. But it must not be forgotten that during the contraction of the sac of an empyema the diaphragm, with the stomach below, may on the left side be drawn upward so far that percussion may yield a tympanitic sound over a considerable area, when complete dullness might otherwise have been expected. A similar state of affairs may also arise when the lung is affected with cirrhosis. Probably one might avoid any error of diagnosis by re-examining the patient after having made him swallow a large quantity of fluid. In a case of Wintrich's a subdiaphragmatic abscess, which arose from a perforating ulcer of the stomach and consequently contained air, was mistaken for a pneumothorax. It is

also necessary to bear in mind the fact, mentioned at p. 911, that in some instances the percussion note is tympanitic over a part of the lung affected with pneumonic hepatization. For whenever this is observed the suspicion cannot but arise that there is air in the pleural cavity.

But, after all, the mistake which is most apt to be made in regard to pneumothorax is not that it is taken for any other affection or any other affection for it, but that its presence is overlooked. This is due to the fact that the *symptoms* of pneumothorax, which are sometimes of the most striking character, are in other cases altogether absent. The amount of dyspnœa produced by the escape of air into a pleural sac depends upon two conditions; first, upon whether the patient's vital functions are or are not being actively carried on at the time; and, secondly, upon whether he has or has not been accustomed to make full use of the lung on that side in breathing. A healthy person always experiences great distress when attacked with pneumothorax. Among those who are the subjects of disease the distress is greater in proportion as they are well nourished and able to take food and to bear exertion. It is also greater among those who have chronic pulmonary disease in proportion as the lung on the side of the pneumothorax took a greater share of the work of respiration before the supervention of the accident. It accordingly reaches its maximum when a man who has one lung extensively diseased, but whose health is nevertheless fairly good, becomes attacked with pneumothorax on the opposite side. A directly fatal result is inevitable. And thus pneumothorax has to be remembered among the possible causes of sudden death in persons who are walking about and earning their living. One morning, in the year 1874, there was brought into Guy's Hospital the body of a man who had fallen dead while on his way to his work; he was found to have pneumothorax on the right side and chronic tubercular disease of the lung on the left side. On the other hand, if air escapes into the pleura of a person who is wasted and whose functions were already at a low ebb, and especially if the lung on that side had before been rendered almost useless by advanced tubercular mischief, the supervention of the pneumothorax may give rise to no symptoms whatever. I believe that this fact was first pointed out in the "*Medical Gazette*" for 1844 by Dr. Hughes, who was one of the best auscultators of that day. But even in persons who are already in the last stage of phthisis it is possible for pneumothorax to produce a shock that may be directly fatal. The patient is, perhaps, found dead in bed without anything having occurred to attract the attention of the nurse; in one such case, however, it was noticed that the body was bent, as if the end had been painful.

Between the two extremes described in the last paragraph there are all degrees of severity in the symptoms of pneumothorax. The most typical cases are those in which the patient is suddenly seized with an agonizing pain in the side, and has a sensation of something having given way or even of a stream of air or of fluid trickling down within his chest. His dyspnœa is extreme; the respirations may reach forty or even sixty in the minute, while the beats of the heart, although accelerated, are not so to any proportionate extent. The pulse is small, the radial arteries being imperfectly filled as a consequence of the deficient flow through affected lungs. The hand, the foot, the cheeks, the lips, and the visible mucous membranes become cyanosed, the extremities and even the tongue feel cold; a cold sweat breaks out over the body; the temperature, even in the rectum, falls considerably. The voice is weak and may even be altogether toneless. There may be complete inability to cough. The patient is usually obliged to sit up in bed, sometimes he finds it more comfortable to incline toward the affected side, sometimes toward the healthy side.

Treatment.—In such cases a great deal can be done to diminish the

patient's sufferings and even, perhaps, to avert a fatal termination. Cupping, dry or wet, often gives remarkable relief; and if the patient was previously in a fair state of health, there is no reason why venesection should not be employed. A small dose of morphia should be injected subcutaneously, or, as Dr. Walshe recommends, a very little chloroform may be given by inhalation from time to time. He also says that he has seen musk in five-grain doses afford much relief.

If great enlargement of the side and depression of the diaphragm suggest that the pressure of air within the thorax is greater than the atmospheric pressure, paracentesis should be performed with a fine trocar. But, as I have already remarked, displacement of the heart alone is not evidence of increased pressure; it may be simply due to the elasticity of the mediastinum. Fräntzel appears to have tapped the pleural cavity rather frequently; he speaks of a dissertation by Bärensprung, in which are recorded a number of his cases treated in this way with success. He says that, if possible, it is well to postpone the operation until three or four days have elapsed, by which time the aperture in the pleura that allowed the escape of the air may generally be expected to be closed. Between the fourth and the eighth day it may be advisable to introduce a trocar, even when the symptoms are not very urgent. He has often found the pressure of the air such that a considerable quantity passed out through the instrument; and even when this is not the case it is easy to close the wound, and no harm is done. An aspirator ought never to be employed, on account of the risk of reopening the original aperture. Fräntzel uses an ordinary trocar with a valve of gold-beater's skin. If cough arises during the operation, which is not uncommon, he either gives an injection of morphia and waits for a time before withdrawing the trocar, or else he keeps up pressure upon the seat of puncture for a little while afterward until the cough has ceased. By either of these methods the escape of air along the track of the trocar may be prevented, which would otherwise have led to a general subcutaneous emphysema, causing great inconvenience to the patient.

Prognosis.—In some cases of pneumothorax the symptoms continue with unabated severity until the death of the patient, which may take place after a few hours, or in a day or two. But in other cases they subside as the shock of the accident passes off; the breathing may remain rapid, and yet the patient may experience little or no distress, as in a case in which Dr. Walshe counted the respirations at fifty-two in the minute. In some very exceptional instances the air gradually undergoes absorption, and complete recovery takes place. Vogel's patient, to whom I have referred at p. 952, got quite well within four weeks, being from that time able to work as well as ever.

But, as we have already seen, what usually happens is that after a few days pleurisy sets in. Even then it is not impossible for the disease rapidly to subside. Dr. Walshe says that he has seen two cases in which, in the course of two months, all signs of air and fluid in the pleura disappeared; in all probability the exudation was sero-fibrinous. As a rule, when an empyema is developed, one can give the patient a chance of recovery only by making a free external opening, so as to allow the cavity to become obliterated by the process of granulation. The same treatment is, of course, necessary when the entrance of air into the pleura is secondary to pleurisy. But in cases in which there is already advanced phthisis it is scarcely ever right to perform such an operation, as the resulting inflammation of the pleural space is apt to assume a putrid character, so as to carry off the patient very rapidly. On the other hand, Czernicki has pointed out ("*Gaz. Hebd.*," 1872) that in some phthisical patients the supervention of pneumo-

less numerous cavities), that in the middle of the organ there are cheesy patches, and that in the lower lobe there are gray tubercles, scattered or in groups. Or, again, the affection in one lung may appear to be typically fibroid, or typically pneumonic, throughout; yet in the opposite lung, in which the disease is of more recent origin, there may be clusters of tubercles; and these may themselves be caseating, whatever the character of the change in the organ first affected. Lastly, the pulmonary lesion may appear to be pneumonic, or to be fibroid, not a single tubercle being discoverable, even in a state of caseation; yet in some distant part of the body there may be tubercular lesions of the most characteristic description. What is most frequent is, no doubt, that the small intestine should present the transverse, hard-edged ulcers, with tubercles in the peritoneum beneath and around them, of which the nature is so unmistakable. But I could cite many other facts, all of a similar kind. Thus, in 1876, I examined the body of a girl, aged sixteen, who died of what was regarded as pneumonic phthisis; in one kidney, traversing its cortex from the surface to the medulla, was a single linear tuberculous mass. In 1878, in a case of pneumonic phthisis, in which the affected part of the lung showed only a cheesy infiltration breaking down into sinuous cavities without any distinct walls, there were not only small, caseating points and ulcers in the intestine, but in the liver several tubercles as typical in their characters as it would be possible to conceive. So, again, in 1876, I made an autopsy upon a child, aged six, whose lungs presented a remarkable example of fibroid phthisis; in her intestine there were a large number of ulcers, with most abundant subserous tubercle. In 1879 I examined a typical case of fibroid phthisis of the apices of the lungs, with indurated tubercles lower down; in the kidneys and the prostate of the same patient there were caseating vomicæ. In 1878, in a woman of thirty-three, I met with an instance of phthisis, consisting mainly in gray induration of the pulmonary tissue, there being very little tendency to caseate; both adrenal bodies contained cheesy nodules, and there were yellow tubercles in the liver.

A few years ago it might have been objected that some of these cases were useless as evidence, inasmuch as tubercles, whether in the lungs or in other organs, when they appeared to be of more recent origin than a pneumonic or a fibroid phthisis, could be supposed to have arisen secondarily from it by a process of infection. In particular, some pathologists thought that tubercular ulcers in the small intestine were frequently caused by swallowing matters expectorated from the lungs, when the disease in these organs was itself of a non-tubercular kind. But as soon as it was proved that the infection of tubercle depends upon the presence of a specific virus, all such objections fell to the ground. It must, however, be observed that many of the instances cited in the previous paragraph fail to adjust themselves readily to any infective theory. When, in a case of phthisis, an isolated tubercle, or a cheesy mass, or a vomica, is found in some distant organs, such as the kidneys, or the liver, or even the testis, it is more natural to suppose that it arose independently and by a repetition of the same process which caused the pulmonary lesion; and this conclusion is not invalidated by the presence of a number of tubercles in close proximity to one another in a single organ, even though we suppose their multiplication to have been due to a kind of local infection within the limits of its area.

Again, all that I have seen of the way in which phthisis begins confirms my belief that it is at the outset a tubercular affection. I have several times found in the apices of the lung lesions which were quite recent, and evidently in their very earliest stage, when death had been due to some other cause. These cases seem to me of sufficient importance to justify me in briefly enumerating them. In 1881, a man, aged thirty, died in Guy's

Hospital, of caries of the spine with psoas abscess; in the apex of each lung there were gray translucent tubercles, some scattered, some in clusters, occupying about a square inch of the cut surface; they were more numerous on the right side than on the left; the other parts of the lungs were quite free. In the same year a man, aged thirty, died of delirium tremens; in the upper parts of both lungs there were miliary tubercles in groups. In 1879, a youth of nineteen died of spinal disease, with a scrofulous kidney; in the apex of the right lung there was a single cluster of the most typical, firm, gray tubercles, none of which showed any tendency to caseate. In the same year a man, aged thirty, died of "sacro-iliac disease," which, although the result of injury, was accompanied with tubercular affections of the prostate, kidney, spleen, and lymph glands; the extreme apex of the right lung contained a number of scattered gray miliary tubercles, without the slightest caseation, and with no induration of the surrounding pulmonary tissue. In 1877, a boy, aged ten, was killed by fracture of the spine; he appeared to have been strong and healthy, but in the apex of each lung there were miliary tubercles. Dr. Moxon's experience, when he taught pathology at Guy's Hospital, was very similar. In 1869, a child, aged two and a quarter, died of croup; in the left lung, below the apex, there were found several clusters of gray tubercles, one of them with some cheesy material in its centre. In 1867, a man, aged twenty-seven, was killed by accident, with fracture of the skull; at both apices, especially the right, there were recent miliary tubercles, in smaller or larger clusters. In the same year a man, aged twenty-two, died of typhus; in the right upper lobe there were many clusters of miliary tubercles, some already softening. In 1868, a woman, aged twenty-one, died after amputation of the thigh for disease of the knee joint; in each apex there was early phthisis with clustered tubercles, some caseating; there was also a small vomica. Even when the appearance of an incipient pulmonary lesion is such that its tuberculous character might fairly be doubted, one may discover elsewhere morbid changes the nature of which is indisputable. Thus, in 1874, in examining the body of a girl, aged eighteen, who had died after excision of the knee joint, I found in the apex of the right lung a mass, the size of a marble, consisting of a cluster of yellow softening granules, which might naturally have been set down to a catarrhal pneumonia; the bronchial glands were caseating, but one of them contained the most typical gray tubercles. At the time this appeared to me to be a most instructive case.

Seat of the Tubercles.—If we consider that there is scarcely a structure in the human body which is not liable to the growth of tubercles, we shall surely think it very improbable that in the lung their development should be limited to any one, rather than another, of the various tissues which make up the organ. Rindfleisch, however, has recently maintained that the morbid process in phthisis begins definitely just where the bronchioles open into the alveoli, the earliest change being a "tuberculous infiltration of all the edges and processes" which exist at these points, and which contain muscular and elastic tissues, as well as fibrous. The occurrence of such a change at the extremities of several adjacent tubes, and its extension along the walls of the tubes themselves, would no doubt account satisfactorily for the "racemose" distribution of pulmonary tubercles on which Carswell used to insist, so that the cant phrase "Carswell's grapes" was invented to keep it in recollection. But it has always appeared to me that Carswell's drawings illustrating this point are highly artificial and untrue to nature, and that it requires an eye of faith to perceive, except, perhaps, in some rare cases, anything like a "peribronchial" distribution of clustered tubercles. And Dr. Hamilton, in the "*Practitioner*" for 1880 describes tubercle in the lung as generally beginning in a little cellular projection on one side of an alveolus, which

monly regarded as tubercular disease in the lungs was in reality pneumonic, and that softening of the organ with excavation of its substance might occur without any tubercle being present. But it is to be observed that he was very far from maintaining that an absolute distinction could be drawn; in his well-known essay, read before the Guy's Hospital Physical Society in 1845, he described first a "pneumonic" and then a "tuberculo-pneumonic phthisis;" and the final sentence of this work is, that "in every form of phthisis, inflammation constitutes the great instrument of destruction." Thus, after all, it may be said that Addison's teaching bore upon the theoretical question whether tubercles should be regarded as distinct from what Laennec used to term tubercular infiltrations, quite as much as upon the practical question whether a separate kind of phthisis is to be recognized apart from the tubercular. Since his time many pathologists have asserted, in the most positive and dogmatic manner, that a "catarrhal" or (as it is often termed) a "caseous" pneumonia is the essential morbid change in many, if not in most, cases of phthisis. And another form which has also been declared to be non-tuberculous is the so-called "fibroid phthisis." But there is no real consensus of opinion among the most advanced histologists with regard to these questions. Rindfleisch, who at one time maintained that the "tubercular granulations of Laennec" consisted in an inflammatory infiltration of the alveolar parenchyma round the smallest bronchi, now teaches (in Ziemssen's "Handbuch") that they are true tubercles.

For my own part, I believe that all ordinary cases of phthisis are essentially of the same nature. The varied appearances which may be found in the lungs after death seem to me to depend mainly upon whether the tubercles and the tuberculous infiltration become caseous or undergo fibrous changes. This, to a great extent, rests upon the degree of rapidity with which the disease has advanced during life. Thus, pneumonic phthisis is, I think, generally equivalent to a phthisis which has progressed quickly; fibroid phthisis to one which has been slow in its course.

The ultimate decision of this question must rest, if Koch's discovery of the tubercle bacillus should be confirmed, upon whether this organism is or is not present in cases of phthisis supposed to belong to different forms. In his earliest communication on the subject, Koch stated that he had found bacilli in twelve cases of "cheesy bronchitis and pneumonia," which appear to have been all the examples of that variety of phthisis which he had examined. In fibroid phthisis one would doubtless have to look for them in the parts of the lungs most recently affected. For in the caseous form of the disease they were generally limited to the edge of the infiltrated tissue, where, however, they were very abundant. Sometimes nests of bacilli were met with even in the interior of parts of the lungs which had undergone infiltration. In most vomicæ they were present in great numbers. The little cheesy fragments which are so commonly found in vomicæ consist, according to Koch, almost entirely of masses of bacilli. It is also worthy of notice that, after speaking of the presence of the bacillus in the "Perlsucht" of cattle (v. p. 91) Koch goes on to say that he detected it also in cases in which there were round, smooth-walled nodules, filled with a cheesy pulp, such as are not generally reckoned to belong to perlsucht, but are regarded as due to bronchiectasis. It would thus appear that pathologists have had a tendency to confine within too narrow limits the conception of tubercular diseases of the lungs in animals as well as in man.

In discussing the subject of tubercle in general (*supra*, p. 81) I have endeavored to show that histology opposes no insuperable difficulties to the doctrine of the unity of phthisis; and, indeed, that there are no difficulties at all which are not equally in the way of the recognition of miliary tuberculosis as a definite affection. But my own opinions with regard to phthisis have

been based not so much upon microscopical investigations as upon the results of careful study of the appearances seen in the post-mortem room of Guy's Hospital during a long period of years. And I cannot help thinking that any unprejudiced observer who should use his eyes would inevitably be driven to the same conclusion. Without wishing to detract from the importance of histological inquiries, I am under the impression that the practice of setting aside minute fragments of diseased organs for study at a future time, when the general morbid anatomy of the case has been forgotten, is very apt to lead to one-sided and partial views. What I have found is, that in the same body lesions which would be universally admitted as tubercular are associated inextricably with other lesions, of which the tubercular nature would by many pathologists be denied.

Locality.—Before citing these facts in detail, I must insist on a point which has long been known both to physicians and to pathologists, namely, that the upper parts of the lungs are almost invariably affected with phthisis, in whatever form, before the lower parts; and that in all but the most exceptional instances, the disease spreads downward from apex to base, often with almost perfect regularity.

It is difficult to find any satisfactory explanation of this proclivity of the upper lobes of the lungs to phthisis. As I have already stated at p. 94, the same thing is observed in miliary tuberculosis, in which disease the pulmonary affection is believed to be due to an infection of the tissues at a number of different points through the blood stream. Hence I cannot accept Dr. Hamilton's view, expressed in the "*Practitioner*" for 1880, that the proclivity of the apices depends upon their being the driest parts of the lungs, so that caseation of catarrhal products is more apt to take place there than elsewhere. He also maintains that there is less expansion of the apices during breathing, and that catarrhal products are consequently more likely to accumulate in them than in other parts of the lungs. Almost exactly the same line of reasoning is adopted by Rindfleisch in Ziemssen's "Handbuch." He insists that the upright position of the body in man and in the Quadrumana causes the weight of the shoulders and arms to fall upon the upper ribs, and so interferes with their play and leads to a deficiency in the movement of air in the apices as compared with that in the lower lobes. On the other hand, it is certain that the proclivity of the apices is no greater in men than in women, who notoriously use those parts of the lungs far more than men do. And I have heard Dr. Moxon assert that in persons who are confined to bed the regions which become the earliest seats of tubercle are the anterior edges, a fact for which he found an explanation in the supposition that under such circumstances these parts are more active than any other in the function of respiration. I must not omit to add that the general rule of the proclivity of the apex is liable to some other exceptions. In certain cases the tubercles appear a little lower down, leaving one or two cubic inches at the extreme summit of the upper lobe free from them. But sometimes the middle of the organ is first affected, or even the lower lobe, the upper angle of which is, indeed, very frequently the seat of a vomica in ordinary instances of phthisis. But I believe that it never happens that the tubercular process spreads upward from the base of a lung into and through the upper lobe. And it is certain that what sometimes has been called "basal phthisis" is a distinct affection, which has been described above under the name of "chronic pneumonia." (*Supra*, p. 920.)

According to what has been stated in the preceding paragraph, we shall scarcely ever be wrong, when we find a lung extensively affected with phthisis, in assuming that the disease is oldest at or near the apex, and that its most recent stage is situated toward the base. Now, a very common appearance is that the upper lobe presents a dense fibroid mass (perhaps containing more or

less numerous cavities), that in the middle of the organ there are cheesy patches, and that in the lower lobe there are gray tubercles, scattered or in groups. Or, again, the affection in one lung may appear to be typically fibroid, or typically pneumonic, throughout; yet in the opposite lung, in which the disease is of more recent origin, there may be clusters of tubercles; and these may themselves be caseating, whatever the character of the change in the organ first affected. Lastly, the pulmonary lesion may appear to be pneumonic, or to be fibroid, not a single tubercle being discoverable, even in a state of caseation; yet in some distant part of the body there may be tubercular lesions of the most characteristic description. What is most frequent is, no doubt, that the small intestine should present the transverse, hard-edged ulcers, with tubercles in the peritoneum beneath and around them, of which the nature is so unmistakable. But I could cite many other facts, all of a similar kind. Thus, in 1876, I examined the body of a girl, aged sixteen, who died of what was regarded as pneumonic phthisis; in one kidney, traversing its cortex from the surface to the medulla, was a single linear tuberculous mass. In 1878, in a case of pneumonic phthisis, in which the affected part of the lung showed only a cheesy infiltration breaking down into sinuous cavities without any distinct walls, there were not only small, caseating points and ulcers in the intestine, but in the liver several tubercles as typical in their characters as it would be possible to conceive. So, again, in 1876, I made an autopsy upon a child, aged six, whose lungs presented a remarkable example of fibroid phthisis; in her intestine there were a large number of ulcers, with most abundant subserous tubercle. In 1879 I examined a typical case of fibroid phthisis of the apices of the lungs, with indurated tubercles lower down; in the kidneys and the prostate of the same patient there were caseating vomicæ. In 1878, in a woman of thirty-three, I met with an instance of phthisis, consisting mainly in gray induration of the pulmonary tissue, there being very little tendency to caseate; both adrenal bodies contained cheesy nodules, and there were yellow tubercles in the liver.

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Seat of the Tubercles.—If we consider that there is scarcely a structure in the human body which is not liable to the growth of tubercles, we shall surely think it very improbable that in the lung their development should be limited to any one, rather than another, of the various tissues which make up the organ. Rindfleisch, however, has recently maintained that the morbid process in phthisis begins definitely just where the bronchioles open into the alveoli, the earliest change being a "tuberculous infiltration of all the edges and processes" which exist at these points, and which contain muscular and elastic tissues, as well as fibrous. The occurrence of such a change at the extremities of several adjacent tubes, and its extension along the walls of the tubes themselves, would no doubt account satisfactorily for the "racemose" distribution of pulmonary tubercles on which Carswell used to insist, so that the cant phrase "Carswell's grapes" was invented to keep it in recollection. But it has always appeared to me that Carswell's drawings illustrating this point are highly artificial and untrue to nature, and that it requires an eye of faith to perceive, except, perhaps, in some rare cases, anything like a "peribronchial" distribution of clustered tubercles. And Dr. Hamilton, in the "*Practitioner*" for 1880 describes tubercle in the lung as generally beginning in a little cellular projection on one side of an alveolus, which

afterward becomes somewhat pedunculated and hangs into the alveolar cavity. When these alveoli lie adjacent to one another it may project into all of them at once. At first it pushes before it the epithelium and even the alveolar capillaries. But soon it breaks through and destroys the alveolar wall, so that a uniform rounded mass results, in which the outlines of the original air vesicles are barely recognizable. The cells of the tubercle may, Dr. Hamilton thinks, be derived either from the connective tissue elements of the alveolar wall, or from the endothelium of certain of its capillaries, or from both sources at once. Sometimes a tubercle sprouts from the inner coat of a branch of the pulmonary artery, starting, perhaps, from the endothelium, but soon involving the rest of the *tunica intima*, and almost occluding the channel of the vessel. Other tubercles lie in the course of the pulmonary lymphatic vessels contained in the periarterial and peribronchial sheaths, the interlobular septa and the deep layer of the pleura.

In some instances tubercles, scattered or in clusters, spread slowly through one lung, or even through both lungs, with little or no change in the intervening portions of the pulmonary tissue. But, as a rule, this undergoes consolidation at an early period, so that the tubercles come to be embedded in a more or less homogeneous mass. Sometimes, the substance of the lung is involved uniformly from the apex downward, the edge of the consolidated area having a festooned outline, not unlike that of the border of a malignant new growth. Much more frequently, even when part of the upper lobe is universally affected, there are more or less numerous independent nodules of various shapes and sizes lower down; and between and below these, again, scattered tubercles may generally be seen in abundance. The character of the infiltrating material varies widely in different cases. It may be a soft, semi-translucent, pinkish substance, the less recently formed parts of which are found to have passed into a state of caseation. Or it may be firm, dark, and tough, constituting what Addison used to call "iron-gray induration."* Or it may have a fibrous texture crossed by bands and seams of well-developed fibrous tissue, and of all degrees of depth of pigmentation, up to perfect blackness. In general, the softer kinds of infiltration are associated with caseating forms of tubercle, the harder with tubercles which themselves become tough and fibrous. But there are comparatively few cases in which some cheesy masses are not to be found in one part of the lungs or another; and where the parts which were earliest affected are fibrous and of an iron-gray color, or black, it often happens that recently involved parts are soft and yellow.

Vomicæ.—In all but very exceptional cases of phthisis the process of consolidation is followed more or less quickly by one of ulceration, leading to the formation of cavities or (as they are termed) *vomicæ*. The tubercles themselves doubtless soften in their centres, as is the case in every other organ in which tubercles occur, and as, indeed, can often be seen in the more recently affected parts of the lungs. Sir Robert Carswell used to declare that sections of bronchial tubes with pus in their interior were very frequently mistaken for softening tubercles. In this I feel confident he was wrong. In two or three exceptional instances I have thought, on first glancing at the cut surface of a lung, that I saw tubercles, when there were really only the open mouths of swollen tubes; but an instant afterward I have perceived my error, because the slightest pressure below has made pus well up from them in large quantities. There seems to be no reason why a vomica of considerable size should not result from the breaking down of caseous material derived by extension from a single original tubercle. But, as a matter of fact, tubercles are generally scattered too thickly to admit of such an occurrence, and there can be no doubt that formation of cavities ordinarily

* [This corresponds to the "induration ardoise" of Cruveilhier.—Ed.]

involves the destruction of infiltrated lung substance as well. One very characteristic appearance, which is exactly analogous to what may be observed in various other organs besides the lungs, is the presence of a cheesy zone of definite thickness, which on one side bounds a vomica, while on the other side it is embedded in the pulmonary tissue. Such a zone is generally an indication that the affection is still actively spreading; it affords one of the chief means by which vomicæ increase in size. As they enlarge, cavities originally distinct are very apt to open into another and to coalesce. In this way a single chamber of very irregular form may be produced. Sometimes the destructive process remains limited by the lobar septum; sometimes this becomes ulcerated through, so that both the whole of the upper lobe and a large part of the lower lobe are included in one wide, open sac. Sooner or later, however, the further extension of every vomica becomes arrested; that is, if the patient should survive long enough to allow time for it. The indication of this change is that the interior of the cavity ceases to be rough and shaggy with adherent portions of cheesy *débris*. A fibrous wall of greater or less thickness becomes developed round it, and its inner surface gradually assumes a smooth, polished appearance, exactly like that of a mucous membrane. Such smooth-walled vomicæ are often caused by fibrous bands or (as they are termed) *trabeculæ*, which consist each of a mass of condensed pulmonary substance with fibrous tissue that, perhaps, originally belonged to interlobular septa. In all probability trabeculæ, of which these are the only constituents, are remains of partitions that at one time separated from one another vomicæ which were originally distinct but have since coalesced. But other trabeculæ often contain obliterated branches of the pulmonary artery, and sometimes several of them can be seen to spread away from a single point situated on that side of the cavity which is nearest the root of the lung, so that their formation has obviously been the result of the resistance offered by the arterial walls to the process of ulceration. Ultimately the trabeculæ themselves often give way and rupture, and their loose ends may then be seen hanging into the interior of the vomicæ. In very large cavities a bundle of such ruptured trabeculæ may sometimes be seen, the relation of which to the pulmonary artery is at once shown if a probe is passed into that vessel from the heart. Sometimes a pervious channel persists for some little distance along the interior of a trabecula, a fact which we shall hereafter see to be of considerable clinical importance. I must not omit to mention that, according to many observers of authority, smooth-walled cavities, such as I have been describing, have often an entirely different origin from that which I have assigned to them, being dilatations of bronchial tubes, instead of being formed by ulceration. Tubes do, indeed, almost invariably open into them more or less freely, the branches of the bronchial tree possessing no such power of resisting ulceration as belongs to the arteries. The idea of regarding the cavities in question as "bronchiectatic" seems to have originated with Laennec. In all probability what first suggested it was the difficulty of understanding how a vomica formed by ulceration could acquire anything like a mucous lining. But this goes for very little, now that we know how readily such a structure can be pushed forward over a raw surface from an edge of skin or of mucous membrane, as, for example, in the case of a rectal fistula. As a matter of fact, however, I am not sure whether smooth-walled pulmonary cavities ever have a continuous epithelial lining. Dr. Ewart, in his Gulstonian Lectures for 1882, says that this is wanting, except where there are "scattered islets of mucous membrane," the remains of "outlying bronchi intersected by the cavity wall." But Dr. Hamilton, in the "*Practitioner*" for 1879, declares that cavities which I cannot accept as dilated bronchial tubes often have an epithelium which is

"most typically columnar and ciliated." This pathologist attempts to explain the occurrence of bronchiectasis, and the sinuous and irregular outlines of the cavities which he believes to be of such a nature, by referring it to the traction of bands of fibrous tissue radiating away from the sides of the cavity at different points. I cannot say that the explanation appears to me to be altogether satisfactory. But what has always convinced me, in the post-mortem room, that the cavities in question were really vomicæ is that one never gets an opportunity of observing the earlier stages of the process of dilatation. If the view adopted by Dr. Hamilton were correct, one ought, toward the margin of the affected part of the lung, to see tubes which could still be traced on to their extremities, but the sides of which were beginning to bulge out here and there. On the other hand, what one does commonly find are all possible transitional varieties between smooth-walled cavities and unmistakable vomicæ. The former are seen toward the apex, where the mischief is of oldest date; the latter lower down, where it is of more recent origin. Moreover, smooth-walled cavities often riddle the substance of a diseased lung in all directions, communicating freely with one another on every side, so that an ulcerative process must clearly have been concerned in their formation.

The contents of vomicæ vary widely in kind and in amount. When they are recent they often show masses of cheesy *débris*. Such masses, even if they are at first too large to pass out through a bronchial tube, probably crumble into fragments in the course of time and are expectorated. Cavities of old date usually have pus in their interior and sometimes they are quite full of pus. This, of course, implies that there should be no very free communication with the bronchial tubes. And the fact is that in old vomicæ there is a tendency for the orifices of the tubes to contract until they become very narrow. One often finds that a tube of considerable size, into which a large catheter tube might be passed, has an opening into a cavity that will but just admit a probe. When such is the case a turgid condition of its mucous membrane may easily block it altogether. It is remarkable how seldom the contents of phthisical cavities putrefy; they often have a faint, sickly odor, but they very rarely become actually fetid, nor do they often undergo that peculiar acid fermentation which is so apt to arise in cases of chronic bronchitis with dilatation of the tubes. When there is free escape of pus from vomicæ their lining membranes may continually pour out fresh quantities of it, so that a large amount of expectoration may occur from day to day. But sometimes the walls are found at an autopsy perfectly dry, and the interior is quite empty; in such cases there may during life be no expectoration whatever. In 1854, Dr. Bristowe showed to the Pathological Society a specimen of such a quiescent cavity having adherent to its inner surface a soft, greenish, powdery mass of fungus, consisting of a branching mycelium, and of a fructification, in which the spores were arranged upon rounded heads raised on thick stalks.

Adhesions.—A very constant attendant upon phthisis is a local pleurisy, which leads to the gradual closure of the upper part of the serous space on the affected side of the chest. As a rule this affection is non-tubercular, and Rindfleisch insists on the extreme vascularity of the fibrous tissue which unites the two surfaces, as contrasting with the deficiency of vessels in the substance of a fibroid lung. Not infrequently the adhesions are of enormous thickness and density, so that after death it is impossible to remove the lung without the very free use of the knife.

Involution.—To complete my description of the morbid anatomy of phthisis I have still to discuss the processes by which tuberculous lesions in the lungs become obsolescent, so that they may cease to threaten the patient's life, or even to impair his health. Relics of former mischief are, indeed, dis-

covered very frequently in the lungs of persons who have died at various periods of life and of every kind of disease or injury. In May, 1880, Dr. Heitler, of Vienna, brought before the Medical Society of that city an analysis of all the cases of this kind that had been met with in a series of 16,562 autopsies between the years 1867 and 1879. Excluding altogether all cases in which death was due to pulmonary tuberculosis (and among which there must, of course, have been many other instances of a previous attack of the same affection), he found that there were no fewer than 780 (or almost exactly 5 per cent.) in which obsolete tuberculous masses were present. Of the patients 503 were males, 277 females. The number of those who died of tuberculous affections of other organs was 101. A point of great interest is that the proportion of cases at different ages went on regularly increasing for each decennial period up to sixty years of age. Among persons aged from ten to twenty there were 12; from twenty to thirty, 105; from thirty to forty, 131; from forty to fifty, 156; from fifty to sixty, 157; from sixty to seventy, 36; from seventy to eighty, 153. It is true that no positive conclusion can be drawn from this fact, in the absence of information as to the proportion of persons at different ages in the total number of autopsies, but I think it is difficult to escape the inference that the time at which the pulmonary lesions were originally developed must, in a considerable number of instances, have been during adult life. In no fewer than 651 cases both lungs were affected, though generally to an unequal extent; in sixty-eight the right one was alone affected, in sixty-one the left lung. Some observers have shown extreme reluctance to admit that such appearances really indicate the remains of phthisical disease, or that such disease can be said to be ever curable. Strictly speaking, indeed, one should speak of phthisis as "cured" only when its favorable issue has been mainly due to medical treatment, whereas in all probability the subsidence of the morbid process in the cases in question was generally spontaneous. The only instance that I know of in which there is a definite history of a former pulmonary affection is one cited by Rindfleisch in Ziemssen's "Handbuch." It occurred in a man, over fifty years of age, who died in hospital, of enteric fever, fourteen years after having been treated in the same institution for an attack of serious lung mischief, attended with hæmoptysis and with infiltration of the right upper lobe, down to the level of the third rib. He completely recovered, resumed his former occupation, and remained well until he took the fever, a week before his death. The part of the lung that had been diseased was found to be indurated and shrunken, with surrounding emphysema and dilatation of bronchial tubes. About the fact that the relics of long-past pulmonary mischief belong to the same affection which, when it goes on and destroys life, is called phthisis, there ought not, as I think, to be the smallest doubt or hesitation, even in the minds of those who hold the narrowest views with regard to tubercle. Almost invariably the seat of such relics is in or near to the apex of the lung. The affected part is more or less indurated; it is often puckered on the surface and adherent to the chest wall. On section it presents fibrous bands, or tough masses of fibrous tissue, parts of which are generally deeply pigmented, and in which there are often embedded cheesy or calcareous nodules of greater or less size. The cheesy nodules may look very like gummata; they are enclosed in fibrous capsules; not infrequently they are gritty, from the deposition of lime salts in them, or this process may have gone on until they have become converted into hard, smooth bodies, apparently made up almost entirely of mineral constituents. Those pathologists who hold that a caseous pneumonia is often the commencement of phthisis ought, I think, logically to accept even such appearances as these as conclusive evidence of the former existence of a morbid condition which, if it had advanced further, would have termi-

nated in that disease. But in many instances one also observes gray or black indurated tubercles, which obviously have themselves long been obsolete, and which may very probably have been of the same date as the cheesy masses. When the fibrous bands have given rise to much puckering of the pulmonary tissue, they often look very like cicatrices, and at one time it was actually taught that they represented former vomicæ which had undergone obliteration. But, as almost all observers seem now to be agreed, there is no proof of this, and it is not likely that a pulmonary cavity is capable of thus completely disappearing. There is no doubt at all that its walls may shrink, so that in course of time it may become much reduced in size. Dr. C. T. Williams has pointed out that this process of contraction of a vomica is often attended by a shifting of its position. Unless its anterior surface is closely in contact with a firmly adherent pleura, the more fixed part of its wall is that which contains the openings of bronchial tubes; consequently it often shrinks away from the front of the lung toward the root. Dr. Ewart, in his "Gulstonian Lectures" for 1882, gives diagrams showing that the pulmonary pleura, if not too extensively fixed by adhesions, may be drawn inward over such a receding cavity until it forms a deep chink or fissure. The space created by the shrinking of a vomica may be filled up by an enlargement of the adjacent pulmonary tissue. Even when there are merely solid relics of former mischief at the apex, without any evidence of cavitation having occurred, the surrounding lung substance is sometimes found to be highly emphysematous, the bullæ having been probably formed during inspiration, after the manner suggested by Dr. Gairdner (see p. 895). More frequently, however, the lower part of the upper lobe of the lung, or (in the case of the right lung) the fore part of the middle lobe, is uniformly enlarged, so that one might, perhaps, almost regard it as hypertrophied. Or if there is a considerable amount of mischief, the upper lobe of the opposite lung may increase in size until it passes across the median line. Other organs at the same time undergo development. The liver or the stomach is dragged upward, according as the right or the left lung is the one which is diseased, and the heart may be pulled over either to the right, or beyond its natural position to the left. Lastly, the upper ribs are drawn inward, so that the chest wall, especially below the clavicle, appears flattened or even hollowed.

Symptoms.—The clinical recognition of phthisis, as of pulmonary diseases in general, is based partly upon symptoms, partly upon physical signs. But there is no other disease in which it depends so completely upon the concurrence of the two kinds of evidence. Symptoms alone, when no signs can be detected, may justify a strong suspicion that phthisis is present; but, unless it is confirmed by their subsequently appearing, this suspicion never reaches certainty. On the other hand, when one discovers physical signs of the disease in a person whose health appears perfect—as sometimes happens, for example, in a candidate for life insurance—the proper inference is, I think, that they depend upon a lesion which, although it was phthisical, yet is now obsolete, at least for the time. I doubt whether in phthisis physical signs ever develop themselves to any extent without symptoms being also present, although it is, of course, true that the patient himself may fail to notice or may wilfully ignore them. Evidently, therefore, a description of the symptoms of the disease should precede that of the signs.

Now, the symptoms of phthisis fall into two groups. One group includes those which belong to the body as a whole, or which concern remote organs; the other, those which point directly to the lungs.

(1) Of the symptoms which belong to the body as a whole, one, which is very important, is *progressive emaciation, or loss of flesh.* This often occurs

with extreme rapidity. Rühle mentions the case of a very bulky woman who had weighed 240 lbs., and who lost 40 lbs. in the four weeks before she came under his care for hæmoptysis, at which time no physical signs of mischief in the lungs could be detected. Ultimately, phthisical patients commonly become reduced by a quarter or even a third of their weight. The explanation of the wasting is often by no means obvious. There is sometimes a great loss of appetite, and especially a distaste for fat in every form; or the occurrence of vomiting may appear to account for it. But other patients emaciate who eat well, and appear to digest what they eat. Nor does the loss of flesh seem to be constantly proportionate to the degree of pyrexia or to the amount of the sweating. It probably affects all the tissues more or less; but a point worthy of notice is, that the heart becomes much less reduced in size in phthisis than in some other wasting diseases, as, for example, malignant tumors. The reason seems to be that the right ventricle has so much work thrown upon it by the destruction of blood vessels in the diseased parts of the lungs. Among the minor alterations in nutrition which accompany phthisis are those which affect the hairs. The straight, lanky whiskers and beard of consumptive patients often suggest to one the nature of their disease. The hair, too, may become prematurely gray. Dr. Walshe speaks of having often noticed in males that the hair on the chest has become quite white when the change was only just beginning in the hair of the head and in the whiskers. Along with the emaciation of phthisis goes a more or less marked failure of strength and energy. The patient becomes no longer able to walk far without fatigue. If he has duties to perform, they tire him in a way to which he is not accustomed. After his day's work is over, he is glad to get home as quickly as possible and to lie on the sofa until he goes to bed, and in the morning he gets up feeling weary and unfit for the labor which is before him. Anæmia is another early symptom of phthisis. The face becomes pale, the hands are white and bloodless. In women, scantiness or suppression of the catamenia may be one of the first indications that the health is failing. Œdema of the ankles often occurs as the disease advances, but it is seldom considerable, unless there be venous thrombosis.

When one is consulted by a person who has thus become thin and weak, the first thing to do is to ascertain whether there is any *pyrexia*. And it is important not to be contented with taking a morning temperature; during two or three days the thermometer should also be used at bedtime, or in the evening. One should notice whether the palms of the hands feel hot, and whether the cheeks are flushed, and the patient must be asked whether he has noticed any unusual tendency to perspire, especially in the latter part of the night. The pyrexia of phthisis is altogether atypical, and in different cases it varies widely in character and in degree. It is scarcely ever altogether absent. Dr. C. T. Williams, however, in vol. lviii of the "*Med.-Chir. Transactions*," says that in several of his cases in which active disease was going on in one or both lungs, no rise of temperature took place. And he gives details of an instance in which, although five observations were made every day for a week, the thermometer was never found above 99°.

In the most acute cases of all, which in Germany have the name of "phthisis florida," the pyrexia may be continuous throughout the twenty-four hours; the temperature may reach 104°, and may never fall below 102°, unless profuse sweating should occur, when it may be lowered for the time to about 100°. It is rather a remarkable circumstance that, even when there is such high fever, delirium and other cerebral symptoms are often altogether absent, but they may occur, and sometimes the patient passes into a "typhoid" condition, with stupor, sordes on the lips, and a dry, brown

tongue. Another point which may be noted is, that phthisical patients often retain a much better appetite than would be present in most other diseases attended with a like degree of pyrexia. Nor is there generally much complaint of thirst. Scarcely less acute is the course of other cases in which the daily range of the thermometer is very wide, the maximum, perhaps, reaching 103° or 104°, while the minimum may be 98.4°, or even lower still. Rühle says that the occurrence of a subnormal temperature, alternating with a high temperature at different periods of the day, is more unfavorable than when the fall is nearly to the normal point. Sometimes the patient experiences a slight rigor, or a sensation of chilliness, and then passes through hot and sweating stages, very like those of a paroxysm of ague, for which disease I have, indeed, known phthisis mistaken by a careless observer. In other less severe cases the range of the temperature is comparatively slight; the thermometer may indicate 100° or 101° toward evening, but during the rest of the day it is, perhaps, scarcely, if at all, above the normal point. In the same patient there may be all possible variations in the thermometric readings. Even when pyrexia is generally present, it sometimes happens that none can be detected during intervals of days or weeks. Rühle says that it has not yet been made out whether the sweating of phthisical patients is invariably preceded by a rise of temperature. This comes on especially during sleep, and some individuals cannot doze for half an hour during the day without their clothes becoming soaking wet. In all probability a paroxysm of cough, of which the patient himself may be unconscious, is often the starting point of such outbreaks of perspiration. Of the cause of the differences in degree of pyrexia in different cases of phthisis, no satisfactory account can as yet be given. Dr. Wilson Fox (*"Med.-Chir. Transactions,"* vol. lvi) thinks that it is generally largely proportioned to the extent of the intercurrent "inflammation." But he seems to admit that there are many exceptions, and to be not unwilling to accept a conclusion which Lebert drew from a very elaborate series of investigations, to the effect that the temperature course is more influenced by individual idiosyncrasy than by anything else. This, of course, is no explanation at all.

The *pulse* is nearly always accelerated in phthisis, and its rate is almost as valuable an indication of the activity of the disease as the temperature itself. Like the pyrexia, it is highest in the evening. It is apt to be much affected by slight exertion, and even by a change of position from sitting to standing. It is generally soft and feeble in quality. Sometimes its rapidity is out of all proportion to the degree of fever; in all probability this depends upon the patient's being anæmic.

Vomiting is sometimes a conspicuous and early symptom of phthisis, and I have known instances in which medical men have been led by it into the grave error of supposing that the patient's complaints were all due to disorder of the stomach. The suggestion was many years ago made by Mr. Hilton that it was due to interference with the trunk of the pneumogastric nerve by tuberculous bronchial glands, but I do not think that he brought forward any actual observations in support of this opinion, which, indeed, could hardly be directly substantiated. At a later period of the disease, cough often leads to ejection of the contents of the stomach.

Another symptom which not very rarely attracts a large amount of attention in phthisis is *diarrhœa*. It is generally due to the presence of tubercular ulcers of the small intestine, or rather, perhaps, to a catarrhal state of the mucous membrane in general accompanying such ulcers. Sometimes diarrhœa from this cause persists for many weeks before any physical signs of pulmonary disease can be detected. But I do not remember any instance in which a tubercular affection of the intestine has been found post-mortem without the lungs sharing in the mischief. In advanced cases another

cause of diarrhœa is, of course, the development of a lardaceous change in the mucous membrane.

It is a curious peculiarity of consumptive patients that they generally remain all along hopeful as to the result of their illness. As Rühle says, they order new shirts within the last few weeks of their lives. This writer also observes that certain qualities of character grow more marked as the disease advances. Those who are naturally gentle become more gentle; those who are rough become more rough.

The *aspect* of a phthisical individual often indicates the nature of his disease to the experienced physician at the first glance. Apart from the question (to which I shall advert presently) of there being a special configuration indicative of a diathesis, a bright eye, and a flushed cheek, associated with a wasted frame and lanky hair, at once suggest tubercular mischief. A minor point, on which French writers chiefly have insisted, is the presence of a pink line on the gums close to the teeth. I am very doubtful whether it is seen more often in persons who are consumptive than in those who are not so. Nor should I have supposed that much value can be assigned to the existence of patches of tinea versicolor on the chest and elsewhere, although some observers still mention this as a suspicious circumstance.

(2) Of the symptoms which point directly to the lungs, *cough* is naturally the first to be mentioned. Indeed, it is often the earliest indication that the patient is otherwise than well. At first it may be very slight, hardly more than a clearing of the throat; or it may occur only in the early morning, or when the patient happens to exert himself in the course of the day. It may even disappear for a time, to return later on. But ultimately it becomes more and more frequent, until it may cause very great distress. It is when cough has been the first symptom noticed that the disease, as is so often the case, is said to have arisen out of a "neglected cold."

There may be no *expectoration*, or a greater or less quantity may be ejected of a frothy fluid, either watery or slightly viscid in character. Or the sputa may consist of a glairy, grayish material, in which the microscope shows large, round, granular cells, being fatty epithelium derived from the pulmonary alveoli. Streaks and spots of blood are in most cases present from time to time. As the local process advances the expectoration becomes muco-purulent; and it may ultimately be almost pure pus, or pus so intimately mixed with blood that it has a uniform brick-dust red color. This, when abundant, may accumulate in the spit jar as an almost homogeneous mass. But in other cases the expectoration consists of pellets that remain distinct from one another, even after they have settled upon the interior of the vessel. It is then usual to speak of "nummular sputa," from their resemblance in size and shape to coins. If received into a layer of water they are seen to have a loose, flocculent surface, as if they were portions of wool, or as if they had been "nibbled at," to copy an expression employed by German writers. They generally contain no air, and, therefore, they fall rapidly to the bottom, unless they are held up by stringy mucus. Their characters seem to show that they have been formed in a space of some size, not in a narrow tube through which air was constantly passing backward and forward. Accordingly, the opinion, which is generally held, that nummular sputa are distinctive of phthisis is not without reason. But it must be remembered that the necessary conditions for their production are afforded by dilated bronchial tubes, as well as by pulmonary vomicæ. Probably this affords the explanation of a case mentioned by Sir Thomas Watson, in which he wrongly diagnosed phthisis when extensive chronic bronchitis was the disease from which the patient was suffering.

It has long been known that the sputum of phthisical patients often contains fragments of lung tissue, the nature of which can be identified

microscopically, as the shape of the alveoli is still plainly visible. Dr. Fenwick, in the "*Med.-Chir. Trans.*" for 1866, showed that their detection is much facilitated by boiling the sputum with an equal part of a solution of pure caustic soda (gr. xv to ℥j). This dissolves the mucus in three or four minutes. The resulting liquid is then poured into a conical glass which is filled up with pure water; and the deposit which forms is carefully examined in a very shallow cell. Dr. Fenwick in one case found 800 fragments in the expectoration of twelve hours. He did not discover them in any case which was at so early a stage that there were no physical signs, but he often succeeded when the signs were such as might have led to the opinion that ulceration or "softening" had not yet begun. The method is also of great value in cases in which phthisis supervenes upon chronic bronchitis and emphysema, and in which the signs are apt to be ambiguous. At an advanced stage, when cavities were obviously present, Dr. Fenwick never failed to find elastic fibres in the expectoration, even though the disease had appeared to be quiescent, both from the general improvement in the patient's condition, and from his coughing only in the morning and spitting up merely a little semi-transparent mucus.

More recently, the detection of the *bacillus* of tubercle in the sputum has become an important means of diagnosing phthisis. According to a paper by Dr. Heneage Gibbes, of King's College, in the "*Lancet*" for 1882, the best method of preparation is the following. A thin layer of the sputum is spread out upon a cover glass and is allowed to dry. The glass is then passed through the flame of a small Bunsen burner, and afterward cooled. It is next placed face downward in a watch glass upon two or three drops of a freshly-filtered solution of magenta crystals (two grm.) and pure anilin (three grm.) in equal parts of alcohol (sp. gr. 830) and distilled water (twenty ccm. of each). After fifteen to twenty minutes the cover is removed, and washed with a dilute solution of nitric acid (one part to two of water) until all color has gone. Then it is washed with distilled water, when a faint color reappears. Next it is placed face downward upon a few drops of a saturated solution of chrysoidin, until it has taken a brown color. After being removed, it is allowed to become perfectly dry in the air, and it is then mounted.*

Dyspnœa is a much less marked symptom than might, perhaps, have been expected. The gradual onset of the disease and the development of anæmia *pari passu* with the destruction of pulmonary tissue, probably accounts for the fact that a patient, even with advanced phthisis, is often able to breathe quietly, and to carry on conversation with comfort, so long as he is sitting still. And, as Sir Thomas Watson observes, nothing is more common than for persons who fear, but will not believe, that they are consumptive, to fetch a deep breath, and bid us remark how thoroughly they can distend their lungs. But any effort or exertion is almost always attended with obvious hurry of breathing in those in whom the disease has passed beyond its earliest stage. And, toward the last, orthopnœa is sometimes present in the most extreme degree, the patient gasping for breath, and having the face and hands livid and purple and bathed in sweat. It is probably as a consequence of obstruction to the pulmonary circulation that persons affected with chronic phthisis so often get clubbed finger ends with incurved nails; a change which, however, is, after all, a less marked feature of this disease than of some others.

Nor, again, is *pain* commonly distressing or troublesome in cases of phthisis. There may be pain in the shoulder or beneath the collar-bone

* [See also Dr. Gibbes's paper in the "*Lancet*" of August 5th, 1883; and Dr. Klein's account of Koch's original method, with those of Ehrlich and Weigert, in his "Micro-organisms and Disease," p. 119.—Ed.]

or lower down. But in many cases even this seems often to be muscular rather than deeply seated. The pleurisy which invariably fixes the lung to the surrounding structures as the disease advances must be supposed to be painless, for otherwise pain would scarcely ever be absent. But pleurisy lower down, where there is more movement of the parietal upon the pulmonary layer, is not uncommonly attended with sharp and piercing pain.

Hæmoptysis may be said to be present in every case in which mucous or purulent sputa contain streaks of blood, or in which they are uniformly discolored by admixture with it. But in practice it is necessary to distinguish from such conditions the expectoration of blood in a pure state, or frothy with air. In a very considerable proportion of cases this occurrence is the first thing which suggests that there is anything wrong with a patient's lung, or, indeed, that he is otherwise than perfectly well. He, perhaps, feels a little tickling in the throat and finds that his mouth contains a fluid which has a salt taste. He looks at his handkerchief and is horrified to see that it is stained with blood. He may either bring up a large quantity at once, or he may remain free from further hemorrhage for some hours, and then have a recurrence of it to a great amount.

From the days of Hippocrates it has been thought that the hæmoptysis is in such cases the cause of the consumption which ultimately develops itself; and two centuries ago Dr. Richard Morton included a *phthisis ab hæmoptoë* among his species of that disease. Recently the same doctrine has been revived by Niemeyer. Now, for my part, I think that only the most overwhelming evidence should lead to the acceptance of the opinion that the extravasation of blood into a lung is ever the starting point of disease spreading through its substance and destroying it. Under various other conditions—as, for example, after injuries to the chest, and in chronic heart disease—we have frequent opportunities of observing the effects of hæmoptysis. And I believe that no pathologist will assert that he has ever seen it give rise to phthisis. Nor have I ever observed any appearances which would lead me to believe that blood extravasated into the air passages is capable of being inhaled into the pulmonary tissue, so as to form solid nodules, as has recently been maintained by Dr. Reginald Thompson, in the "*Med.-Chir. Transactions*" for 1878. I have repeatedly met with cases in which inhalation of blood into the lung had obviously taken place; and what I have seen has always been a fine mottling of the cut surface of the organ with red or purple spots, impalpable, devoid of induration, and offering not the slightest resistance to the finger when passed over them. The formation of infarcts or nodules of pulmonary apoplexy is quite a different matter, and occurs only when the pulmonary circulation is in an abnormal condition. I cannot help maintaining the opinion that the cheesy, congested bodies to which Dr. Thompson refers are really relics, not of hemorrhages, but (at least in most cases) of tubercular lesions. According to Dr. Thompson, however, they are most often found in three situations—in the upper lobe, in the axillary region, and toward the base, but not posteriorly—which, he says, are notably those where inspiration produces the greatest expansion of the lungs.

A point mentioned by Niemeyer, and to which a certain importance has since been attached by some of those who have discussed this question, is that in one case, four weeks after an attack of hæmoptysis, he found a bronchial tube filled with adherent softening clot, giving it exactly the appearance of a vein obliterated by thrombus. A similar case has since been recorded by Dr. Weber in vol. ii of the Clinical Society's "*Transactions.*" It is to be noted, however, that in each instance the tube so affected was situated in the lower lobe of the lung. And I agree with Traube in thinking that such an appearance is so exceptional that very little significance can be

attached to it. As a rule, unless a patient has actually been suffocated by hæmoptysis, one does not find any clots in the bronchial tubes post-mortem. Sometimes, indeed, a clot of considerable size, with branches that had evidently extended into a number of the bronchi, is expectorated a few days after an attack of pulmonary hemorrhage. In the museum of Guy's Hospital there is such a specimen.

Otherwise, the evidence brought forward by Niemeyer in support of the existence of a *phthisis ab hæmoptoë* was mainly clinical. It consisted partly in the fact that hæmoptysis in patients who subsequently die of consumption often takes place at a time when no signs of any mischief in the lungs can be detected on the most careful examination; partly in the fact that the hemorrhage is frequently followed by fever, acceleration of the pulse, and signs of inflammation of the pulmonary tissue and of the pleura. The first point is, I think, worth noting. We shall presently see that auscultation and percussion frequently fail to reveal lesions which are really present in the lungs, if they happen to be situated deeply or to be scattered in the substance of the organ, widely apart from one another. Take, for example, the case of a patient who is attacked with hæmoptysis, but who recovers from it completely without the subsequent development of any disease, so that the origin of the hemorrhage remains a mystery. I recently had under my care an old lady, about seventy years old, who on two successive occasions brought up several ounces of blood, but who got quite well afterward and is now living, and who has at no time had any signs of mischief in the lung. The probability, I believe, is that she really had, and still has, a small old cavity or relic of a former phthisis, and that this was the seat of the hemorrhage. Dr. Weber has remarked that some of the patients who appear to get a *phthisis ab hæmoptoë* have had a tendency to epistaxis, and suggests that there is no reason why blood should not come from the mucous membrane of the bronchi in such persons as well as from that of the nose. But I cannot say that this appears to me likely. It often happens that hæmoptysis is directly traceable to some violent effort or strain, such as rowing, running a race, or lifting a heavy cask. But, of course, that fact is quite compatible with the existence of disease in the lung at the time.

Niemeyer's other point was that hæmoptysis is often followed within two or three days by an increase in the temperature in the body and in the frequency of the pulse, and by signs of inflammation of the lung and pleura. Traube remarks, in reference to this, that none of the cases cited by Niemeyer show that pyrexia was not really present at the time when the hemorrhage occurred. But, at any rate, a chart given by Bäumler in vol. ii of the Clinical Society's "*Transactions*" shows a rapid rise of temperature from the second morning after the commencement after the bleeding until the sixth day, when it reached 103.8°, and then a gradual fall until the eleventh day, when it became normal. And I think that it must be in the experience of every clinical physician that such a febrile attack of variable duration is of frequent occurrence after an attack of hæmoptysis, and that before it subsides one can often make out distinct signs of consolidation of one apex, which were absent when it began. Still, I cannot accept Niemeyer's interpretation of such facts. It seems to me far more probable that the hæmoptysis is itself a direct effect of the development of tubercles in the pulmonary tissue. When I come to speak of miliary tuberculosis of the lung, I shall point out that hemorrhage is no uncommon symptom of that affection, and may even be immediately fatal at a time when there is neither ulceration nor obvious consolidation of the lung substance, and when the only lesions found post-mortem are recent miliary tubercles which had apparently produced no other symptoms whatever. There is no doubt a difficulty in saying how the bleeding is brought about, but it seems very

likely that the growth of tubercles in the walls of the alveoli may be attended with an invasion and softening of the coats of many of their capillaries, while at the same time the blood pressure in them is augmented in consequence of compression of other capillaries. Rindfleisch, in Ziemssen's "Handbuch," gives a microscopical drawing showing the coats of a minute artery actually perforated by a tubercular cell growth.

But in other cases of phthisis, hæmoptysis is due to a very different cause, namely, to the rupture of the wall of a branch of pulmonary artery crossing the side of a vomica or enclosed in a trabecula. Rasmussen, of Copenhagen, first made known the fact that in many instances of this kind the hemorrhage is preceded by an aneurismal bulging of the coats of the vessel. A translation of his paper may be found in the "*Edinburgh Medical Journal*" for 1868. Since that time the occurrence of such aneurisms in vomicæ has been noticed by many observers. In the "*Pathological Transactions*" for 1871, Dr. R. Douglas Powell tabulated a number of cases that had been inspected by him. I have even met with a specimen in a child under three years of age. This instance is in itself sufficient to show that the formation of the aneurism is not the result of atheroma, like that of an ordinary aortic or popliteal aneurism. Rasmussen was inclined to attribute it to the unsupported state of the walls of the vessel when one side of it is exposed in a vomica. But Dr. Powell points out that the coats are much swollen, semi-gelatinous, and glistening; and I think it is clear that their yielding to form a pouch depends on a previous inflammatory change, more or less like that which causes aneurism in an artery occluded by an embolus. The size of an aneurism in a vomica is commonly from that of a pea to that of a nut. But Dr. Powell speaks of one which was as large as a Maltese orange. The vomica in which it is found is usually an old one, with fibrous walls. Hemorrhage may have recurred on several different occasions, at intervals of days or weeks, before the fatal issue. Indeed, death is not by any means always the direct result of an attack of bleeding, the patient, perhaps, sinking exhausted after having ceased to spit any blood for some days. But in other cases he may die almost instantaneously, with a rush of blood from the mouth and nose. Or he may even be choked by the blood before any of it appears externally, so that the occurrence of hemorrhage is not suspected until an autopsy is made. The point of rupture is usually a little hole or fissure just large enough to admit a probe. Again, instances are not uncommon in which, instead of having found an aneurism, the branch of pulmonary artery from which fatal hemorrhage had occurred is found to be simply perforated by a process of ulceration. In my experience at Guy's Hospital the one condition has been as frequent as the other. And, lastly, in some cases, even of advanced phthisis, in which the lungs contain many vomicæ, it is not possible, after the most careful search, to discover what has been the source of the hæmoptysis. No part of either lung may seem to be more deeply stained with blood than all the rest, even though death may have occurred almost immediately.

A point of some importance in regard to cases of ruptured aneurism, or laceration, of a branch of the pulmonary artery is, that the blood which is expectorated by the patient is usually found to be of bright red color. For some writers have insisted that when the source of the hemorrhage is doubtful, such an appearance must prove it to have been derived either from a bronchial artery or from a pulmonary vein. The only instance, however, in which I remember blood from the lungs being described as dark colored is one related by Niemeyer in his "Clinical Lectures." The patient had brought up enough blood to fill three basins within a few minutes; it was found to have a thin, frothy layer on the surface, but below this it was

coagulated into a dark, almost black cake. Any one not acquainted with the facts might, says Niemeyer, have supposed that it came from a profuse venesection. In all probability the bright red arterial appearance which is usually seen depends on its having become aerated either after its expectoration or while it is in the bronchial tubes, where it certainly often is freely exposed to the air, as is shown by the frothy state in which it reaches the surface of the body. At any rate, it is clear that in no case of hæmoptysis can the fact of the blood being bright red be taken as proving that it came from one kind of vessel in the lung rather than another. A further point of great interest, on account of its bearing on the question of a *phthisis ab hæmoptoë* is that in none of Rasmussen's cases of hemorrhage from aneurisms of the pulmonary artery was any recent pneumonia found at the autopsy, even when the patient had lived for some weeks. I do not know of any accurate temperature observations in such cases; but if it should hereafter be found that no pyrexia develops itself when, in a patient previously free from fever, hæmoptysis results from ruptured aneurism, or from laceration of a branch of pulmonary artery, it would give the *coup de grâce* to Niemeyer's view.

Hæmoptysis, like so many other hemorrhages, has been supposed by some observers to be frequently *vicarious* of the catamenial function. Sir Thomas Watson, for example, says that this is not at all uncommon, and that it is not usually attended with any peril to life. He even cites a case which was observed by Pinel at the Salpêtrière, that home of all that is marvelous in disease, in which a woman was said to have menstruated through her lungs from the age of sixteen to that of fifty-eight, often to the extent of two quarts of blood during a period of two days, while she nevertheless remained plump and healthy. A very different view of this question is taken by Rühle, who will only admit that in patients who already have lung disease suppression of the catemenia (or, in other instances, of a hemorrhoidal flux) may be followed by vicarious hæmoptysis. He speaks of having seen cases in which this recurred at intervals of from four to six weeks, until a few leeches were applied to the anus with a corresponding regularity.

In almost all cases of hæmoptysis, if the bleeding should cease, there is for some little time afterward a continuance of expectoration of a deeply blood-stained material—clotted blood, or mucus intimately mixed with blood. This is gradually found to alter in appearance, becoming reddish brown or brownish black in color. Such a change in it should be carefully noted, because it shows that the hemorrhage is in reality no longer going on, and, perhaps, that treatment may be made less active. But, of course, there is still reason to fear that fresh oozing may at any time occur.

Concretions.—When the tubercular process in a part of the lung has become quiescent, and calcification of some of the cheesy material has occurred, it not uncommonly happens that the patient ultimately spits up the concretions which are thus formed, and which may be of all sizes up to that of a pea. Sometimes their detatchment from the tissues in which they had been embedded is attended with a little hemorrhage, and Rühle seems to think that there must necessarily be at the time some fresh softening, so that a further advance of the disease may be anticipated. Indeed, that the expectoration of pulmonary concretions is unfavorable was long ago stated by Morgagni. But the little experience I have had with regard to this occurrence would lead me to believe that in many cases it is not followed by any serious consequences. I have known it take place at a considerable interval of time after the subsidence of all active symptoms of lung mischief; and the patient to whom I refer is actually living at the present time. It must be remembered, too, that exactly similar concretions may come from the substance of a mediastinal gland, having reached the trachea, or one of the

bronchi, by ulceration. A case in point occurred among my out-patients in 1874; the man, who had been spitting up pieces of calcareous matter every two or three weeks, was admitted into the hospital and died there; and at the autopsy it was found that round the affected gland there was an abscess which had opened into the œsophagus as well as into the right bronchus.

Physical Signs.—The physical signs of phthisis are those of a slow and progressive consolidation, followed by excavation of the affected parts of the lungs.

At the commencement of the disease the signs may be very slight and doubtful, and repeated examinations at intervals of some days, or even two or three weeks, may be required, before one ventures to express a positive opinion as to whether mischief is developing itself or not. Among the earliest changes to be detected is often a diminished mobility of the upper part of the chest on one side. Standing behind the patient, with one hand placed lightly below each of his clavicles, the physician can feel that the expansion of the two sides is not equal; one lags slightly behind the other, or one stops in its movements while the other still continues to rise. On percussing with great care, and comparing closely corresponding regions of the chest, he may make out that there is more or less decided deficiency of resonance, amounting, perhaps, to actual dullness, either in front or behind. A good plan is gently to flick the two clavicles in turn with the finger; the resulting "osteal" sound may then be mixed with unequal degrees of pulmonary resonance on the two sides. It is important to examine the spaces above the clavicles as well as those below them; and by employing different amounts of force in succession one may sometimes find that a particular kind of stroke elicits an impairment of resonance better than others. The suprascapular regions must also be carefully percussed; a firm blow is required to bring out differences of sound there. On auscultation it may be found that the vesicular murmur is not alike on the two sides. If over one apex it is permanently deficient, or even absent, there can be no doubt that that is the lung which is affected, but it must not be forgotten that a temporary disappearance of breath sound may be due merely to plugging of a bronchial tube with mucus. In other cases the presence of tubercles causes the vesicular murmur to be louder or harsher than natural. It is then often difficult to determine by auscultation alone which of the two lungs is most likely to be the seat of disease, for an abnormally loud vesicular murmur, instead of indicating mischief when it is heard, may be "compensatory" of mischief on the opposite side. Various modifications in the character of the vesicular murmur may also be present in early phthisis. It may be interrupted or divided into two or three distinct parts corresponding with irregularities in the play of the chest walls. Or it may have a peculiar jerking quality, which has been compared with the sound produced by a revolving cogged wheel. This must not be taken as necessarily showing that disease is present, for Dr. Walshe has "observed it at one or both apices, when free from consolidation of any kind." The case to which he alludes was that of a female, and in all probability the "cogged-wheel rhythm" was due to the action upon the healthy lung of an irritable heart. For I have repeatedly noticed that the separate sounds which make up cogged-wheel breathing are synchronous with as many cardiac pulsations, and Potain in 1877 pointed out the same fact in the "Revue mensuelle." It seems likely that when a portion of the lung is partially solidified by tubercles the shock given to it by the beating of the heart, whether directly or through the blood-vessels, may produce a greater effect than normally on the air-cells which still receive air. And it may also be noted that one of the signs of tubercular disease of the anterior edge of the lung is an in-

creased loudness of the cardiac sounds in the corresponding subclavian region. In some cases, too, there is heard over the pulmonary artery a systolic murmur, which is supposed to be due to compression of that vessel by the lung. Another early sign of phthisis is the presence of moist sounds at the affected apex. In some cases they are audible only just after the patient has coughed, so that one must never conclude that they are absent until one has listened over the apices while making him cough. If limited to the upper lobe, moist sounds are of special diagnostic significance, since a simple catarrh is probably never thus localized.

No doubt a considerable amount of consolidation may take place in the apex of a lung without any physical signs being audible beyond those which are mentioned in the last paragraph, but as the process of solidification goes on it almost always happens before long that bronchial breathing is discoverable. At the same time dullness on percussion becomes more marked than before, and the voice is transmitted to the stethoscope with increased loudness, constituting bronchophony. These signs possess an importance which cannot be exaggerated, but at the same time it is essential that one should always bear in mind what has been stated at pp. 869, 874, about the normal presence of bronchial breathing and of bronchophony in certain regions of the chest, especially in some people. And I think it should be added that throughout the right suprascapular, supraclavicular, and subclavicular regions the voice may in health be heard more loudly than in the corresponding left regions, though the difference is too slight to justify our speaking of the sound on the right side as bronchophonic. Moist sounds may or may not accompany the bronchial breathing of phthisical consolidation. When they are present, they generally have a markedly consonating character. A very common combination is for the inspiration to be attended with râles of various degrees of fineness, so that no blowing sound is noticeable, whereas during the expiration, immediately afterward, a blowing sound is heard, but no râles.

It might have been expected that tuberculous consolidation of a part of the lung should tend rather to enlarge it than to reduce it in size, but the contrary is, in fact, the case. From a very early period of the disease the regions above and below the clavicle on the affected side are commonly found to be flattened or even slightly hollowed. Rühle also lays stress on the fact that even when no dullness on percussion can be detected in the supraclavicular space one can often make out that resonance reaches upward for an inch or an inch and a quarter above the clavicle instead of an inch and a half or two inches.

The quality of the bronchial breathing in a case of phthisis may be modified to tubular, or even to cavernous, without there necessarily being any further change in the affected part of the lung beyond consolidation, and, conversely, excavation may take place to a considerable extent, without the physical signs necessarily indicating it by any modification in their quality, but such instances, one way or the other, are doubtless altogether exceptional. The formation of vomicæ is an occurrence so nearly universal that one is scarcely ever wrong in diagnosing the presence of a vomica at whatever spot happens to be the seat of well-marked "hollow" sounds.

On the other hand, it is not infrequent for a phthisical cavity to become so large that the recognition of the physical signs to which it gives rise is really the prominent feature in the diagnosis of the case. The corresponding part of the chest may fill out, and even bulge slightly, when the previous consolidation had given rise to a capacious hollow, as in a case observed by Dr. Walshe. One might, perhaps, have anticipated that under such circumstances the percussion sound should become hyper-resonant. It has, however, long been known that this is not so; the thick, adherent pleura and

the condensed lung tissue round the wall of a vomica serve effectually to check the vibrations of the thoracic parietes, so that a toneless noise always forms a large part of the sound which is elicited on percussion. But mixed with this are tones of varying quality, due to the vibration of the air within the vomica itself, and thus the sound as a whole may present all those modifications enumerated at p. 866, from "osteal" to "tympanitic." A further peculiarity of the percussion sound over a large vomica is, in many cases, that it resembles the noise produced by striking coins together, or by striking over one's knees the hands loosely clasped. Laennec called such a percussion sound the *bruit de pôt fêlé*, a term which in English has been rendered into "cracked-jar sound." For its production in a perfect form the walls of the cavity itself and the thoracic parietes must be elastic and yielding, the percussion stroke must be heavy and forcible, and the cavity must communicate freely with the bronchial tubes, and these again with the external air through an opened mouth. The reason is that the *bruit de pôt fêlé* depends upon the expulsion of air from the cavity, just as in striking the hands over the knee one drives air out through a chink between them. The most marked example of this kind of percussion sound that I have ever met with was in a patient who had, outside the thorax, beneath the pectoral muscles, an abscess cavity which contained air, and which communicated with the pleural space (itself filled with air) by a narrow hole through the intercostal muscles. But there are, in fact, many other diseased states of the respiratory organs in which the *bruit de pôt fêlé* may occur. Thus, according to Dr. Gee, it is sometimes obtained over the upper part of the front of the chest in cases of pleuritic effusion, sometimes over islets of unsolidified lung embedded in tissue hepatized in acute pneumonia, sometimes in cases of malignant tumor. Nevertheless, in phthisis I believe that there is no likelihood of one's falling into error by taking it as significant of the presence of a cavity.

Precisely similar in its mode of origin to the *bruit de pôt fêlé* is a phenomenon which sometimes attracts the notice of the patient himself as well as of other persons, namely, the transmission of the heart sounds outward so that they can be heard, like the ticking of a watch, at a distance of several feet from him. Many years ago my father showed me a case of this kind, which had come under his observation. The sounds were sometimes audible across a good-sized room, but I found that when the patient, a young woman, was made to close her mouth, I could instantly stop them by pressing together her nostrils. Just such a case was brought under the notice of the Clinical Society in 1880, by Dr. Frederick Taylor. They would doubtless be much less rare than they are were it not for the fact that a cavity of sufficient size to have a good quantity of air driven out of it by each pulsation of the heart very seldom exists in any part of the lung except the upper lobe. Indeed, the disease in Dr. Taylor's patient was supposed to be, not phthisis, but chronic pneumonia; and perhaps I ought to have dealt with this form of "cardio-pulmonary" bruit when speaking of that disease rather than in the present chapter.

On auscultation over a large vomica one may obtain any modification of bronchial breathing up to the amphoric. Another modification, which appears to be heard only when a cavity has been formed, has recently received from Seitz the name of "metamorphosing murmur." It is probably not very different from what Laennec long ago described somewhat vaguely as the "souffle voilé." It is said to be characterized at the commencement of inspiration by an unusually harsh sound, which lasts only during one-third of the inspiratory period, giving place during the remaining two-thirds to bronchial breathing accompanied by a metallic echo, or to ordinary râles. All kinds of metallic phenomena may present themselves

in a very large vomica, exactly as when there is pneumothorax. The moist sounds are often very "large," so as to claim the designation of gurgling. Vocal resonance often amounts to pectoriloquy. On the other hand, Dr. Walshe insists on the fact that over a large cavity, at least at its upper part, there *may be dead silence*, both respiratory and vocal.

This is, perhaps, the most convenient place for me to mention one very rare effect of excavation of the lung, namely, the production of subcutaneous emphysema. A case in point came under my notice in 1882. The patient had been slowly sinking for weeks, and the last time I saw him I happened to notice a slight crackling below the clavicle and at the root of the neck. Before his death, which took place a day or two later, the affection became unmistakable, as I was informed. Fräntzel, in Ziemssen's "Handbuch" (vol. iv, Heft ii, p. 545, 2te Auflage), alludes to similar instances. As pneumothorax is not present, it must be assumed that ulceration extends through both layers of the pleura, the space between having been previously closed by adhesions.

The physical signs of quiescent or retrogressive phthisis vary widely in different cases. Shrinking of the upper part of the chest may go on until the clavicle is seen to be obviously at a lower level than on the healthy side. Dr. Walshe also says that the corresponding suprascapular region may be distinctly hollower than its fellow. Unless excavation of the lung has gone on to a great extent the percussion sound is usually very dull; indeed, Rühle remarks that extreme dullness in phthisis is usually a sign that the case is likely to run a favorable course. The heart becomes uncovered by retraction of the lung, especially if the left is the one affected. Its impulse may be seen and felt over a much more extensive area than is naturally the case even as high as the third or the second intercostal space. The stomach also may be drawn upward to the level of the sixth or the fifth rib. On the other hand, if the right lung is diseased, the heart's apex may be displaced to the right side of the sternum; and the liver may be dragged up as high as the fourth rib.

In all cases of phthisis it is, of course, very important that, while one is watching the changes that take place in the region first affected, one should also be on the lookout for signs of extension to other parts of the same lung as well as to the opposite lung. The frequency of cavitation in the apex of the lower lobe, to which I have already drawn attention, makes it advisable to auscultate very carefully over the scapula, below the spine of that bone, as I find Rühle remarking. In advanced cases, the question to which one should mainly direct one's attention is very often not what parts of the lungs are diseased, but what parts remain capable of carrying on the function of respiration. And it is surprising to how small an area, at the extreme base of one lung, one may find the presence of a vesicular murmur restricted. At this point, however, it is exceedingly harsh and loud, affording, in fact, a most typical example of "compensatory" or "puerile" breathing. On the other hand, one must not overestimate the significance of crepitations and râles, when heard over the whole of the back of a lung, as proving, even if they are consonating in character, that the corresponding lung substance contains more than scattered or clustered tubercles. In speaking of miliary tuberculosis I shall mention instances in which it had appeared during life that large tracts of the pulmonary tissue were breaking up, and in which it yet turned out that the pulmonary tissue between the tubercles was still crepitant. But in the majority of cases of phthisis, the discrepancy between physical signs and post-mortem appearances is in the opposite direction. Clinically, disease is perhaps discovered in the upper lobe of one lung; the autopsy shows that nearly the whole of that lung is affected, and also the upper lobe of the other lung. This is only in part to be explained by the

extension of the mischief in the interval that may have elapsed. On the contrary, I believe it to be very important to recognize frankly the fact that the presence of well-marked disease in one apex adds greatly to the difficulty of the detection of early mischief in the other apex. The reason obviously is that one has lost the standard of comparison on which one is accustomed to rely.

The *diagnosis* of phthisis, which (as we have seen) must be based upon symptoms as well as signs, is often very simple and easy. But there are cases in which there is the greatest difficulty in arriving at a right conclusion, and in which, indeed, the only safe course is to reserve one's opinion, at any rate, for a time. As between phthisis and other pulmonary affections, I have already spoken in previous chapters. In practice the doubtful cases are generally rather those in which physical signs are either wanting or at least slight and obscure, so that one hesitates as to whether the disease is in the thorax, or whether there is not rather some deeply-seated new growth, or some lesion of the internal lymph glands, or of the thoracic duct, or of the great abdominal nerve centres, by which the patient is wasted and worn down. Very often, however, although the nature of the affection cannot be determined, it is clear that he is stricken by fatal disease of some kind. For my own part, I must confess that under such circumstances my inability to give an exact diagnosis disturbs me scarcely at all. It is quite otherwise when, as is sometimes the case, the prognosis depends absolutely upon the opinion one may form. The doubt generally, then, is whether the patient, if a man, may not be merely suffering from the syphilitic cachexia, or be the victim of hypochondriasis and of aggravated dyspepsia; if a woman, whether she is not hysterical. In all cases of this kind the thermometer is of the greatest value. One hysterical affection which has often been mistaken for phthisis—the "anorexia nervosa" of Sir William Gull—will be fully described elsewhere. But there are other cases in which the suspicion of lung mischief is based mainly upon the fact that the girl, as is said, "spits blood." A glance at the sputum is sometimes sufficient to remove all uneasiness about this. What is expectorated may be found to be a rather slimy liquid, uniformly tinged of a pink or purple color, so that it looks exactly like the juice of plums or of some other fruit. It is, in fact, saliva or secretion from one part of the mouth; and the blood comes from the vessels of the mucous membrane. Rühle remarks that this sort of hemorrhage often occurs in the night, from the patient making sucking movements of the lips and cheeks during sleep. Thus the pillow may show stains of blood, the origin of which seems at first to be inexplicable. Another variety of sanguineous expectoration, which is equally unimportant, is due to the rupture of small vessels at the back of the fauces during violent coughing, or "hawking up" of phlegm.

The *course* of phthisis varies greatly in different cases, but its duration is almost always a matter of several months, and sometimes of many years. Trousseau is, no doubt, right in saying that the only *phthisie galopante* is miliary tuberculosis of the lungs. Traube, indeed, related in the "*Berlin. Klin. Wochenschrift*" for 1867 the case of a man, aged twenty-eight, who died, after thirteen days' illness, of "acute tubercular (caseous) pneumonia." The attack began with rigors and fever; a few days later hæmoptysis set in and became one of the chief symptoms. At the autopsy all parts of the left lung presented patches of lobular hepatization, the centres of which were caseating, especially in the upper lobe. A similar affection, in an earlier stage, existed also in the right lung. Both apices, moreover, showed traces of old mischief. I have notes of eight cases which occurred in Guy's

Hospital, and in each of which there was a definite history that the duration of the patient's illness, from its commencement to its fatal termination, was only from five to twelve weeks. In two instances the attack was attributed definitely to a chill; one man said that he got wet through while working in a potato field, after which he shivered and became hot, and was never well again; the other, that on a particular occasion he slept with his window open. In almost every one of these cases vomicæ had formed before death, especially in the upper lobes, in the centres of the cheesy masses, which formed the most conspicuous lesions observed at the autopsy. It must, however, be borne in mind that the distinction from miliary tuberculosis may, sometimes at least, be not very apparent. As I shall remark in the following chapter, the dissemination of the tubercular virus by the blood current may, if the tubercles to which it gives rise in the lungs are not very numerous, have no apparent effect until they, in their turn, become starting points of a local infection, when a disease exactly like ordinary phthisis may be supposed to arise.

The sudden commencement of some of the rapidly fatal cases of phthisis to which I have been referring is of great importance in regard to their diagnosis from cases of acute croupous pneumonia of the upper lobe of a lung. The most serious errors of diagnosis have been made between the two diseases; but I was until lately under the impression that the mode of onset afforded a ready means of arriving at a right judgment. It is, however, evident that this is not always the case; and the only point on which one can fall back seems to be one to which Traube has drawn attention, namely, that in acute phthisis bronchial breathing is not discoverable until much later than in pneumonia of the upper lobe—not until the end of the second week, or even for a longer time still. It is a striking fact, that when croupous pneumonia occurs in a person who already has phthisis it often seems to run as favorable a course as if it had arisen in one who was healthy. Thus, Andral is said by Rühle to have seen a single phthisical patient pass through from twelve to fifteen successive attacks of pneumonia. If one finds very extensive consolidation in a case of phthisis when it first comes under one's observation, one should always think of the possibility that it may, in part at least, be the result of croupous pneumonia, and, therefore, that the prognosis may be far less grave than it otherwise would have been.

But even acute phthisis—"phthisis florida," as German writers term it—may, instead of running on straight to a fatal termination, become arrested, and afterward run a chronic course. Rühle relates a case in a girl who seemed to have but a short time to live when she was transferred to his charge from that of his predecessor at Greifswald, Niemeyer. Yet her symptoms subsided, and she was discharged from the hospital with signs of a cavity in the left upper lobe, and did not die until the following year, having in the meantime given birth to a child.

The progress of ordinary chronic cases is, almost without exception, interrupted by intervals, during which the patient may actually seem to regain his health. Cough may almost disappear, even the evening temperature becomes normal from day to day, the appetite returns, the face is no longer pale, the weight of the body becomes as great as it used to be. It is, of course, true that this favorable change commonly takes place under medical advice, and we shall presently see how important it is that the advice should be well carried out. But sometimes it occurs even in those persons who are not able to do so, and who have gone on working in spite of their illness. In January, 1874, a hatter, aged thirty-seven, came to me with signs of phthisis at both apices, who said that he had been ailing for six months. His morning temperature was 101.4°. His father had died of

consumption. Notwithstanding my urgent recommendation to him to give up work he did not rest for a single day. The only difference he made was that instead of living away from his workshop in the Borough, so that he was exposed to changes of temperature in going backward and forward, he now slept in the same building. For a week or two the physical signs increased, moist sounds becoming audible all over the left lung. But his symptoms quickly improved, and by the end of May he was as stout as ever and said that he felt nearly well. The signs at the apices, however, still remained. In the following year I heard incidentally that he was in good health, with only a little occasional cough. But in 1879 his symptoms returned, and he ultimately died in September, 1881.

Different observers have made widely different estimates as to the duration of phthisis. Sir Thomas Watson cites Dr. Gregory, of Edinburgh, as having stated that the "ordinary duration" of the disease was about six months. Laennec and Andral each put the "mean duration" at about two years. Dr. Pollock, analyzing 3566 cases observed by him at the Brompton Hospital, found that the "average duration" of these cases while under observation was more than two years and a half, and in the course of that time only 127 ended fatally. What was the real average length of the disease among the whole number of cases he could not tell, but it must clearly have been much larger still. It is, however, very difficult to believe that Dr. Pollock's cases fairly represent the ordinary course of the disease. There must, I think, have been a very undue proportion of exceedingly chronic cases, and cases running a rapid course must in some way have become excluded. Still more extraordinary are the statements made by Dr. Theodore Williams, in vol. liv of the "*Med.-Chir. Transactions*," with regard to the duration of life among 1000 cases of phthisis in private practice. Of the patients in question 198 were known to have died; in them the average duration of the disease was nearly seven years and three quarters. In the remaining 802 patients who were alive when last heard of, its average duration had already been more than eight years. Among these cases, however, none were included which had not been at least one year under observation, and this restriction, besides keeping out of the list all rapidly fatal cases, doubtless weeded it of the majority of those who failed to improve for a time under the treatment recommended. That life is sometimes maintained for a great length of time after phthisis has developed itself has long been well known. Sir Thomas Watson alludes to a patient of Dr. Gregory's who was at least seventy-two years old when he died, and who from the age of eighteen had never been free from symptoms, "being often hectic, and frequently spitting blood."

On the other hand, the fatal termination of the disease is not seldom, when it does occur, sudden and unexpected. In 1866, a gentleman, aged twenty-six, who had long been ill, went up to London from Brighton one day, to transact some business. At the London Bridge Station he was seized with alarming symptoms, and was taken down to Guy's Hospital, where he died within a quarter of an hour from the beginning of the attack. In 1868, a laboring man, aged twenty-three, who had been indisposed for some time, was at his usual work near Guy's Hospital, when about 2 P. M. he began to suffer from dyspnœa; this rapidly got worse, and he was carried to the hospital and died in two hours. In neither case did the autopsy show why death should have occurred at the time.

Pneumothorax, however, often brings more or less immediate danger to life in those who have phthisis, as I have pointed out above (p. 952); and another accident that may happen to such patients is pulmonary *embolism*, resulting from the thrombosis of femoral veins.

Perhaps *syncope* is sometimes the cause of death; or it may arise from

sudden exhaustion of the respiratory centre, as in the cases related at p. 25. Indeed, it is not, I think, uncommon for consumptive patients to be found unexpectedly dead in the course of the night.

In other instances phthisis ends fatally by the supervention of tubercular disease elsewhere than in the lungs, by *tubercular meningitis* or peritonitis, or by tubercular disease of the kidney, or by solitary tubercle of the brain or spinal cord. Or the main feature of this case toward the last may be *diarrhœa* resulting from tuberculous ulceration of the intestine, or the dysphagia and the other distressing symptoms produced by a like affection of the larynx.

Some affections which appear to be inflammatory rather than tuberculous are also of sufficient frequency in phthisis to deserve mention. One such is suppurative peritonitis from impaction of the cæcal appendix, and another is simple abscess of the brain.

The coincidence of *fistula in ano* with phthisis is one which requires brief mention. Dr. Pollock points out that it occurs far more often in males than in females, and most commonly in persons who are no longer young, the most frequent age for it being from thirty-five to forty-five. The lung affection has very generally already advanced to the formation of vomicæ before the fistula appears. Many observers, including Dr. Pollock, are of opinion that in such cases no operation should be attempted; for, when it is successful, the phthisis is very apt to assume increased activity two or three months later. But I am not aware that there is any evidence that the cure of a fistula in a person not already consumptive renders him more liable than before to the supervention of pulmonary disease.

Finally, *lardaceous degeneration* plays a very prominent part in bringing to a close many cases of phthisis. If the intestines be involved, an intractable diarrhœa may result, which I believe cannot be distinguished during life from that which would arise from tuberculous ulceration. But it is chiefly by affecting the kidneys that this kind of degeneration acquires its clinical importance. General dropsy sets in and the patient acquires more or less of the appearance usual with those who are affected with Bright's disease. Indeed, I should mention that epithelial nephritis sometimes comes on in phthisis without there being any lardaceous change discoverable in the renal glomeruli or vessels even with the microscope. It is, therefore, not safe to diagnose a lardaceous affection of the kidneys from the mere fact that the patient has albuminuria. According to observations made by Dr. Williams, recorded in a paper read before the Royal Medical and Chirurgical Society in 1822, the occurrence of albuminuria in phthisis has the effect of masking the other symptoms, and especially of making the temperature range lower. Post-mortem, however, he found the tubercular lesions still advancing in his cases.

Prognosis.—From what has been stated in the preceding paragraphs it may easily be imagined that to give a correct prognosis in phthisis is no light matter. And, in point of fact, I believe that those physicians who have the largest experience are precisely those who most strictly abstain from attempting to predict the duration of life among their patients. Many writers divide the course of phthisis into three stages: the *first* stage they associate with the "formation" of tubercles, the *second* with their "softening," the *third* with their "elimination" by the process of excavation. Now, as I have already pointed out at p. 914, the moist sounds which are supposed to indicate "softening" are very apt to be fallacious. It is another objection to these so-called stages that at the best they have reference only to the local process in certain parts of the lungs, and not at all to the disease as a whole. For while vomicæ exist in one or both apices, tubercles are commonly being formed lower down. But the strongest objection of all is that to speak of

stages of phthisis leads almost inevitably to a complete misconception of their significance in prognosis. To every patient, as well as to his friends, it cannot but appear to be a matter of course that in a malady which, like phthisis, is almost inevitably fatal, the third stage must be the worst. And yet it is no paradox to say that the exact contrary would be nearer the truth. A factor which, more than any other, requires to be taken into account in attempting to determine the probable cause of the disease, is its greater or less tendency to advance rapidly in that particular patient. In different cases the differences in this respect are enormous; and it does not appear that any explanation of them can be given, except that, as a rule, the progress is quicker in those who have a strong inherited tendency to consumption than in those who have no such tendency. Now, the formation of a cavity of any size takes a considerable amount of time, especially if its walls are to acquire a smooth lining. Hence, whenever the morbid process spreads with much rapidity through one or both of the lungs, the opportunity for such cavities to develop themselves is wanting. In other words, the fact that a case presents the physical signs of the third stage is proof that its course has been such as generally warrants a comparatively favorable prognosis. And, in reality, patients with large vomicæ often go on year after year with but little change in their condition and even with fair enjoyment of life. Dr. Walshe speaks of two singers—a distinguished contralto and an excellent soprano—as having within his knowledge continued to perform at the opera, "while the excavating process advanced in their lungs."

On the other hand, one is in most cases compelled to speak very guardedly of the probable duration of phthisis, if physical signs indicate that the morbid process is still actively going on in any part of the lungs, whatever may be the stage to which it has reached in the apices. One must not forget, too, that although in the lung first affected its progress may have been slow, it may yet rapidly hurry on to a fatal termination when it passes to the other lung. The degree of severity of the general symptoms is, of course, very important in regard to prognosis, especially the rate of the pulse and the height of the temperature. But it must be remembered that debility and exhaustion may render the pulse rapid as well as activity of local mischief. And the existence of pyrexia, as has been shown by Dr. Theodore Williams, is not incompatible with gain of weight—nor even, I may add, with the subsidence of many of the other symptoms of the disease—provided that the patient eats and digests well.

It is a fact, to which I have not yet alluded, that the majority of cases of acute or "pneumonic" phthisis occur in young subjects, whereas "fibroid" phthisis, which is necessarily a chronic form of the disease, is most frequent in those who are advanced in years. This seems to have led to the idea that the prognosis should be more favorable in proportion as the patient is older. Dr. Walshe, however, says that his observations at the Brompton Hospital failed to confirm such an opinion; and Lebert (in vol. xi of the "*Deutsches Archiv*") has pointed out that age seems to have little influence on the intensity of the pyrexia, which we have seen to be one of the most important factors in determining the rate of progress of the disease. Indeed, it is obvious that the greater frequency of very acute and of very chronic cases respectively in youth and at an advanced period of life affords no real reason for supposing that cases of what may be called an average degree of severity shall run a more rapid course at one age than at another. It can hardly be doubted that in this disease, as in almost every other, the patient's power of resistance and his capacity for repair must alike diminish as he grows older. And as we have seen that the prognosis of phthisis is always to be based mainly on the rate at which it seems to be actually

advancing in the particular case under consideration, I think it is clear that the patient's age may be altogether disregarded.

Contagion.—In discussing the subject of tubercle in general, I have maintained that although the growth and diffusion of a bacillus among the tissues of the body seems to be mainly concerned in determining the spread of tubercular lesions when they have once begun to develop themselves, clinical observation is nevertheless altogether opposed to the idea that infection from without is the most essential part of the ætiology of phthisis and of other tubercular diseases. Of the fact that consumption is not ordinarily communicable from one person to another, no better illustration could be given than a statement published in 1867 by Mr. Victor Edwards, who had then for seventeen years been resident medical officer at the Brompton Hospital. In that period he remembered personally fifty-nine resident medical assistants whose duration of office averaged quite six months. Of these he believed all but two to be alive; one had died of aneurism, one of some cause unknown; three, still living, were said to be consumptive. Very many nurses had been in residence for periods varying from months to eight, twelve, or even twenty-four years. Of the head nurses, who slept each in a ward of fifty patients, only two were known to have died—one of apoplexy; the other, after an unhappy marriage, of phthisis. No under nurse, so far as he was aware, had died of phthisis. The matron and her two predecessors, as well as the chaplain and his two predecessors, were all alive. Of the physicians, whether for in-patients or out-patients, all were living, except two; one had died of causes unconnected with disease of the lungs, the other from some disease of unknown nature, after twelve years' absence from the hospital. Mr. Edwards himself, at the end of seventeen years, was still in good health. The circumstance that phthisis does not ordinarily spread from a patient who remains in his own home to brothers, or sisters, or other relatives, is the more striking because they must be supposed to have very often inherited a more or less strong predisposition to the disease.*

Medical literature does, however, contain a single series of observations which tend to show that phthisis may be directly communicated from one human being to another. I refer to Dr. Weber's cases, recorded in the Clinical Society's "*Transactions*" for 1874, in which the disease seemed to pass immediately from husbands to their wives. The husbands, all of whom were affected before marriage with pulmonary mischief, were nine in number; but the deaths from phthisis among their wives were as many as eighteen; one lost four wives in succession, one lost three, four lost two each, three lost one each. In seven out of the nine husbands there was a decided family taint; the wives were with one exception free from any such taint, and they were all healthy at the time of marriage. The lung affection ran in all the wives a very rapid course, terminating in several instances within twelve months, and being never prolonged beyond eighteen months. It cannot be supposed to have been caused by anxiety or fatigue in nursing the husbands, for the husbands were all in fair health so far as appearances went, and none of them succumbed to phthisis until long after their wives. All the wives, with one or two exceptions, bore children to their husbands, so that it is, perhaps, possible that infection took place through the fœtus; but about the state of health of the children nothing is said. Dr. Weber seems to have been more disposed to think that the mere absorption of the seminal fluid led to the transference of the disease. But even if we admit that this was probably the case, I doubt whether it follows that there must have been an actual conveyance of a specific contagion. May not such cases merely afford another instance of that inexplicable influence of impregnation which stamps

* [On this subject see the facts recorded in the "Report of the Collective Investigation Committee of the British Medical Association," July, 1883.—Ed.]

on the female organism the characters of the male, so that they can be transmitted long afterward to offspring by a different male? Cases of this kind among the lower animals are well known to be frequent; and I believe that similar instances occur as the result of sexual intercourse between human beings belonging to different races. Not long ago I met with what, if not merely accidental, was probably an example of the same thing. A candidate for life insurance, whose mother had had two husbands, told me that the first husband and several of his children had died of phthisis; the second husband was free from all tubercular tendency, but the eldest of the offspring of this marriage nevertheless became affected with the disease. I ought, perhaps, to mention that Dr. Weber was acquainted with thirty other consumptive husbands whose wives escaped phthisis. But that figure must not be taken as indicating, in conjunction with the other, the proportionate frequency of such transmission, for this is probably very much more rare. Among twenty-nine consumptive wives who married healthy husbands, only one lost a husband from consumption.

Ætiology.—If now we turn to consider what are the conditions which in clinical practice are concerned in bringing about the development of phthisis, we shall, I think, find that they may be roughly arranged in three groups.

I. Some affect the original constitution of the individual from before birth.

II. Some affect the general health of the individual in the course of life.

III. Some affect the lungs themselves.

I do not mean to imply that it is always easy to say to which category a particular cause of phthisis belongs. It is enough for my purpose if it be admitted that all these groups exist.

I. *Conditions which affect the original constitution of the individual from before birth.*—The first of these to be discussed is *hereditary transmission.* It is a matter of universal experience that in some families deaths from phthisis occur, generation after generation, with terrible frequency. Parents and their offspring are swept off in turn, so that sometimes there is hardly a survivor to maintain the stock. Actuaries are so impressed with these facts that whenever it can be ascertained, in reference to a candidate for life insurance, that he has lost a parent or more than one brother and sister from consumption, it is held at almost all offices that an addition to the premium is absolutely necessary to cover the increased risk, and if both parents have died of the disease, or more than two other near relatives, the "life" is generally regarded as almost uninsurable, at least on reasonable terms. It might at first sight appear strange that an augmented liability to what is (after all) only one among a great many other possible causes of death should be taken as diminishing to so great an extent the general "expectancy" of the candidate, but the requirements of the offices are in practice found to be fair and equitable. From a scientific point of view, however, the question of the inheritance of consumption requires far more consideration than has generally been given to it. Among persons actually affected with phthisis, the proportion of cases in which the occurrence of a like disease can be traced in their relatives appears from certain investigations made by Dr. Theodore Williams, and recorded in the "*Med.-Chir. Trans.*" for 1871, to be 48.4 per cent. The patients were seen in private practice, so that the results are probably as little inaccurate in the way of omission as can ever be expected in such inquiries. It would be an advantage, for the purpose of comparison, if we knew to what extent a similar family tendency exists in the population generally or among those who are not themselves phthisical. But the truth is, I think, that it is impossible to accept the figures given by Dr. Williams, or any similar figures, as really indicating in any scientific sense the exact extent to which consumption is transmitted by inheritance.

The point on which I would insist is often brought out very clearly by proposers for life insurance themselves. A candidate has had, perhaps, two or three brothers who were consumptive, but one, he says, brought on the disease by dissipation and intemperance; another was in the army, and was stationed first in India and then in Canada, at a few months' interval; a third may have got a chill in bathing; and he winds up by declaring that phthisis has not been a "family complaint" after all. Now, among the cases collected by Dr. Williams, 484 in number, in which phthisis was traced among the relatives of patients themselves phthisical, there were 120 in which the disease had existed in one or both of the parents, but 224 in which it affected only brothers or sisters. And I have not the slightest doubt that if inquiries had been made as to the existence of definite "exciting causes" of the disease in these cases they would have been found to be very often present. I am satisfied that it is impossible to draw a line anywhere between what might be called respectively "hereditary" and "accidental" phthisis. Probably there is no family in which the consumptive tendency is so strong that it could not be kept in abeyance by hygienic precautions if they were thoroughly and vigorously carried out, and, on the other hand, there are very few families, if any, in which the disease may not show itself in such members of it as systematically neglect their health, or are exposed year after year to unfavorable circumstances.

I must not be understood to mean, in urging these considerations, that I disbelieve altogether in the transmission of a definite tendency to phthisis from parent to child, nor even that when the disease appears in several children of the same parents, who themselves are healthy, it may not have been derived from a more remote ancestor. (The occurrence of such *atavism* has not, so far as I am aware, been hitherto verified in regard to this disease.) But I do maintain that it is impossible at present to determine in what proportion of cases the so-called "family predisposition" to consumption implies the actual transmission of a tendency to the disease, and in what proportion of cases it is merely the expression of a general delicacy of constitution, or (as the Germans would say) a "vulnerability," which belongs to all those who are derived from certain stocks, and renders them liable to be attacked by the disease in succession, as they happen to come under conditions suitable to its development. From the point of view of the insurance offices the distinction is not material, for in either case the demand for an enhanced premium is equally justifiable and necessary. One fact which tells strongly in favor of the opinion that family predisposition is often a mere vulnerability, is that the liability to consumption is believed to be also much above the average in those who come from parents already failing in health from any cause, in those begotten by a father very advanced in years, in those born of a very young mother, and also in the later offspring of a woman exhausted by very frequent and rapid child-bearing. The same opinion is further supported by the circumstance that very little seems to have been ascertained as to the existence of a specially strong tendency to phthisis in the children of parents actually consumptive, one or both of them, at the time of procreation.

Diathesis.—It is a very old suggestion that persons of a particular bodily frame and physiognomy are especially liable to tuberculous diseases, but little value can be attached to the statements of early writers on the subject, because "scrofula," as it was called, was not so very long ago confounded with rickets. And, according to Sir Thomas Watson, the numerous signs of the "scrofulous diathesis" varied widely with the "temperament" of the individual, whether "nervous," "sanguine," or "bilious." Obviously this was very confusing. It, therefore, seemed a great step in advance when Sir

William Jenner, in 1860, proposed to distinguish two separate diathetic states, which he termed respectively *tuberculosis* and *scrofulosis*.

As leading features of *tuberculosis* he gave the following: "Nervous system highly developed; mind and body active; figure slim; adipose tissue small in quantity; organization generally delicate; skin thin; complexion clear; superficial veins distinct; blush ready; eyes bright; pupils large; eyelashes long; hair silken; face oval, good looking; ends of long bones small, shafts thin and rigid; limbs straight. Children the subjects of tuberculosis usually cut their teeth, run alone and talk, early."

Scrofulosis he described as follows: "Temperament phlegmatic; mind and body lethargic; figure heavy; skin thick and opaque; complexion dull, pasty looking; upper lip and alæ of nose thick; nostrils expanded; face plain; lymphatic glands perceptible to touch; abdomen full; ends of the long bones rather large; shafts thick."

Among the pathological tendencies of the former morbid conditions he mentioned not only "deposits or formations of tubercles," but "fatty degeneration of liver and kidneys, and inflammation of the serous membranes." To the latter he assigned "inflammation of the mucous membranes of a peculiar kind; so-called strumous ophthalmia; inflammation of the tarsi; catarrhal inflammation of the nose, pharynx, bronchi, stomach, and intestines; inflammation and suppuration of the bronchial glands on trifling irritation; obstinate diseases of the skin; caries of bone."

Many physicians still believe that Jenner's descriptions correspond with two great types, the recognition of which is really important in practice. But the idea of their representing separate diatheses is quite incompatible with modern views as to the relation between tubercle and caseating affections of lymph glands; no less incompatible with Buhl's theory of infection than with the more recent opinion, which I hold, that the glandular affections are themselves tuberculous. And it is clear that there has never yet been any general consensus among observers of experience with regard to the outward signs which indicate a tendency to tuberculous diseases. For my own part, while admitting that many of those who die of phthisis present the configuration spoken of by Jenner as characteristic of scrofulosis, I think that a very much larger proportion are ill-grown or badly-developed individuals, without any definite indications of a special diathesis. And, on the other hand, his description of tuberculosis seems to me to be made up merely of those characters which, while not incompatible with rapid and symmetrical growth or with physical beauty, show a want of real vigor and robustness. I doubt whether any more exact definition of the signs of scrofula, or of a liability to tuberculosis, can be given than that they include whatever indicates delicacy of constitution, incomplete growth, or imperfect development. Mr. Francis Galton and Dr. Mahomed have recently recorded in the Guy's Hospital "*Reports*" (1881) the results of "An Inquiry into the Physiognomy of Phthisis by the Method of Composite Portraiture." Their conclusions seem to me to bear out the view which I have stated. For although they were able to obtain from the photographs of 442 phthisical patients two types of face—the one of narrow ovoid shape, the other a broad face with coarse features—yet this was only by the careful selection of a few out of the whole number of cases; and they actually found a larger proportion of narrow ovoid faces among patients who were not phthisical than among those who were.

There still remains the question whether an inherited tendency to phthisis is indicated by any particular *configuration of the chest*. Dr. Gee describes two shapes of chest as occurring in "phthinodes," or persons predisposed to consumption. One of them he terms the "alar" or "pterygoid" chest, following Galen and Aretæus, who used the same name for it centuries before; this, he says, is narrow and shallow, the antero posterior

diameter being especially small, and the angles of the scapulæ projecting like wings; its peculiarities depend upon a drooping or undue obliquity of the ribs, as the result of which the shoulders fall and the length of the thorax from above downward is increased; the alar appearance is caused by the falling of the shoulders. The pterygoid chest is often accompanied by a prominent throat, due to a long neck, and by the head being carried unduly forward. The other is called by Dr. Gee the "flat" chest; this, instead of being rounded, is flat in front, the rib cartilages losing their curve and becoming straight. Nay, the sternum may actually be depressed below the level of the costal ends of the cartilages. Now, there can be no doubt that it is very important to be on the lookout for the flatness of chest, in judging the configuration of this part of the body. Persons who are extremely flat chested often have broad shoulders; so that, as one stands facing them, one might fancy them to be by no means ill developed. Traube and other German writers lay great stress on the significance of a flat chest as indicating a liability to consumption, and I have repeatedly heard Dr. Wilks insist on it strongly. But I must confess that I have never been able to satisfy myself of the existence of any definite relation of this kind. It seems to me altogether misleading to contrast the "flat chest of phthisis" with the "rounded chest of bronchitis," because the latter is merely an acquired condition and the result of that disease, of which it is no doubt an important clinical sign. I cannot make out that either the alar or the flat chest is seen among phthisical patients with more frequency than other varieties of ill-shapen chest, which so often result from neglect during childhood, and are consequently so very common among the poorer classes of the population. And, in general, I believe it to be impossible to say how far a badly-formed chest, in relation to the liability to phthisis, is important as interfering with the play of the lungs, and whether its significance is not merely that it is an indication of a defective development of the body in general.

Again, one cannot disassociate congenital from acquired deformities of the thorax in regard to their possible influence on the subsequent occurrence of tubercular disease of the lungs. Freund, in 1859, maintained that what caused a small and contracted chest was often a premature ossification of the cartilage of the first rib, occurring even in early infancy. It is worthy of notice that Dr. Hutchinson, in advocating the use of his spirometer, did not suggest that a defective vital capacity of the lungs indicated a tendency to phthisis, but rather that it was a sign of the actual presence of the disease, even if at an early stage. Again, deformity of the chest from lateral curvature seems certainly not to carry with it any increased liability to consumption. And it appears to be very doubtful whether the habit of stooping at a desk, or in the work of a tailor or shoemaker, or weaver, although it cannot but be injurious to the health, favors the development of lung disease. I also quite agree with Dr. Walshe that there is no evidence that the foolish practice of compressing the base of the chest by stays is capable of producing such an effect.

II. The conditions which, *affecting the general health of the individual in the course of life*, act as causes of phthisis are both numerous and varied.

a. Foremost among them I am disposed to place the *habitual breathing of air rendered impure by overcrowding or by defective ventilation*. It may, indeed, be a question whether this should not be placed under the third head, as affecting the lungs themselves, rather than the general health. But, on the whole, I think that it may fairly be dealt with here, though, of course, I leave to be discussed presently the no less important influence of such impurity of the air as results from the presence of minute particles of mineral or organic substances—fine grit, coal dust, powdered clay, flax-

dust, cotton dust, etc. The organic matter exhaled from the lung during respiration appears to possess directly poisonous products. Dr. Parkes cites some experiments made by Gavarret and by Hammond with air from which the carbonic acid and water produced by respiration had been removed, so as to leave only the organic matter; a mouse placed in such air died in forty-five minutes. Dr. Parkes says that he has known instances in which breathing for three or four hours air contaminated by having been previously used in respiration, caused headache and febrile symptoms which lasted one or two days. It is important to remember that such organic substances probably differ from gases like carbonic acid in having far less tendency to rapid diffusion through the atmosphere; they readily adhere to textile fabrics, especially those which are dark colored, and cling to them obstinately. Every physician is familiar with the peculiar odor belonging to the clothes of the women and children of the poorer classes; one perceives it as soon as they enter the out-patient room, even though they may have just been walking in the open air for a considerable distance. Precisely the same smell exist always in the rooms in which these people live. The organic matters which cause it cannot be removed by merely causing a current of air to blow through a room for a few moments in the day; still less will they escape through a door toward which there is no active draught. In all probability the only way of getting rid of them is by oxidation; and one may speculate as to the part which ozone is likely to play in accelerating their destruction, and also as to whether they do not disappear more rapidly when they are exposed to full sunlight than in comparative darkness.

Strictly speaking, overcrowding and defective ventilation are not convertible terms; but in practice one of them scarcely ever occurs apart from the other. It is not, indeed, inconceivable that one person or a few persons occupying a room of good size should so close up all the openings into it as to render the air in it impure. But, on the other hand, there is never overcrowding without bad ventilation, because when many persons are huddled together in a small space, the needful admission of fresh air always exposes some of them to cold draughts, the fear of which is sure to lead to the shutting up of one aperture after another.

The proof that impure air is a cause of phthisis rests mainly upon the evidence of statistics as to the frequency of the disease among soldiers and certain classes of workmen and among the inmates of prisons. As regards soldiers, a Royal Commission upon the Sanitary Condition of the Army, which reported in 1858, brought to light the fact that the death rate from consumption in all branches of the service was in excess of that of the civil population of large towns, and (what was most remarkable) that among the Foot Guards it was more than twice as great as that of the civil population. The only explanation that could be offered was that it came from defective barrack accommodation, since neither the clothing of the soldier, nor his food, nor the nature of his occupation, could be supposed to be the cause of it. There was evidence that in barrack dormitories the cubic space actually given to each man was often not more than one-half or two-thirds of the amount of 450 feet, which was the minimum allowed by regulation. It was also shown that the air in these rooms became very nasty and offensive before morning. The accuracy of the conclusion at which the Commission arrived has since been confirmed by the fact that a great fall in the consumptive death rate, especially among the Foot Guards, has followed the introduction of sanitary improvements.

As to workmen, we have evidence given by Dr. Guy before the Commission of Inquiry into the State of Large Towns, of which the Duke of Buccleugh was president, and which reported in 1844. Dr. Guy had

most elaborately investigated the relative liability to phthisis of different classes of the population of London. He found that the disease was more fatal to artisans than to tradesmen, and more fatal to tradesmen than to gentlemen (including professional men). Even hawkers, standing about in the streets and exposed to all inclemencies of weather, had the advantage over men employed in workshops. Among printers he instituted a very close comparison as to the frequency of symptoms of lung disease, arranging the men in classes according to the amount of air space in the rooms in which they worked. Of 104 men having less than 500 cubic feet of air to breathe, 13 had suffered from blood spitting and 13 others from catarrh; of 115 men having from 500 to 600 cubic feet of air, 5 had suffered from blood spitting, 4 from catarrh; of 101 men having more than 600 cubic feet of air, 4 had suffered from blood spitting and 2 from catarrh.

As for prisoners, there is the contrast between two prisons of Vienna cited by Dr. Parkes in his "Practical Hygiene." In the Leopoldstadt prison, which was very badly ventilated, there died in the years 1834–1847, 378 prisoners out of 4280, or 86 per 1000; of whom no fewer than 220, or 51.4 per 1000, died from phthisis. In the well-ventilated House of Correction in the same city there were from 1850 to 1854, 3037 prisoners, of whom 43 died, or 14 per 1000; and of these 24, or 7.9 per 1000, died of phthisis. Diet and mode of life were, it is believed, the same in both establishments. It is a flaw in the case that the average length of the periods during which the prisoners were detained in each prison is not given; but Dr. Parkes thinks that no correction on this ground, even if any correction were needed, could account for the discrepancy in the death rate. The great prevalence of phthisis among prisoners in general was long ago pointed out by Dr. Baly, as the result of an examination of the "Records of the Millbank Penitentiary." But he was unable to determine what part in the ætiology of the disease should be assigned to defective ventilation, and what part to cold, poorness of diet, want of active bodily exercise, and a listless or dejected state of mind.

Indeed, it obviously is only in very exceptional instances that overcrowding or defective ventilation can be so isolated from other conditions that may themselves be injurious to health, as to show clearly that they are the main cause of phthisis. But when once their power to produce the disease is admitted—and most persons will allow that there is *a priori* a strong probability in favor of it—one cannot doubt that they must play a most important part in producing the disease among the poor generally. For example, I shall have to refer presently to a very important investigation made by Dr. Greenhow in 1860 and in 1861 as to the origin of the great mortality from phthisis among the workpeople employed in many of the largest industrial occupations of the country, and to insist on the fact that the inhalation of dust of various kinds is the main cause of the disease so far as these persons are concerned. But Dr. Greenhow himself pointed out that another cause was certainly the "working in ill-ventilated or overheated factory rooms or workshops, as in those of some of the silk mills of Coventry, in the domestic weaving shops, and in the watchmakers' factories and workshops of the same city, in the button makers, and various other workshops of Birmingham, in the factory rooms of Blackburn and Nottingham, and in many domestic shops and warehouses." Abundant details in support of this conclusion are to be found in the report which he furnished to Mr. Simon.

β. *Food insufficient in quantity and bad in quality* is believed to be among the conditions which tend to produce phthisis, but I am not aware of any positive proof of this such as would be afforded by cases in which it could be shown to be the principal cause of the disease.

γ. In the female, *child-bearing* seems often to play an important part

among the causes of phthisis. On *a priori* grounds one would certainly think it likely that pregnancy should be apt to bring on the disease in persons naturally delicate or predisposed to it. But it is a well-known and a very remarkable fact that during gestation even the most rapid form of consumption scarcely ever runs on to a fatal issue. The patient almost always survives until labor is over, after which all the symptoms assume an increased urgency, and death may occur very rapidly. And in many cases the disease appears to begin rather during lactation than before delivery, or is attributed to the occurrence of profuse puerperal hemorrhage. According to Dr. Pollock it is not merely prolonged suckling that seems to set up phthisis, but the mere fact of suckling at all. Cases associated with child-bearing generally run a particularly acute course.*

δ. Alcoholic intemperance I believe to play an important part in the ætiology of phthisis. Clinical experience certainly shows that drunkards, both those who are engaged in the liquor traffic and those who are not, die in large numbers of this disease. No doubt it is often difficult to exclude the operation of other causes, such as defective food, bad ventilation, exposure to cold and wet, etc. But the impression produced on my own mind is that the alcohol itself is really concerned in the result.

It has of late been a favorite idea with some physicians that alcoholic phthisis is usually "fibroid" rather than tuberculous in character. I believe that Huss, of Stockholm, first suggested this opinion about 1850, and it has been supposed to derive support from the fact that alcohol causes a growth of interstitial fibrous tissue in the liver, and, perhaps, also in the kidneys and elsewhere throughout the body. But I do not know of any instance of the production by alcohol of a local fibroid change spreading slowly through an organ from one particular region of it. And, on the other hand, it is certain that tubercular peritonitis of the most typical kind is often seen in intemperate persons who also have hepatic cirrhosis. I have already expressed my disbelief in the existence of "fibroid phthisis" as an affection essentially distinct from the tubercular form of the disease (p. 87). I must now add that in the post-mortem room I have repeatedly had occasion to observe that the diagnosis of fibroid phthisis, which during the life of the patient had been partially based upon a history of alcoholic intemperance, has been utterly upset, the affection turning out to be most typically tuberculous.

ε. Diabetes is a frequent cause of a phthisis which is peculiarly pneumonic in character; its relations to the ordinary tuberculous disease are still doubtful. It may hereafter be shown that the diabetic lung affection is really not distinct from the more acute variety of tuberculous phthisis.

ζ. Syphilis is sometimes followed by the development of a destructive disease of the lung, which on post-mortem examination is found to be typically tuberculous. I have notes of no fewer than thirteen cases of this kind which occurred at Guy's Hospital between the years 1863 and 1873. But one must not forget that among syphilitic patients, especially in hospital practice, many are intemperate, destitute, and careless of their health in all ways. Consequently, it is quite possible after all that the relation between the lung affection and the syphilis may in many of the instances just referred to have been one of mere coincidence. On the other hand, we have in the production of lardaceous changes in the viscera an illustration of the fact that syphilis may set up a morbid process which is nevertheless capable of arising from other causes in persons who have not

* [These considerations are so important in regard to life insurance, that Sir Risdon Bennett and myself have for several years advised the office with which we are connected to count all deaths of mothers "in childhood" or "after delivery" as due to phthisis, unless there is explicit evidence of previous good health.—Ed.]

had syphilis. It therefore seems not unlikely that the disease, by depressing the general health, may constitute the starting point of a tuberculous lung affection. Is such a hypothesis incompatible with the clinical fact, which is attested by many observers of the highest authority, that the administration of iodide of potassium is often followed by most striking improvement in the physical signs and symptoms of what had appeared to be phthisis in a syphilitic subject, or even by their complete subsidence? I am not sure of it, because, as we shall hereafter see, any treatment which is capable of effecting a marked change for the better in the general health of a patient who has tubercular disease of the lung may also be followed by an apparent cure of the pulmonary affection. Still, it is obvious that a far simpler way of explaining the striking action of antisyphilitic remedies in the cases in question would be to recognize a syphilitic lung disease, capable of simulating phthisis, but pathologically distinct from it. My difficulty in adopting this view is that I have never been able to satisfy myself of the existence of such a disease. There is, of course, no reason why gummata should not develop themselves in the lung as well as in any other organ; but the recorded instances in which such lesions are stated to have been found in the post-mortem room seem to me to be generally open to more or less doubt. Take, for example, Dr. Goodhart's case, of which there is a colored plate in vol. xxx of the "*Pathological Transactions*." He himself mentions that Dr. Reginald Thompson considered the supposed gummata to be relics of pulmonary hemorrhage (*loc. cit.*, p. 236), and they certainly do not seem to me to differ at all from the encapsuled cheesy masses which are often found in ordinary cases of phthisis. Moreover, one cannot admit in the present state of pathology that the undoubted gray tubercles which also existed in large numbers in Dr. Goodhart's case were merely secondary results of the caseation of gummata, causing infection of the pulmonary tissue, although a few years ago such an interpretation of the facts would to most observers have appeared satisfactory. In vol. xxviii of the "*Pathological Transactions*" may be found a detailed discussion of the relations between syphilis and phthisis by Dr. Goodhart (pp. 313–329); and there are also cases in which the lungs appear to contain syphilitic lesions, recorded by Dr. Pye-Smith and Dr. Green (*ibid.*, pp. 334, 331).* But I fail to discover, either there or anywhere else, such examples of extensive lung disease, obviously and unmistakably different from tubercular phthisis, as certainly ought now and then to be met with if the views held by these observers were correct. Remembering how, for at least a quarter of a century, pathologists have vied with one another in the attempt to carve out of tubercular phthisis as many different forms of disease as possible, which they have supposed to be distinct from it, one is surely justified in scrutinizing all such statements very closely. Dr. Moxon when pathologist at Guy's Hospital was much impressed with the fact that in several syphilitic cases he found in the lungs indurated patches, the centres of which had sloughed away so as to form cavities in the interior of which shreddy masses were still hanging. And I think that he came to the conclusion that such appearances were almost characteristic of a syphilitic affection. But the difficulty is that they are altogether unlike those which syphilis is known to produce in other organs. For my own part I feel that the relations between this disease and phthisis still require to be investigated afresh by unbiassed observers.

III. The conditions which affect the lungs directly, and thus act as causes of phthisis, appear to be mainly the following.

a. Inhalation of Dust.—Ramazzini in his work, "De Morbis Artificum," published in 1703, seems to have been the first to point out that certain

* [See also cases in the same vol. by Drs. Greenfield (p. 258) and Gowers (p. 330).—Ed.]

classes of workmen are liable to have their lungs injuriously affected by dust given off by the materials which they employ. In the present century the question has been studied very thoroughly, especially in England and in Germany; and Zenker has proposed to term the pulmonary diseases due to this cause *Pneumonokoniosis* (κόνις = dust). The nature of the mischief set up in the respiratory organs by breathing dust varies widely in different cases. It may be a mere catarrh of the trachea and bronchial tubes, leading to emphysema, and tending to destroy life (if at all) by dilatation of the right heart and dropsy, as I have shown at p. 923. But in many instances it consists in local indurations of the lung substance, which may ultimately lead to the formation of cavities, and spread through the pulmonary tissue from apex to base, as in phthisis. Indeed, it has been usual to speak of "miners'," "weavers'," "knife grinders' consumption." Pathologists, however, have almost, without exception, maintained that the lesions found in such cases are independent of the presence of tubercles. Whether tubercle was regarded as a deposit from the blood in a special dyscrasia, or as a particular kind of new growth, it appeared to be equally necessary to exclude the possibility of its being due to a mere local tissue irritation. These views were, of course, still further strengthened by the wide adoption of Niemeyer's doctrines with regard to the non-tubercular nature of phthisis in general. And, still more recently, the theory of the local development of tubercles, as the result of tissue infection from lesions themselves inflammatory, seemed to remove all difficulties, by affording a ready explanation of the fact (which cannot be denied) that in many cases the more recently affected parts of the lungs show tubercles, when none can be discovered in the parts which were earliest diseased. This theory, however, is undoubtedly false, as I have shown at p. 92, *seq.* According to the conception of the tubercular process as a modification of inflammation, which I have endeavored to unfold in the chapter on tubercle, p. 84, there seems to be no absolute necessity for adopting the views of those who declare that all the Pneumonokonioses should be separated from phthisis. It may, perhaps, be thought that the reports of autopsies are conclusive in the matter, since they generally affirm that the appearances found in the lungs are altogether different from those which are seen in tubercular cases. But several points must be borne in mind. One is, that the character of the lesions in the lungs is often greatly obscured by the discoloration produced by the foreign particles deposited in the pulmonary tissue. Another is, that, as a rule, the destructive lung diseases due to dust inhalation occur at rather a late period of adult life, and advance slowly to a fatal termination; in both respects they resemble that form of ordinary phthisis which is regarded by so many pathologists as distinct from the tubercular and termed "fibroid." The remarks made at p. 920 with regard to the morbid anatomy of the so-called fibroid phthisis are, indeed, strictly applicable to the descriptions which are to be found in modern medical literature of the diseased lungs of miners, potters, or stone workers. The use of the word tubercle is often carefully avoided in such descriptions, but mention is almost invariably made of "nodules," from the size of a millet seed to a pea, scattered in the pulmonary tissue, of an opaque, grayish-yellow color, and, perhaps, with a small central cavity. The microscope is called into requisition for the purpose of proving the negative fact that the nodules in question fail to present whatever histological structure may happen to be regarded as characteristic of tubercle at the time when the observation is made. Or, possibly, it is asserted that they are distinguished by their hardness and resistance to pressure, the fact being overlooked that tubercles themselves, if they fail to caseate, undergo conversion into precisely similar bodies. I must, therefore, for my own part, decline to accept the validity of such

reports of autopsies until they are confirmed by some pathologist whose mind is open with regard to the general question of the unity of phthisis. Nor does it seem that the clinical course of the lung diseases due to dust inhalation differs essentially from that of the more chronic forms of phthisis, with which I believe them to be identical. Dr. Greenhow, who is one of the leading authorities on the subject, has insisted (in the Pathological Society's "*Transactions*," vol. xvi, p. 61, *et passim*) on "the coincidence of a cool skin and a quiet pulse with wheezy, asthmatic cough and copious muco-purulent expectoration" as rendering "the diagnosis from tubercular phthisis comparatively easy." And elsewhere he says that "shortness of breath almost invariably precedes by some considerable time the appearance of cough, and the patient is often ailing for many years before being disabled from work." But with regard to the suggestion that there is something distinctive in the early dyspnœa, it must be remembered that bronchitis and emphysema are as marked effects of the inhalation of dust as the distinctive changes in the pulmonary tissue with which alone we are now concerned; the only examples of phthisis in which we can expect to find a strictly parallel course are, therefore, those not very uncommon cases which supervene upon simple chronic bronchitis. Moreover, it is quite possible, as Seltmann has suggested in a valuable paper in the "*Deutsches Archiv*" for 1867, that when foreign matters are deposited in the lungs in very large quantities, the respiratory surface may be so much diminished that anæmia and dyspnœa are necessary consequences.

The materials which give off dust that may enter the respiratory organs are of various kinds. They may be classified as follows:

(1) *Carbon*, from coal, charcoal, soot, or smoke. The resulting affections of the lungs have been grouped together under the name of *Anthracosis*, a term originally proposed by Stratton in 1837. It is, however, only recently that pathologists in general have come fully to recognize their true relations. As far back as 1813, Pearson, in the "*Philosophical Transactions*," threw out the suggestion that the black discoloration of the lungs and of the bronchial glands, which is found in most adults, but not in children, consists of particles of carbon "introduced with the air in breathing," and originally "derived from the combustion of coal, wood, and other inflammable materials." Soon afterward a similar opinion was expressed by Laennec. Then in 1831 Dr. Gregory, of Edinburgh, recorded a case occurring in a man who had been employed in the coal mines of Dalkeith, in which both lungs were throughout of a uniform black carbonaceous color, and yielded, when washed, a black matter that was found by Dr. Christison to resist the action of concentrated nitric acid and of chlorine, and to yield by distillation products just like those which result from the distillation of coal. The conclusion seemed irresistible that the organs had been discolored by the penetration of coal dust into them from without. "Spurious melanosis," therefore, became generally adopted as the name for this condition of the lungs, which was understood to be only an extreme degree of the blackening that occurs more or less in all persons as they advance in years. But two great German pathologists, Hasse in 1841 and Virchow in 1847, each refused his assent to the proposition that the black matter was derived from the air inhaled in the act of breathing. It was not until 1860 that a case was recorded by Traube in the "*Deutsche Klinik*," which finally established the real existence of anthracosis. The patient was a man who for about twelve years had been engaged in loading and unloading wood charcoal, during which occupation he was constantly exposed to the dust of it. He had for a long time been accustomed to expectorate a black substance, and when he died his lungs were found to

be almost everywhere of a black color, yielding to pressure a black, frothy fluid which stained the fingers like thin Indian ink. Both in the sputa, and in the pulmonary tissue, there were found numerous pieces of the most irregular forms, with angular pointed processes. That these were fragments of wood charcoal was evident not only from a comparison of them with particles of charcoal brought directly from the place where the man had worked, but also from the fact that some of them showed the circular disks characteristic of the woody fibres of coniferous trees. And some had a reddish tint instead of being quite black; this was due to their being imperfectly carbonized. A second similar case occurred to the same observer six years later. Traube did not, indeed, himself at first conclude from these two cases that the finely granular material which gives a black color to healthy lungs as life advances is also inhaled carbon, but in the meantime the current of opinion began to set steadily in that direction. It was greatly helped on by Zenker's discovery (to which I shall presently allude more fully) that the lungs of those who work with red oxide of iron become full of red particles, occupying precisely the same positions as the black. In 1866, Virchow took an opportunity of recanting his former views, and Rindfleisch, in his "Handbook," ascribes to anthracosis almost every form of black discoloration of the pulmonary tissue. Modern histology has, indeed, removed one of the greatest obstacles in the way of the acceptance of such a doctrine, by showing that leucocytes are capable of taking particles of foreign materials into their substance, and that animal membranes are permeable by such particles in a way that formerly was hardly suspected. In 1858, in describing in the "*Edinburgh Medical Journal*" a specimen of "miner's lung" that had been sent to him from Scotland, Virchow had insisted that the seat of the most abundant deposition of black matter accorded ill with the theory of a progressive absorption from the air passages. Scarcely any of it, he said, was found in the interior of the pulmonary alveoli; and in their walls it lay not beneath the endothelium, but between the elastic fibres and the connective tissue. Moreover, very much larger amounts of it occupied the interlobular and peribronchial fibrous tracts, and accumulated beneath the pulmonary pleura. Nay, it was even present in the costal and diaphragmatic pleura, as well as in the bronchial glands. I may add that I once found some of it free in the upper and back part of the pericardial space, separated by the fibrous wall of the sac from an intensely black gland that lay just outside. Recently, however, this question has been studied experimentally by von Jus, whose observations are recorded in the "*Arch. f. exp. Pathologie*" for 1876. He injected cinnabar into the air passages of dogs, and found that the particles were rapidly taken up by cells which he believed to be altered leucocytes, so that five days later scarcely any pigment was left in the pulmonary alveoli; within six hours after the commencement of the experiment some of it reached the bronchial glands, being deposited first in their cortical layer, but ultimately reaching their medullary structure; a great deal of it, however, remained in the lungs, being accumulated in the connective tissue, between the lobules, round the vessels and the tubes, and beneath the pleura. In other words, von Just found that its distribution corresponded precisely with that which had been described by Virchow in the case of the miner's lung.

The phthisis associated with anthracosis is attended with one special symptom, which I must mention in this place, because I shall have no other opportunity. This is the "black spit" which is often ejected in considerable quantities for a length of time, even by miners who have entirely ceased to follow their occupation. There is no doubt whatever that it is often due to the gradual disintegration of the blackened and infiltrated parts of the lungs, or comes from vomicæ such as are sometimes found after death to be

full of a black liquid. Thus, Dr. Greenhow showed to the Pathological Society, in 1869, the lungs of a collier who, about ten days before his death, suddenly spat up a considerable quantity of matter closely resembling black paint, and continued doing so to the amount of four or five ounces daily until he died; in the right lung there was a large, irregular cavity, containing a quantity of black, pulpy *débris*. But the former of Traube's two cases shows that the sputa may contain much black matter without there being any lesion of the pulmonary tissue beyond the anthracosis; and he is, no doubt, right in insisting on the detection of fragments of elastic tissue as the only proof that ulceration is going on.

(2) *Oxide of Iron.*—In 1864, in discussing the subject of anthracosis, Friedreich asked the question why, if the black lungs and bronchial glands of coal miners were due to inhaled carbon, the workers in red sandstone quarries should not have the corresponding organs reddened by the dust to which they were exposed? Now, it happened that Zenker had at that very time in his possession the lungs of a woman who had, for seven years before her death, been engaged in making the little paper books in which gold leaf is laid. The paper has to be colored red with oxide of iron, and this is rubbed in by means of a piece of felt. The occupation is a very dusty one; and the woman's lungs were found, after her death, to be throughout of a bright, brick-red color, so that their cut surfaces looked just as if they had been daubed over with red paint. The microscope showed that fine granules of oxide of iron were present in all parts of their texture, beneath the pleura, in the interlobular fibrous septa, in the peribronchial sheaths, in the walls of the alveoli, and even in cells occupying their interior. In the twentieth volume of the "*Pathological Transactions*" there is a colored drawing of a thin section of this lung, taken from a specimen in the possession of Dr. Wilson Fox; the tint is, however, far less red and more brown than that represented in Zenker's drawings published in the "*Deutsches Archiv*" two years before. Zenker proposed to name the affection Siderosis (σίδηρος = iron). In 1874, Merkel, in Ziemssen's "Handbuch," was able to refer to seven other cases, one of which occurred in a man who had used the red oxide of iron for polishing glass. He had also met with two instances in which the lungs were blackened by the black oxide of iron, and one in which they owed a black color to the phosphate of iron. There was no difficulty in detecting the presence of iron in the sputa during life, by the action of hydrochloric acid and ferrocyanide of potassium.

(3) *Quartz, Sandstone, or Clay.*—That workmen whose occupations expose them to the dust of siliceous or argillaceous materials are very prone to die of phthisis has long been known. Very full information with regard to the excessive mortality from this cause in certain districts of England is contained in a paper by Dr. Greenhow in Mr. Simon's third "Report to the Privy Council," published in 1861. Merkel has recently proposed to term the resulting lung affection *Chalicosis* [χάλιξ = gravel]. The presence of silica in the pulmonary tissue seems to have been first detected chemically by Dr. Peacock and by Dr. Greenhow. Kussmaul, however, has recently shown (in vol. ii of the "*Deutsches Archiv*") that this substance is present in greater or less quantity in the lungs of all persons (though not in those of a fœtus), having doubtless been derived from the dust of the streets and roads blown up by the wind. In a railway-signal man, stationed in a very sandy district, Meinel found that silica actually formed as much as 18.2 per cent. of the ash of the lungs after incineration; even in a stone mason, who died of phthisis, and whose lungs were analyzed by Kussmaul, the amount was not greater than 24.7 per cent. of the ash. Under the microscope the particles of silica may be seen as bright bodies of round or angular shape. Among the classes of workmen who suffer from this cause are, of course, stone-

masons and millstone makers. Still more fatal is the grinding and polishing of steel instruments—from scythes to needles—such as is carried on in Sheffield, Birmingham and elsewhere. The trades, as is well known, are subdivided to the greatest possible extent; but whether the exact nature of the work be needle pointing or fork grinding, or the sharpening of fish hooks, the result differs but little; a large number of the men die prematurely, some between twenty and thirty, and more between thirty and forty; very few survive the age of forty without suffering more or less from pulmonary symptoms. A point of some importance is that what is termed "dry grinding" is much more injurious than "wet grinding;" the difference is that in wet grinding the wheel, as it revolves, dips into water and deposits a large part of the dust which would otherwise be carried into the air. But even wet grinders are greatly exposed to dust in "hacking" their grindstones, which generally has to be done every day or even oftener. Another dangerous occupation is pearl-shell cutting. Potters, again, are exceedingly apt to be attacked with phthisis; "flat pressers" suffer more than "hollow pressers;" but the worst off of all are "china scourers," whose business it is to rub off the loose flint powder from the china with sandpaper, after it has been baked.

(4) *Cotton and Flax.*—Among occupations which expose the persons engaged in them to injury from the inhalation of vegetable matters may be especially mentioned the carding of cotton, and the hackling and carding of flax. I do not know that fragments of these materials have ever been detected in the pulmonary tissue after death. But it is an interesting fact that in two men who had been engaged in the manufacture of tobacco, Zenker is said to have found "brown spots in the lungs and in the bronchial glands, evidently due to the deposition of powdered tobacco."

General Question of Ætiology.—It is clear, then, that foreign particles of various kinds may find their way into the lungs, may be deposited in the pulmonary tissue, and may either remain there or be ultimately transported to the bronchial glands. But it by no means follows, as a matter of course, that their presence must be injurious. Indeed, since the true nature of the black material found in the lungs, whether in health or in disease, has of late years become established, increasing doubts have been expressed as to whether it can fairly be regarded as the cause of inflammatory or destructive changes. In Traube's first case, referred to at p. 998, there was not the slightest trace of any newly-formed connective tissue nor of induration of the substance of the lungs; the patient's symptoms were probably due to chronic pericarditis, accompanied by double pleurisy, which seemed to have begun three months and a half before his admission into the hospital and four months and a half before his death. It is true that this instance does not prove that in other persons irritation of the pulmonary tissue cannot be set up even by wood charcoal, still less that such a result may not be caused by particles of coal or of lampblack, impregnated as they may be with various chemical products. But the experience of German observers seems to be strongly adverse to such a conclusion. Seltmann, of Zankeroda, near Dresden, asserts positively in vol. ii of the "*Deutsches Archiv*," that among the coal miners of that district there is generally no overgrowth of connective tissue whatever, even in lungs full of black deposits, and that the formation of cavities is of very rare occurrence. As for pulmonary tuberculosis, he thinks that the inhalation of carbon is actually antagonistic to its development—an opinion which appears to be shared also by Merkel.

If, therefore, English miners are greatly more liable to destructive disease of the lungs, the explanation is, in all probability, that the galleries in which they work are so often badly ventilated; the real cause of their being

attacked with phthisis is not that the air which they breathe contains coal dust or smoke, but that it is rendered impure by the products of respiration, exactly as in crowded workshops or sleeping rooms. Indeed, in his fourth report, Mr. Simon drew special attention to the fact that the colliery miners of Durham and Northumberland differ from all other miners in not suffering any important excess of pulmonary or other disease; and that the reason for this is the good ventilation of the mines in which they work; but he was still disposed to think that this operated mainly by removing the coal dust and powder smoke which would otherwise be diffused through the air. The great heat to which miners are exposed is in all probability another important factor in the ætiology of the lung diseases to which they are liable.

On the other hand, there seems to be no doubt that destruction of the pulmonary tissue is an almost necessary result of the entrance of some other kinds of foreign particles into the lungs. Merkel says that in his cases of siderosis fibrotic changes were never wanting even where the patient had died of some independent disease. And among needle grinders, potters, and other classes of workmen exposed to the inhalation of flint dust or finely powdered clay, the prevalence of phthisis is far too great to be accounted for in any other way. Merkel says that the only opportunity which he ever had of examining a grinder's lung was in the case of a boy, aged sixteen, who was killed accidentally after having been at his occupation four and a half years, and who was said to have been in perfectly good health. His lungs already contained small, tough, black nodules of the size of a pin's head, as well as minute particles of sandstone and of iron.

Whatever may be the nature of the irritant which sets up destructive changes in the lungs, the resulting affection appears to have exactly the same characters. This was strongly insisted on by Dr. Greenhow when, in 1865–1870, he successively showed at the Pathological Society's meetings the lungs of a collier, a copper miner, a razor grinder, a stone worker, a potter, a flax dresser, and a pearl-shell cutter. And it is to be observed that the lungs may be almost, if not quite, as black in those patients in whom the affection was set up by sand or clay as in the miners themselves. The explanation is doubtless that there has been bronchitis also, and that this, by interfering with the natural ciliary action of the bronchial mucous membrane, causes the particles of carbon in dust and smoke, which in greater or less amount are inhaled by every one, to become deposited in the pulmonary tissue in excessive quantity instead of being mostly swept back into the trachea. But, further, when any destructive process is set up in a lung, the affected parts are very apt to become more deeply blackened than the rest of the organ. One of the points on which Virchow insisted, when he upheld the view that the discoloration was due to a pigment derived from altered hæmatin, was that even in children the development of phthisis was sometimes attended with a blackening of the tissue which at their age could only be regarded as altogether exceptional. The experiments of von Jus, referred to at p. 999, enable us, I think, to understand why the foreign material should accumulate in newly-formed fibroid tissue, and even in fibroid tubercles of a diseased lung, just as it does in the connective-tissue tracts of the healthy organ rather than in its alveoli.

The complete identity of the pulmonary affections to which so many different classes of operatives are liable, is in itself sufficient to show that a common pathological process is concerned in producing them. And if I am right in maintaining that the phthisis of colliers is but the result of general anti-hygienic conditions, the whole question is brought at once under that of chronic or fibroid phthisis in general, which has been fully discussed already. But there is one other argument, upon which I must especially insist, because I think it has generally been completely ignored by those

who, like Dr. Greenhow, have asserted that the lung diseases of those who work in factories or in mines are often merely due to a chronic interstitial pneumonia, and should be sharply distinguished from those in which tuberculosis plays an essential part. It is that since the irritants which excite these several diseases act upon all parts of the lungs alike, the resulting lesions must necessarily be diffused uniformly over all parts of them unless some other factor is involved in their ætiology besides the mere irritation. In reality they attack one lung before the other in most cases, and they almost invariably begin in the upper lobe, and spread gradually downward through the organ toward its base. I have repeatedly had occasion to point out that such a course is almost constantly taken by all tubercular affections of the lungs, indications of it being apparent even in many examples of miliary tuberculosis.

β. Cold and Wet.—No idea is more firmly rooted in the public mind than that consumption is often the result of accidental causes, such as getting chilled by remaining in wet clothes or by exposure to a draught when heated in dancing, and generally that there is danger, especially in those who are hereditarily predisposed to the disease, lest it should supervene upon a cold, or a succession of colds, if due care be not taken. But the weight of medical opinion has in our day tended strongly toward the rejection of all such notions. The only statistical facts that I know of which are in favor of the popular view are those given by Dr. Theodore Williams in vol. liv of the "*Med.-Chir. Transactions.*" Out of 1000 cases of phthisis he found that no fewer than "140 had originated in, or been closely preceded by, pleurisy and pleuro-pneumonia, and 118 by bronchitis:" but it does not appear probable that this tabulation can have generally rested on any higher foundation than the unsupported assertions of the patients themselves, who doubtless consulted him or his father, Dr. C. J. B. Williams, at variable periods after the commencement of their illness, and often when a considerable time had elapsed.

The considerations which led the older pathologists to reject the idea that phthisis could arise out of a common catarrh were, I think, in the main identical with those which induced them to regard as necessarily non-tubercular the cases due to dust inhalation. And such theoretical opinions were indefinitely strengthened by the practical observation that many persons, even of delicate aspect, suffer for years from an extreme liability to bronchial attacks without ever becoming consumptive. Indeed, Rokitansky actually declared that pulmonary emphysema and dilatation of the bronchial tubes, if carried to a sufficient extent to cause venosity of the blood and cyanosis, afford exemption from the liability to pulmonary tuberculosis. But I have certainly met with several striking exceptions to such a rule. In 1864, for example, a girl of seventeen was admitted into Guy's Hospital with extreme dyspnœa and dropsy, and with clubbing of the fingers and toes. The bronchial tubes were found widened out into great sinuous passages, so that the cut surfaces of the lungs showed hollow spaces as extensive as the remains of the pulmonary tissue. Yet there were scattered yellow tubercles, especially in the left lung, spreading from the apex downward. In 1874, a woman, aged thirty, came in, who said that she had long been more or less subject to cough, which for nine months past had become continuous. There was extreme emphysema of the bases and anterior parts of the lungs, and the tubes contained a large quantity of pus; but both lungs also showed scattered gray tubercles and patches of translucent gray consolidation, with points of caseation breaking down here and there into cavities. I should have no difficulty in citing a good many other similar cases from my note books were it worth while. Another fact which indicates that catarrh is apt to lead to the development of phthisis is the

frequency with which, in children, pulmonary tuberculosis follows whooping-cough or measles. There is, of course, the difficulty that catarrh, so far as is known, spreads from the larger tubes into the smaller in all directions, or perhaps toward the bases of the lungs in preference. We certainly have no proof whatever of the possible occurrence of what is so often spoken of—a catarrh localized at one or both of the apices; and I entirely agree with those who think that the adoption of such an expression is meaningless, except for the purpose of covering a doubt on the part of the physician as to whether there is anything the matter with the lung, or for that of soothing the mind of the patient and inducing him to submit to treatment without being dismayed. But on the view which I am endeavoring to inculcate with regard to tuberculosis—that it is a modification of the inflammatory process which is especially apt to arise at the apices of the lungs from a morbid tendency inherent in those parts of them—I see nothing improbable in the supposition that irritation of the whole of the pulmonary tissue by cold may set up phthisis there, while failing altogether to do any damage elsewhere. I have met with more than one instance in which the disease has appeared to be distinctly traceable to residence in a damp, newly-built house, the patient having been quite well up to the time of the change of abode.

It is true that caution is required in accepting the statements of patients themselves with regard to the origin of their illness. In 1869, a young man died of phthisis in Guy's Hospital, who attributed the disease to his having slept with his window open one night five weeks before. He admitted, however, that nine months previously he had been ill for a week with a cough. At the autopsy, besides a very acute phthisis of pneumonic character, there were found at the left apex old clustered gray and black tubercles and cavities. This, therefore, could not be cited as a case caused by exposure to cold.

Soil.—It is, I think, in no other way than by the increased liability to attacks of slight bronchial and pulmonary catarrh, which must necessarily follow residence in damp situations, that we can account for certain remarkable observations which have recently been made with regard to the influence of *soil* upon the frequency of phthisis. In 1862, Dr. Bowditch, of Boston, took occasion, in addressing the Massachusetts Medical Society, to bring forward a mass of evidence which led him to believe that, in that State, consumption, instead of being equally diffused through all parts of it, prevails especially in such places as are situated upon a damp soil, and seldom occurs when the soil is dry. This evidence consisted chiefly of an analysis of the replies of medical men living in 183 townships to inquiries as to the frequency of the disease in their practice, and as to the moisture or dryness of the localities. It also includes some striking instances in which phthisis had carried off in succession a number of persons living in certain houses surrounded by wet meadows, or placed by the side of a mill pond, or shut in by luxuriant trees.

Far more conclusive, because resting upon an accurate statistical basis, is a body of facts which were collected by Dr. Buchanan during the years 1865 and 1867 in England, and published in Mr. Simon's ninth and tenth reports to the Privy Council. The inquiry began in a tour of inspection, made for the purpose of ascertaining the results of sanitary works that had been carried out in twenty-five towns, containing an aggregate population of 606,186 persons. It was found that in several places there had been a great diminution in the general death rate, and that the prevalence of enteric fever had become much less, especially where a good had been substituted for a bad water supply, and where drainage works had taken the place of cesspools or middens. But in other towns it was by a decrease in the number of cases of phthisis that the good effects of sanitary improvements appeared to be manifested; and the particular change which coincided

with this result was found to have been a drying of the ground by drainage of the subsoil. The following table shows the amount of change in the phthisis death rate in twenty-four of the towns visited by Dr. Buchanan :—

Town.	Previous death rate per 10,000 from phthisis.	Degree of change in death rate from phthisis.		Influence of sewage works on subsoil.
		In total population.	In females between 15 and 55.	
Salisbury	44⅓	—49 p.c.	?	Much drying.
Ely	32	—47 p.c.	?	Much drying.
Rugby	28½	—43 p.c.	—48 p.c.	Some drying.
Banbury	26⅔	—41 p.c.	—36 p.c.	Much drying.
Worthing	30½	—36 p.c.	—41 p.c.	Some drying.
Macclesfield	51½	—31 p.c.	—22 p.c.	Much drying.
Leicester	43⅓	—32 p.c.	—16 p.c.	Drying.
Newport	37	—32 p.c.	—13 p.c.	Local drying.
Cheltenham	28¾	—26 p.c.	—25 p.c.	Some drying.
Bristol	33½	—22 p.c.	—18 p.c.	Some drying.
Dover	26⅓	—20 p.c.	—18 p.c.	Local drying.
Warwick	40	—19 p.c.	—10 p.c.	Some drying.
Croydon	*	—17 p.c.	?	Much drying.
Cardiff	34¾	—17 p.c.	?	Much drying.
Merthyr	38½	—11 p.c.	—12 p.c.	Some recent drying.
Stratford	26⅔	— 1 p.c.	— 4 p.c.	Some local drying.
Penzance	30¾	— 5 p.c.	0	No change.
Brynmawr	28⅓	+ 6 p.c.	— 8 p.c.	No notable change.
Morpeth	30½	— 8 p.c.	+12 p.c.	No change.
Chelmsford	32⅓	0	+11 p.c.	Slight drying.
Penrith	39⅓	— 5 p.c.	+27 p.c.	No change.
Ashby	25½	+19 p.c.	+10 p.c.	Some drying.
Carlisle	32	+10 p.c.	+11 p.c.	Drying (with local defects).
Alnwick	28⅓	+20 p.c.	+36 p.c.	No drying.

It is, perhaps, worth while to give some details as to one or two of these towns, as the full significance of the change that has been effected in them by drainage works can hardly be appreciated otherwise.

In 1851, Mr. Rammeli had reported of Salisbury as follows: "Numerous streams of water, supplied by the Avon, run through most of the streets. . . The soil is a porous gravel, containing everywhere a great deal of water, which rises to within a short distance of the surface. There have been several instances of the cathedral being flooded by the water of the subsoil. The foundations of the houses are almost without exception damp." . . "The drainage system of the principal part of the town is formed by the open channels which run along the footpath at the side of the street, and the house drains open into them from both sides. The fall of these drains is necessarily very small, they are frequently in their whole length below the level of the water in the open channel, and consequently, so far from being able to eject their contents into the common channel, they are in such cases generally filled with water from the channel." The water supply is from wells "dug about eight or ten feet deep, the water rising to within three or four feet of the surface." In 1853 sanitary works were commenced in Salisbury, and they were completed in 1855. The drainage consists of brick mains, and of glazed earthenware pipes opening into them. The mains have floors of hollow stoneware pieces laid without cement, and at the sides of the pipes are placed drain tiles. These arrangements are for the purpose of carrying off the subsoil water, of which there is constantly a rapid flow

* Phthisis and other lung diseases together were previously 59½ p. c. Reduction of this rate is what is above given for Croydon. (Ninth Report, 1856, p. 48.)

through the mains. In 1865 Dr. Buchanan writes as follows: "The subsoil is now dry, and cellars of considerable depth can now be made in different parts of the town which do not become flooded at any time. On an average the subsoil water has been lowered four or five feet all over the city. The cathedral has never been flooded since the drainage works." As is shown in the table, the annual death rate from phthisis fell in Salisbury from 44⅓ per 10,000 in 1844–1852 to 22⅔ per 10,000 in 1857–1864.

Of another town, Banbury, Mr. Rammell had reported in 1850 in the following terms: "The drains are not all at a sufficient depth to drain the cellars of the houses. In the principal streets of the town water is raised from the cellars into the drain by buckets, and creates a nuisance." Sanitary operations were begun there in 1854. The sewerage was done on a uniform system. Most of the sewers have a good fall, and are laid seven to ten feet below the surface. "At present," says Dr. Buchanan, writing in 1865, "the sewers and drains all act efficiently. . . Many of the wells of the town have been dried by the sewers. . . The sewage discharged into the river after 10 P. M. is little more than water from the springs." As appears from the table, the phthisis death rate for 10,000 has declined from 26⅔ in 1845–1853 to 15⅘ in 1857–1864.

It must, of course, be understood that drying of the subsoil is not the only improvement that has been made in these towns. Excreta have at the same time been carried off from the houses, a good water supply has often been provided, and overcrowding has been diminished. Now, as regards filth removal, it does not seem that this has acted to reduce the phthisis death rate. Dr. Buchanan placed the several towns in another list, according to amount of decrease in the mortality from enteric fever, and the order in which they stand in the phthisis list is by no means the same as that in the fever list, which appears to be more affected by removal of filth than by anything else. Many of the towns at the bottom of the phthisis list, such as Alnwick and Brynmawr, have made very good arrangements for the carrying away of excreta. And, on the same hand, Worthing and Rugby, both of which stand high in the phthisis list, are very low in the fever list. The cases in which sanitary works have failed to reduce the death rate from consumption are chiefly those in which the soil previously contained little water (as at Penzance and Brynmawr), and those in which the deep drainage was effected by impervious pipes laid down in compact channels (as at Penrith and Alnwick) so that no extensive drainage would occur, either through or at the side of them.

The importance of these observations appeared to be so great that, in 1867, Dr. Buchanan was directed by the Privy Council to make a special investigation in the three southeastern counties, Surrey, Kent, and Sussex, for the purpose of determining whether any relation could be traced between the prevalence of consumption and the state of the soil as regards moisture. These three counties were chosen because they were the only ones of which the Geological Survey had then minutely mapped out the surface geology; but no other part of England could have been better adapted to the purpose on account of the great varieties of soil which are to be found there, and of the comparative absence of differences other than of soils between the several districts.

The first point was to ascertain the extent to which phthisis prevailed in different parts of the three counties. The basis for this part of the inquiry was afforded by the Registrar General's Returns. Of course, they cannot pretend to exact pathological accuracy. But, seeing that in each registration district the certificates are furnished by several medical men, it is not likely that any serious error can result from their being used for the purpose of comparing the death rate from so common a disease as consumption in

one district with that in another. Moreover, if cases of phthisis are wrongly returned under any other head, it must be generally under that of bronchitis or of some other lung disease. Dr. Buchanan, therefore, took pains to consider the mortality from lung diseases in general, as well as that from phthisis, before he drew his conclusions.

Corrections had to be made in the returns for certain districts on account of the presence of camps or dockyards, causing the population to have a large excess of males in the prime of life; in others the returns were vitiated by their containing hospitals or asylums; in others, again, by their being resorts for invalid visitors; eight districts were set aside as being subject to disturbing influences which rendered their true phthisis death rate uncertain. There were left fifty other districts which it was believed could be fairly compared with one another.

In instituting this comparison, Dr. Buchanan first classified the several districts as having mainly soils *permeable* by moisture, or soils of such a character that water is unable to escape from them, so that they might be called *retentive*. He then massed together the fifty districts into five groups of ten each, according to the greater or less prevalence of phthisis in them, and in this way he obtained the following table :—

Groups of Districts.	Proportion of population (per 1000) residing on	
	1. Pervious Soils.	2. Retentive Soils.
A. With least phthisis	909	91
B. With next least phthisis	877	123
C. Middle as to phthisis	795	205
D. With more phthisis	792	208
E. With most phthisis	642	358

This tabulation, however, is not only open to arithmetical objections, but is also vitiated by certain geological considerations. It is obvious that where the soil is pervious, its being moist or dry must depend entirely upon whether the water which reaches it and sinks into it can escape through it or from beneath it. It is no advantage for a place to be situated on gravel if the subsoil water cannot get away. Roughly one may say that such a district will be *dry* in proportion as it *lies high* in relation to the places round it, *damp* in proportion as it *lies low*. On the other hand, among impervious soils, the question of dryness or moisture is almost entirely one of the *inclination* of the *surface*. Even among clays there is a great difference as regards dampness, according to the *flat* or *sloping* character of the ground.

A more exact comparison between retentive and pervious soils in regard to the prevalence of phthisis is afforded by a limited area, the Wealden, which in part is formed by the Weald clay, in part by the Hastings beds of alternate sands and clays. There are, indeed, no districts wholly of sand to contrast with others wholly of clay; but there are great differences in the proportion of the two soils in different districts. How closely these differences correspond with differences in the phthisis death rate appears from the following table.

The districts are arranged in order of phthisis death rate, those being placed highest in which the death rate is least. Where there are gravels over the Weald clay the figure is divided between the last two columns, it being presumed that they occupy an intermediate position.

District (in order of phthisis death rate).	Percentage of population resident on						Total on	
	Higher beds, mostly greensand		Weald clays.		Hastings beds.		Sands and half gravels over Weald clay.	Clays and half gravels over Weald clay.
	Sands of.	Clays of.	With gravel.	Without gravel.	Sands of.	Clays of.		
Hastings	—	—	—	—	95	5	95	5
Cranbrook	—	—	1	6	84	9	84	16
East Grinstead	—	—	—	12	82	6	82	18
Tunbridge	—	1	24	7	64	4	76	24
Uckfield	—	—	—	1	82	17	82	18
Hambledon	49	—	20	31	—	—	59	41
Battle	—	—	—	—	80	20	80	20
Rye	—	4	—	—	79	17	79	21
Maidstone	43	1	45	11	—	—	66	24
Cuckfield	21	1	—	25	48	5	69	31
Hailsham	—	—	—	34	61	4	61	38
Ticehurst	—	—	—	—	67	33	67	33
Tenterden	—	—	—	29	42	29	42	58
Horsham	—	—	—	56	44	—	44	56
Petworth	30	—	—	70	—	—	30	70

Still more striking, perhaps, are certain comparisons which can be made between particular sets of districts which differ in the manner suggested in the last paragraph but one; if *pervious*, in being *high lying* or *low lying* respectively; if impervious, in being *sloping* or *flat*.

1. As between *high-lying* and *low-lying pervious* soils, a contrast is afforded by districts formed by the chalk. No soil is drier than chalk when it has a fair elevation; at its higher parts there are no streams, water cannot be reached by ordinary wells, and the people can only obtain a water supply from less elevated ground. In many districts, however, the bulk of the population who live on chalk occupy valleys with the water line in the chalk not very far below their houses; and in the south of Sussex a still greater degree of wetness is reached, for a large part of this area reckoned as chalk is a flat plain on the sea level, covered by gravel, with the dip of the chalk here and there inland. Accordingly, we find the phthisis death rate for North Aylesford and Dover (both of which lie high) to be 289 and 296 respectively, while those for Worthing, Lewes, and Westbourne (all of which lie low) are 419, 426, and 498. In general, Dr. Buchanan adds, the connection between a low death rate from phthisis and elevation of the chalk area is not to be mistaken.

So, again, with regard to the population living mainly on the Lower Greensand, there is a great contrast between a southern tract of this formation which lies low and the hills which are made up of it elsewhere. And a corresponding difference exists between the phthisis death rates of Thakeham, Midhurst, and West Ashford (which are 454, 455, and 421 respectively) and those of Reigate and Godstone (337 and 282).

2. As between *sloping* and *flat* impervious soils, a capital contrast is presented by two widely distributed tracts of clay, the London clay and the Weald clay. The former in its main extent throughout the three counties is disposed in long slopes or hills, the latter constitutes sometimes gently undulating, but more often flat, level ground. The former over large areas is covered by gravel reaching to many feet in thickness; the latter has only very level gravels, which are rarely at all thick, occupying its undulations.

The former generally has the direction of drainage from other beds away from it; the latter is always bounded immediately to the north and to the south by higher ground, so that other beds drain more or less into it. All these considerations show that the London clay is commonly much less wet than the Weald clay. The difference between the two formations in respect of their phthisis death rate is, Dr. Buchanan says, unmistakable. All districts that have even a third of their population on Weald clay have a high mortality from consumption, whereas there may be in a district a notable proportion of uncovered London clay without any like result.

I have gone into so much detail in regard to Dr. Buchanan's inquiries, not only because of the necessity that the evidence in support of such grave conclusions should be thoroughly weighed, but also because of the practical importance of the various points which are brought out in his paper. But I must not omit to mention that Dr. Kelly, the Medical Officer of Health for East Sussex, has more recently expressed doubts as to the existence of any intimate relation between dampness of soil and phthisis. He finds that in the years 1861–1870 the order in which the several districts have to be placed in regard to their death rates from phthisis is very different from that given by Dr. Buchanan for 1851–1860. He points out that most of the impervious beds are to the north of the South Downs, and that consumption seems most common in places which are bleak and exposed as well as damp. And in general he insists on the fact that in West Sussex (and, indeed, throughout England and Wales) there has of late years been a great decrease in the mortality from consumption. As there has been no change whatever in the drainage, this certainly throws a doubt on the validity of Dr. Buchanan's earlier inquiry into the effects of sanitary improvements. Dr. Kelly is inclined to attribute it mainly to the progress which has taken place in the social state of the rural population.

Direct Injury.—Whether this can be enumerated among the causes of phthisis is, perhaps, doubtful. In 1880, I examined the body of a man, aged thirty-eight, who had been a patient of Dr. Moxon's. He had been a cab driver, but was said to have been very moderate in his habits; there was no history of consumption in his family. Four weeks before his admission his cab had been upset and turned over upon him, the step coming upon his chest. Afterward he had coughed and spat a little blood. Three days later he had brought up half a pint of blood. His illness was regarded as the direct result of the accident, but the physical signs were exactly like those of phthisis affecting the left lung, and spreading through it from above downward. When he had been in the hospital two months and a half he died. I found that the left lung was universally destroyed by a phthisical affection, of "pneumonic" character. The upper lobe and the upper part of the lower lobe were hollowed out into a number of cavities; the rest of the lower lobe was consolidated by a pinkish-gray infiltration, scattered in which were many yellow tubercles and caseating patches with sinuous edges. But the right lung was affected with a more chronic form of the disease; in the upper lobe was scattered much pigmented gray tubercle; there were also some caseous tubercles, and on each side of the lobar septum there were one or two small vomicæ. The tubercular nature of the disease was confirmed by the fact that the small intestine contained ulcers which, although circular, had thickened edges and showed yellow submucous tubercles in their floors. In the larynx, too, there was a deep ulcer over the left arytenoid cartilage. Evidently the relation of the accident to the phthisis can only have been that it started with fresh activity a morbid process in the lungs which was already in existence before; and one may fairly question whether this was not rather the result of the general shock than of the

local injuries which the man received. I have notes of four other cases in each of which phthisis followed an accident; in one there was fracture of ribs, in another fracture of the collar bone, in the other two injuries of an undetermined kind from a fall into the hold of a ship, and from a railway accident respectively. But in none of these cases is there any proof that the lungs were previously healthy; nor, indeed, is it certain that there was any closer connection between the accidents and the pulmonary disease than mere coincidence. Still it accords with what I believe to be the true ætiology of some other tubercular affections, as in the kidney and in joints, to admit that an injury to the chest may sometimes set up phthisis.

Opposing Pathological Conditions.—To complete our survey of the ætiological relations of phthisis, it is necessary that brief allusion should be made to certain conditions of body which have been, or still are supposed to be, antagonistic to its development.

One of these is habitual exposure to *malaria*. This opinion, however, is now generally discredited. Nor does it seem that there is any obstacle to the simultaneous occurrence of *carcinoma* and of tuberculosis, beyond the facts that each of these diseases proves somewhat quickly fatal, and that one of them attacks chiefly persons who are old, the other those who are comparatively young. I have notes of nine or ten cases which occurred at Guy's Hospital between 1855 and 1874, and in which a more or less active phthisis has been present in patients who have also had cancer of the stomach, or womb, or œsophagus, or some other organ. One was a woman, aged twenty-two; another a man, aged twenty-four; a third a man, aged thirty; the rest were older, one having reached the age of sixty-seven.

On the other hand, it appears to be indisputable that at least one kind of valvular *disease of the heart* is an almost complete bar to the development of phthisis. Mitral stenosis is exceedingly common in young persons, and it often fails for several years to affect the general health to any marked extent. That this lesion should be scarcely ever found in those who die of consumption is, therefore, a very remarkable fact. Traube says that he does not remember to have met with such an instance. In our records of post-mortem examinations at Guy's Hospital, from 1854 onward, I have noticed only four examples of it. One was in a man, aged forty-two, whose mitral orifice was so far narrowed, as the result of rheumatism, that it would only admit two fingers. Another was in a man, aged thirty-one. On the other hand, Traube speaks of having seen several cases in which persons with regurgitant disease of the aortic valves became affected with consumption. Whatever may be the explanation of the rarity of phthisis in those who have mitral stenosis, it can hardly depend upon the venosity of their blood, as Rokitansky formerly thought. For it is now well known that those who have congenital narrowing of the pulmonary orifice are exceedingly apt to die of tubercular disease of the lungs. Traube says that he has seen two examples of this; and two at least have occurred at Guy's Hospital within my knowledge. Traube's view is that mitral stenosis, by causing liquor sanguinis to exude into the pulmonary tissue, prevents the occurrence of caseation; and Dr. Hamilton, in the "*Practitioner*" for 1880, throws out a similar suggestion. But such an interpretation of the facts involves the assumption that phthisis begins as a modification of catarrhal pneumonia, which I am not at all prepared to allow.

Age and Sex.—Phthisis may occur at all periods of life. The idea that it is especially a disease of young adults is not borne out by statistics. At Guy's Hospital I find that up to the age of forty-five there has been little, if any, diminution in the number of fatal cases for each quinquennial period. I have notes of several cases in persons between sixty and seventy; and of

two cases at the age of seventy-two. More men than women appear to die of consumption in the hospitals of London; but Dr. Farr puts the rates of males to females in the population generally as 3.77 to 4.13. It must not, I think, be supposed that age and sex constitute predisposing causes of the disease, in a strict sense of that term. The question may be of the more frequent operation, at different periods of life, and in one sex rather than the other, of the various causes that have already been enumerated.

Prophylaxis.—It would, of course, be far simpler, if we could attribute phthisis to one single cause, instead of looking upon it as the result, in most cases, of the joint operation of several distinct agencies. But I cannot doubt that the latter view is the true one. And it certainly has this advantage, that it opens to the physician a very wide field of usefulness, in preventing the development of this terrible disease. Even the hereditary transmission of consumption is not altogether beyond his scope as regards prophylaxis. He can often do a great deal to prevent the intermarriage of cousins belonging to a phthisical family, and he can exert his influence to induce the members of such a family to select partners belonging to a healthy stock, even if they do not altogether abstain from propagating the species. The latter course seems to me to be seldom, if ever, incumbent on those who are not themselves actually consumptive, provided that they are able to bring up their offspring under conditions favorable to health. If a mother is known to be already phthisical, or even delicate, it is a very grave question whether she should be allowed to nurse her child. Care should be taken that the residence of those who have tubercular tendencies—and indeed, of all persons, however robust, so far as one can influence them—should be on a dry soil. The rooms in which they live or sleep, or learn lessons, or work, should be airy, well ventilated, and so situated as to be exposed to sunlight. Their food should be wholesome and nutritious. They should have plenty of exercise in the open air, and they should be accustomed to exposure to the weather, but within reasonable limits, and only so that they do not become chilled. Cold bathing is advisable, provided that there is always a good reaction after the bath. Special care should be taken during convalescence from whooping-cough and measles; and the recurrence of attacks of bronchial catarrh should be sedulously avoided. Outdoor sports, gymnastics, the use of the dumb bells are probably all good, so long as they are not carried to excess. Still more important is it that study, whether in preparing for examinations, or in the pursuit of literary or professional eminence, should be kept within due bounds. Indeed, as adult life is approached, the necessity for moderation in all things should be impressed on every one who would avoid the risk of phthisis. Temptations to intemperance and to dissipation must be strenuously resisted. If an occupation is to be chosen it should be such as is favorable to health. The son of a phthisical miner, or potter, or weaver, should, even more than others, avoid such dangerous kinds of work. If a youth is to be a clerk, he should, if possible, be put where the hours are comparatively short, and where the duties are of a varied character, and need not be performed under severe pressure.

Treatment.—This naturally falls under three heads:

(1) The early symptoms of phthisis have to be checked, and the disease from being active has to be brought into a state of quiescence.

(2) A strenuous effort must, if possible, be made to arrest its further progress or to prevent a relapse.

(3) If this effort fails, one must aim at retarding the fatal issue to the furthest attainable limit.

(1) We have seen that many cases begin with alarming hæmoptysis. The patient must then be kept strictly in the recumbent position for two or three weeks at least. He should not be allowed to talk. His diet should be limited almost entirely to milk. He should have ice to suck, and everything that is given to him should be cold. Of styptics it is difficult to say which is the best; gallic acid, sulphuric acid, acetate of lead, ergot, digitalis, have each their advocates, and it sometimes seems necessary to try one after another. A large ice bag may also be placed over the chest. When the hemorrhage has ceased, the patient is very cautiously allowed to get up and to move about, and the amount of food is gradually increased, while the pulse and temperature are being carefully watched from day to day.

Now, for my own part, I think that whenever phthisis sets in with well-marked early symptoms, even though hæmoptysis may be absent, the same regimen should be adopted. The patient should be put to bed and kept absolutely at rest. He should be limited for a time to a very light diet, consisting mainly of milk, without wine or other stimulant. Whether hemorrhage may have occurred or not, a very good prescription, if there is much pyrexia, is Niemeyer's pill of quinine (gr. i), digitalis (gr. ½) and opium (gr. ¼), to be taken every six hours. Tincture of iodine should be applied to the affected part of the chest, or even a blister, or the croton-oil liniment. I believe that it is almost an advantage to a patient, if he is to have phthisis at all, that it should set in with hæmoptysis; because then the real gravity of his condition is appreciated and there is no hesitation in carrying out the measures which are necessary. So, again, "catarrhal phthisis," in which an apex becomes quickly consolidated, has been held distinct from the "tubercular" form of the disease, because it yields so readily to treatment; and I quite believe that cases in which rapid consolidation occurs may run a more favorable course than others. But I am inclined to suspect that it is because they are taken in hand more carefully and more energetically.

When the acute symptoms have passed off, the patient may go to the seaside for a few weeks, or to some dry and healthy place inland, such as Tunbridge Wells or Ben Rhydding, if the season be suitable. On the Continent a favorite plan is to send him to Lippspringe, near Paderborn, to drink the water of the lime spring there, or to Soden and other health resorts in the Taunus.

(2) It is impossible to insist too strongly on the importance of not letting slip the opportunity, which occurs in phthisis only at an early stage, of arresting its further progress and of preventing a relapse. The measures by which this—the virtual *cure* of the disease—can alone be effected, all involve, as a rule, a prolonged change of climate. They are as follows:—

a. A long sea voyage, generally either to the Cape of Good Hope (or Natal) or else to Australia by the Cape of Good Hope. About twenty-three days are taken in going to the Cape, twenty-eight days to Natal; the voyage to Australia varies greatly in length according as it is made in a steamship or in a sailing ship, being in the one case about forty days, in the other about three months. With regard to a host of details, a knowledge of which is absolutely necessary to the invalid for whom a long voyage is recommended, information must be sought either in a little book by Dr. Wilson, "The Ocean as a Health Resort," or in a series of papers by Dr. Faber, published in the "*Practitioner*" during 1876–1877. On the whole, Green's sailing ships appear to be preferable to steamers, one among other advantages being the greater length of the voyage, which renders the changes of climate less sudden and trying to the health. There is, of course, considerable heat in crossing the equator, and, on the other hand, during the latter half of the journey to Australia the weather is very cold and stormy. As large a part

of the day as possible should be passed in the open air, and exercise on deck should be systematically taken. It seems to be a very important point that the excessive appetite which generally arises should not be freely indulged. A stay in Australia of from six weeks to three months should be made, at the end of which time the voyage home should, if possible, be made by the Cape, this taking generally three and a half or even four months. Dr. Faber insists that no patient should be sent to the Antipodes who is not quite free from pyrexia in the evening; the climate of the tropics is very apt to cause a great increase of fever and to render it continuous, from having been hectic in type. It is also apt to bring on hemorrhage, so that a marked disposition to hæmoptysis, or the presence of rigid, degenerated systemic arteries in an old person, constitutes another strong objection to a long sea voyage. The extent to which an individual is likely to suffer from sea sickness cannot be foretold, unless it has already been proved by former experience; the result of a short trip across the Channel decides nothing, even if there should have been violent sea sickness, as to the probability of its persisting and causing serious exhaustion.

b. Residence in an elevated mountain region for a length of time, or at least in a dry, bracing climate. It is impossible for me, in the limits of this work, to enter into full details with regard to climatic treatment of phthisis. I must refer the reader to other works, especially the fourth edition of Dr. Walshe's "Diseases of the Lungs," Dr. Weber's translation of Braun's "Baths and Waters," and a little book by Mr. R. H. Otter, entitled "Winters Abroad."*

Within the last few years it has become a common practice to send consumptive patients during the winter to Davos, a village situated in the eastern part of Switzerland, at an elevation of about 5200 feet above the sea. Other places, perhaps, might be found which would yield equally good results; and St. Moritz and Samaden, in the Engadine, had in fact been tried even earlier than Davos, at least by English invalids. The great peculiarity of the weather there is the stillness and the dryness of the air. In the shade the cold is extreme, but as the sun is very powerful, and as the sky is generally perfectly clear, patients are able to take exercise nearly every day—walking, skating, driving in sleighs, or "toboggining." When sitting in the verandahs of the hotels the sunshine is hot. At night the double windows in the bedrooms are left slightly open; yet so motionless is the air that the temperature within scarcely falls below 50° Fahr., even when it is from 2° to 16° Fahr. outside. Many persons who are very liable to take cold elsewhere are free from the tendency at Davos. The proper time for a patient to arrive there is about the first or second week in October, or even earlier. It is generally supposed to be undesirable for him to remain after the beginning of April, when the snow melts. Unfortunately, there is no little difficulty in saying where he should then go. He should on no account return to England before the first week in June; and during the interval the choice seems to lie between Baden-Baden, Wiesbaden, Montreux, on the Lake of Geneva, and Monte Generoso above Lago Lugano. The fact that hæmoptysis has been one of the symptoms does not appear to be an objection to sending a consumptive patient to Davos; but the actual presence of pyrexia is an objection, and still more so irritability of the larynx or trachea.

In America there are mountain climates in which phthisical patients derive great benefit without being exposed to most extreme cold. This was

* [To these may be added, "The Influence of Climate in the Prevention and Treatment of Pulmonary Consumption," by Dr. Theodore Williams (1877); and Dr. Weber's interesting Croonian Lectures on "The Hygienic and Climatic Treatment of Consumption," "*Lancet*," March, 1885.—Ed.]

pointed out long ago by Dr. Archibald Smith, who practiced for many years in Peru; but, indeed, it seems to have been familiar knowledge to the Peruvians themselves, who regard the valleys of the Andes, from 8000 to 10,000 feet above the sea level, as almost omnipotent in the prevention and cure of consumption. As a general rule, it may be said that the nearer the equator the greater the elevation which is necessary to render a mountain region salutary in such cases. The chief resorts in the Cordilleras appear to be Huanuco and Janja. Dr. Walshe recommends, as more accessible, the plateau of Santa Fe de Bogotà in New Granada. A great peculiarity of this place is the equality of its climate at different seasons; the mean temperature of each quarter of the year is within a degree or two of 86° Fahr. Other mountain regions to which phthisical patients may be sent are San Paulo, near Santos, the table lands of Guatemala and of Mexico, and Manitoba in Colorado. One can hardly doubt that in the Himalayas, also, there must be valuable resorts; Dr. Weber is inclined to think that the present military sanitaria there may not be at a sufficient elevation for the climate.

c. A prolonged stay in the Southern Hemisphere during what would be the winter of Europe, but is, of course, summer there. Many parts of Australia are very serviceable to phthisical patients. It must not, however, be imagined that a residence in the large towns is advisable. Melbourne, in particular, is apt to be intensely hot and dusty, with very rapid changes of temperature and piercing winds. The best health resorts appear to be certain places in the interior of New South Wales, especially Bathurst, Goulburn, Boural (3000 feet above the sea), and Currajong, but above all, the Darling Downs, in the south of Queensland, where the weather is cool, dry, and bracing. During a large part of the year, the Riverina, also, has a magnificent climate, but in the hot season it is advisable to go elsewhere. Both Tasmania and New Zealand are suitable for consumptive cases, Hobart Town and Wellington or Auckland being especially well spoken of.

Certain parts of South Africa have climates which appear to be very favorable to phthisical patients, but it is essential that no long stay should be made at the sea coast; Mr. Otter says not within 100 miles of it, nor at a less elevation than 1500 feet. The easiest way of reaching the interior is to land at Port Elizabeth, and to go on by Grahamstown to Cradock or to Bloemfontein, the capital of the Orange Free State. This, however, cannot be done safely by an invalid, except at great expense, on account of the badness of the roads, and the accommodation becomes rougher and rougher the greater the distance from the sea. Bloemfontein has an exceedingly dry climate; the daily range of temperature is great, but this is said not to act prejudicially. The best plan for those who can afford it is said to be to buy an ox-wagon and to "trek" through the Free State and the Transvaal for three or four months, sleeping generally in the wagon.

The *modus operandi* of change of climate, as a curative agent in phthisis, is still uncertain. In many instances the beneficial influence on patients who come from a distance finds its parallel in the fact that natives of the same district are very seldom, if ever, attacked. This appears to be the case, for example, in the high Alps, as well as in the valleys of the Andes, and until recently it was so in Australia, although the disease is now rife among the inhabitants of Melbourne and of other large towns there. But Dr. Walshe and others have rightly insisted that there is no necessary connection between the two things, and there is no difficulty in finding countries, such as Iceland, to which one would not think of sending a consumptive patient, notwithstanding that the natives escape the disease. To me it appears evident that the "aseptic" character of the air of a place cannot ever be the direct reason why phthisis should cease to advance in those who inhale it. Observations showing that meat remains fresh there longer than

elsewhere are altogether inapplicable; the only case that could be in point would be if putrefaction once started should fail to become complete. It is very probable that ozone has a therapeutic action in disease, but I doubt if we can at present say what the nature of such action may be. I am, for my own part, disposed to think that the good effects of change of climate depend upon its improving the general health and increasing the resistance of the organism to the further progress of the disease. The recognition of a tubercle bacillus seems altogether in favor of such a view, which also enables us to understand how climates widely opposed in character may be alike in their operation.

(3) When from any cause the arrest of phthisis (its virtual *cure*) is no longer attainable, a great deal may still be done to prolong the patient's life and to give him relief from suffering. The climates which Dr. Walshe terms *sedative* seem to find their chief uses under such circumstances. I may cite, for example, Madeira, Pau, Torquay and Penzance, and, as far as I am at present advised, I should seldom expect much more from some of the climates which Dr. Walshe classifies as *stimulant*, including St. Leonards and Hastings, the various health resorts of the Riviera, Algiers, and even Egypt. Of the Upper Nile, however, Dr. Walshe speaks in terms of the highest praise. And I can well believe that a Nile voyage is the best thing for patients who dislike cold, and who habitually feel stronger and better the hotter they are. Whether more than temporary benefit can be anticipated from places where the air is impregnated with resinous emanations from pine forests—as Arcachon and Bournemouth—I do not know. It must, of course, be remembered that phthisis sometimes becomes quiescent without any change of climate whatever.

On the other hand, although it is a heavy responsibility to advise or allow a patient with advanced lung disease to take a long sea voyage, or to spend a winter in Australia, in South Africa, among the Andes, or even at Davos, there is no question that, if he chooses to run the risks inseparable from such undertakings, he has at least a chance of unexpected benefit.

I have left to the last the treatment of phthisis by drugs, because I was anxious to mark as strongly as possible the greater influence of hygienic measures. But tonics are, of course, to be administered. Still more serviceable in many cases is the cod-liver oil, in doses of from a drachm to half an ounce two or three times a day. Now that this is taken by almost all consumptive patients, some of whom do not even wait for it to be formally prescribed, one is apt to underrate its real importance as a means of preventing emaciation and keeping up the strength. It is said to be less useful in proportion as the age of the individual is more advanced. It sometimes causes diarrhœa, but Dr. Walshe remarks that it by no means always increases that due to tubercular ulceration. If it gives rise to nausea and vomiting, cream may sometimes be substituted for it, or glycerine, but I doubt their being as good. Malt extract is held by some observers to have a similar action. In children, cod-liver oil may often be rubbed into the skin with advantage.

The cough of phthisis has to be combated by the usual remedies. Most prescriptions contain a small dose of opium or morphia, together with tolu, aniseed, benzoic acid, or some other of the so-called expectorants.

The only other symptom that needs special mention is the night sweating. This may sometimes be checked by sponging the chest and the arms at bedtime with vinegar and water. Sometimes it ceases if a subcutaneous injection of atropine ($\frac{1}{200}$ to $\frac{1}{100}$ of a grain) is given at bedtime, or a dose of belladonna, oxide or sulphate of zinc, gallic acid, or sulphuric acid. But in too many cases it persists in spite of all treatment.

MILIARY TUBERCULOSIS OF THE LUNGS.

DISTINCTION FROM PHTHISIS—MORBID ANATOMY—PHYSICAL SIGNS—CLINICAL SYMPTOMS AND COURSE—DIAGNOSIS BY CONCOMITANT TUBERCULOSIS, ESPECIALLY OF THE CHOROID—ÆTIOLOGY—PROGNOSIS.

Both from a clinical and from a pathological point of view it is necessary to distinguish from cases of phthisis—in which tubercles spread through the lung from the apex downward—those of miliary tuberculosis, in which each tubercle appears to be the result of the deposition in the pulmonary tissue of a particle of virus (probably a bacillus, or the spore of a bacillus) brought to the organ from elsewhere by the blood current. True, the distinction is not absolute. For, on the one hand, in many instances of phthisis, dissemination by the blood current sooner or later occurs, and often constitutes the immediate cause of death. And, on the other hand, it is probable that in some instances of miliary tuberculosis the tubercles, originally not very numerous, at length form the starting points of a process of local infection, extending from each of them as a centre, until an affection exactly like phthisis is developed. The cases which most demand attention in this chapter are those in which the lungs, previously healthy or but slightly affected with phthisis, become suddenly the seat of such immense numbers of tubercles that acute symptoms arise and life can no longer be maintained.

It must, however, be remembered that other organs are almost always attacked at the same time. If tubercles appear in the membranes of the brain, they generally (but not invariably) give to the disease its main clinical features. If the peritoneum is greatly involved, there may appear to be nothing but abdominal mischief. In other words, it is often almost an accident whether a case should be regarded during life as one of tubercular meningitis, or tubercular peritonitis, or of miliary tuberculosis of the lungs. And, lastly, there are cases which run their course without definite clinical localization. One cannot even say absolutely that the degree of severity of the pulmonary symptoms is in every instance directly proportionate to the number of miliary tubercles in the lungs. In cases which are classified as examples of tubercular meningitis, at any rate, the lungs are often intensely affected.

Anatomy.—With regard to the morbid anatomy of miliary tuberculosis of the lungs I have little, if anything, to add to what I have stated in the chapter upon Tubercle in general. As I have there remarked (p. 94), many cases occur in which, from the greater abundance of the tubercles in the upper lobes, and from their more advanced state there than toward the bases, it is clear that the proclivities of the pulmonary tissue in different regions produce their effect on this disease, as well as in phthisis. I have also sometimes noticed that when a general outbreak of miliary tubercles occurs throughout the body of a patient who previously had the apex of one lung affected with phthisis, that lung contains more numerous (or, perhaps,

much larger) tubercles than the opposite one, from which fact I inferred that it had a greater inherent tendency to the occurrence of tubercles in it, and that this manifested itself when their development was acute no less conspicuously than when it was chronic. The characters of the tubercles themselves vary widely in different cases. Sometimes they are mainly lymphoid in structure, sometimes they are almost entirely catarrhal. Sometimes they are gray, tending not to caseation, but to fibrous change or cornification (p. 87). Sometimes they become cheesy almost as soon as they are formed. In some exceptional instances, and only toward the apices, they are found to have already softened in their centres with minute vomicæ.

Signs.—Clinically the recognition of this, as of all other pulmonary diseases, depends partly upon physical signs, partly upon symptoms.

The physical signs of miliary tuberculosis of the lungs are in most cases vague and doubtful. Jürgensen has, indeed, described in the "*Berlin. Klin. Wochenschrift*" for 1872, a case in which during five days he heard over a large part of both sides of the chest a peculiar, soft, rubbing sound, perceptible also to the touch; when death occurred, two days later, the only cause to which this sound could be attributed was the presence of a number of miliary tubercles situated on the right side beneath the pulmonary pleura, which was free, and on the left side in the substance of adhesions which completely closed the cavity. From the very first day the soft quality of the sound led him to conclude that it was produced by miliary tuberculosis and not by pleurisy. The patient complained of no pain and could draw a deep breath without embarrassment. Jürgensen thinks that in future cases a positive diagnosis may safely be based upon this sign. Burkart has since maintained (in vol. xii of the "*Deutsches Archiv*") that he has twice detected with the hand a friction sound, due to the presence of obsolete tubercles, which was not soft but rough.

In all probability miliary tubercles are never set sufficiently close together, even in the apex of a lung, to impair the percussion resonance of the corresponding part of the chest. Sometimes it appears doubtful whether the sound is not slightly dull beneath one or both of the clavicles; but if this is so, the dullness is most likely due, not to the tubercles themselves, but to the collapse of the surrounding pulmonary tissue. Dr. Eustace Smith remarks that in children such an interpretation of it is borne out by the fact that variations may be observed from day to day, the resonance becoming good where it had been deficient; and I shall presently mention the case of an adult patient in which the same thing seemed to occur. On the other hand, it is not uncommon for the presence of pulmonary emphysema to render the percussion sound hyper-resonant; and the progressive emaciation of the patient tends to modify it in the same direction as the case goes on. With the stethoscope one may be able to detect absolutely nothing abnormal, even where tubercles exist in enormous numbers. But in some cases, especially toward the apex, the vesicular murmur has a harsh quality, the cause of which is not very apparent. More frequently the auscultatory signs of bronchitis are present, sometimes to an extreme degree. Not only may sibilus and rhonchus be audible more or less extensively, but there may be also abundant moist sounds, from fine and crepitant up to much larger and coarser râles. The expiration, too, may be prolonged and wheezing. On post-mortem examination bronchitis is found in such cases, the smaller tubes being reddened and filled with muco-pus. But sometimes the moist sounds are so bright and clear—so *consonating* in quality—over the upper lobes that it is difficult to believe that there is not diffused infiltration, with "breaking up" of the pulmonary tissue. In one such case which occurred at Guy's Hospital in 1874, there were in fact a large number of small cavities, especially in the left apex. These were evidently of older

date than the general eruption of miliary gray tubercles, which filled every organ in the body, and it appeared from the history that the patient had had a cough for three months, whereas his more acute illness began only ten days before death. But in another case, in 1868, it is reported that there were "mucous râles" at the left apex, "gurgling" at the left base, and "pneumonic crepitation" over the right upper lobe; and yet the tubercles were nowhere seen softening, the only source of the moist sounds being pus in the smaller tubes. I can remember other instances in which post-mortem examinations have shown that the lesions were much less advanced than had been thought during life. One such case occurred in 1882 in a woman, aged twenty-five, under my care in Guy's Hospital. When she was admitted, on July 19th, the only physical sign was a slight crackling sound heard at the right apex after she coughed. However, on the 28th there was a marked crepitant râle in both upper lobes, and especially along the anterior edges of the lungs; and during the next three or four days its character became so "consonating," that I was almost disposed to look upon the disease as acute phthisis rather than as miliary tuberculosis. But at the autopsy, made on August 4th, the lungs, though bulky and œdematous, everywhere contained air; the tubercles were discrete, nowhere softening, and caseous only in the upper lobes; the tubes yielded a frothy fluid.

Clinical Symptoms.—These fall under two heads. On the one hand there are cough, dyspnœa, and other indications of embarrassment of the breathing; on the other hand there is pyrexia.

There is always more or less troublesome *cough*, and it is generally short and hacking. Sometimes there is no expectoration at all, sometimes it consists of a clear mucus, sometimes it is muco-purulent. There are not infrequently streaks of blood in it, and it may be quite "rusty" in appearance, like the sputa in acute pneumonia, or even plum colored. Actual *hæmoptysis* in any considerable quantity is not common. But in 1869 there was brought into Guy's Hospital the dead body of a child, aged five, who was said to have been well on the previous evening, and to have eaten some herring for supper. In the course of the night it was found to have brought up blood and to be in an alarming state, and it died on its way to the hospital. An autopsy showed that there was an acute general tuberculosis; and some of the tubercles in the lungs were already caseating, especially in the upper lobes. No definite source for the bleeding could be discovered; the pulmonary tissue was mottled with blood drawn into it by inhalation. In all probability the cause of hæmoptysis in such cases is the extremely congested state of the vessels immediately outside the tubercles, which often gives them the appearance of being surrounded by a reddish-brown border after death; in fact, obvious points of capillary hemorrhage may sometimes be seen, not only in the lungs, but in other organs.

Far more significant is *dyspnœa*. At first the breathing is only hurried, the number of inspirations in the minute gradually increasing until it reaches fifty to sixty, or in children eighty to ninety. In the woman aged twenty-five, whose case I mentioned above, it was counted at fifty-six on the very day of her admission. After a time the patient becomes conscious of shortness of breath; there is orthopnœa, the movements of the thoracic muscles are forced, and the nostrils work. The cheeks, the lips, the fingers, and the nails are of a lilac or purple color. This symptom is one which more than any other suggests the idea of pulmonary tuberculosis to the experienced physician, whenever there is no long-standing emphysema or heart disease to account for it. I have notes of only one case in which it is said to have been altogether absent. Sometimes albumen appears in the urine. There is not infrequently slight œdema of the lower limbs, and the face becomes puffy and swollen. In my patient, to whom I have more

than once referred, the urine contained sugar during the first few days after her admission into the hospital, the proportion being, on one occasion, 0.4 grain, and on another occasion 0.265 grain in the ounce. Senator's experiments seem to show that this cannot be attributed to deficiency of oxygen in the blood; and, indeed, as the case went on and marked cyanosis developed itself, the glycosuria ceased.

Pyrexia seems to be invariably present, but it varies greatly in its degree and in its course. Sometimes the temperature ranges up to 104° or 105°, but more often it remains at a lower level, perhaps not at any time exceeding 102°. Its progress is atypical. For two or three days there may be scarcely any differences in the thermometric readings at different periods of the twenty-four hours; and then the usual diurnal variations may appear in an exaggerated form, or what is termed the *typus inversus* may show itself, the morning temperature being higher than that of the evening. Brunniche is said to have observed this in fifteen cases out of seventeen, so that it would appear to be decidedly more frequent than in other febrile diseases. Jürgensen makes it a point that the pyrexia does not yield to tepid baths or to antipyretic remedies like quinine so readily as in the specific fevers. This opinion, however, seems to have been partly theoretical, and based upon the idea that in tuberculosis the high temperature of the body generally is the result of the local morbid process, just as when there is inflammation. But since the discovery of the bacillus, there is surely a probability that the presence of this organism in the blood causes, at least in part, the rise of the thermometer; and, if this is so, the distinction from a specific fever can no longer be maintained. The onset of the pyrexia is usually gradual, and the patient does not take to his bed until he has been ailing for some days. But Rühle, in Ziemssen's "Handbuch," speaks of an initial rigor as not infrequent. There are the ordinary symptoms of headache, malaise, depression, intense thirst, loss of appetite, etc. The skin is often wet with perspiration. Epistaxis occurs in some cases, and herpes may appear about the mouth. The pulse is generally very rapid—often out of all proportion to the height of the temperature. There may be a flush on the cheeks, but the face is more usually pallid before the lividity makes its appearance. At one time it was taught that in miliary tuberculosis enlargement of the spleen is exceptional, and that if it occurs at all it is only slight. But all observers seem now to be agreed that some degree of swelling of the organ is almost constantly to be detected by careful percussion, at least when the disease is at an advanced stage. Rühle says that if tubercles are developed in the spleen it may become as large as in enteric fever, and may be tender on pressure. Toward the end the typhoid state may develop itself, with sordes, a dry, brown tongue, subsultus, delirium, and coma. Death is sometimes preceded by a rise of temperature, sometimes by a fall and by collapse.

Diagnosis.—In many instances one is much assisted in the recognition of miliary tuberculosis of the lungs by indications of a like affection of some other organ. Thus the case may at any period of its course become complicated with symptoms of tubercular *meningitis* in a more or less marked form. Sometimes, though very rarely, there is more or less *jaundice*, due to the presence of very numerous tubercles in the liver. In other exceptional cases the occurrence of a tubercular inflammation in one or more of the *joints* may perhaps aid in clearing up a doubt as to the nature of the disease. The most striking example of this with which I am acquainted was recorded by Laveran in the "*Progrès Médical*" for 1877. A man, aged twenty-two, was attacked with articular pains, especially in the knees. Effusion occurred into the right knee joint, and when admitted into hospital he was supposed to be suffering from subacute rheumatism. However, at the end of a week,

great dyspnœa set in and high fever, the temperature ranging from 102° to 104°. A fortnight later he died, the cause of death being acute tuberculosis. The synovial membrane of the right knee was found to be injected and covered with a large number of grayish granulations the size of pins' heads, which could be felt. Where they had been in contact with a surface of articular cartilage their summits were flattened. A few granulations were present also in the left knee. In 1867, a woman died in Guy's Hospital, of tubercular meningitis, whose right knee had become swollen and painful in the course of her illness. At the autopsy all that was noted was that the synovial membrane was very vascular and œdematous, and that the cartilage over the external condyle of the femur was slightly eroded. But I do not think it is at all unlikely that tubercles were present. In 1880, in a patient who died of cancer of stomach, and who had suffered from a painful affection of the knee attributed to an injury eight months back, I found the soft tissues of the joint full of the most conspicuous tubercles, many yellow and caseating; but this is hardly a case in point. Cornil and Ranvier have shown that in cases of acute tuberculosis miliary granulations may often be found in the cancellous tissue of the bones, especially in the vertebræ, the sternum, and the ribs. It seems quite possible that their presence may sometimes give rise to pains vaguely referred to different parts of the limbs and body, but probably these could not be distinguished from the similar pains which so commonly accompany various forms of pyrexia.

From a clinical point of view, the most important of all the seats of miliary tubercles is one where they can actually be seen during life, although it seldom, if ever, happens that there are symptoms which draw attention to their presence. In 1857, a German observer, Manz, discovered tubercles in the *choroid* of each eyeball in a girl who had died of acute tuberculosis. Subsequently he and Busch recorded other instances of a similar kind. And in 1867–1868 Cohnheim, investigating this point carefully in all the cases of miliary tuberculosis—eighteen in number—that came under his notice in the Pathological Institute at Berlin during a period of fourteen months, found that in every instance one or both of the eyes—almost invariably both of them—showed choroidal tubercles. In April, 1867, the ophthalmoscope was for the first time used, apparently by v. Graefe himself, for the discovery of these during life in a patient of Griesinger's. In November of the same year Mr. Soelberg Wells exhibited to the Pathological Society of London a specimen of choroidal tuberculosis, which he had detected in a little girl under the care of Dr. Garrod, five days before her death. Since then many other observers have recorded similar facts. But further experience seems to have shown that the eyeballs are very far from being so constantly involved in cases of miliary tuberculosis as would be supposed from Cohnheim's statement. It is to be noted that the development of tubercles in the choroid appears to bear no specially near relation to their occurrence in the pia mater of the brain; all that can be said is that the more numerous the organs which are the seats of tubercle in a given case, the more likely is it that the eyeballs will be affected. One of the most remarkable cases was recorded by Fränkel, in 1872, in the "*Berliner Klin. Wochenschrift.*" A delicate girl of six was attacked in May of 1871 with slight shiverings, and her temperature rose occasionally to 100.4°. Then partial ptosis appeared and afterward paralysis of some of the ocular muscles. On May 22d the ophthalmoscope showed a white patch to the upper and inner side of the disc in the left eye; it was as large as the disc itself, and had a rounded form, except that in one direction it was drawn out into a point. By the 1st of June it had increased in size by one half. On account of its characters being so different from those generally described as belonging to tubercles in the choroid, Fränkel hesitated to

diagnose it as tubercular. The child now went into the country with her parents and remained there until August, and when she came back she was apparently in perfect health. The patch in the fundus of the eyeball, however, was more prominent, though not larger than before. On August 21st she became ill with gastric symptoms and pyrexia, and she died on October 1st. On September 10th five fresh miliary tubercles had been detected in the choroid, and afterward a sixth made its appearance; as the case went on they gradually increased in size. Vision remained unimpaired until death. I may remark that the only instance in which any defect of sight had been noted appears to be that of a girl in whom, besides from forty to fifty tubercles in each choroid, Cohnheim found also hemorrhages into the retinæ. In acute cases the number and the size of the tubercles may increase from day to day, as was noticed in a child examined by Fränkel and by Leber. If, however, nothing should at first be detected by the ophthalmoscope in a suspected case of general miliary tuberculosis, the instrument ought to be used again and again as the disease advances. The tubercles vary much in size. The largest seen by Cohnheim was 2.5 mm. in diameter; but Ponfick met with one which measured 5 mm. On the other hand, Cohnheim seems often to have detected them (in the dead body) where they could only be seen after carefully removing the choroidal pigment, and even where they were too small to be visible by the naked eye. It is possible that this may account for the discrepancy between his statements and those of other observers as to the constancy of their occurrence in cases of general miliary tuberculosis. Where there are but few of them they seem to be developed in the neighborhood of the disc or of the yellow spot, more often than toward the equator of the eyeball. When very numerous, some of them may run together into irregular masses. They almost always caseate as soon as they reach about 1 mm. in diameter. With regard to the ophthalmological diagnosis between tubercles and the white spots that are seen in disseminated choroiditis, Graefe and Leber insist on the rounded form generally presented by tubercles, their projecting above the level of the choroid, the gradual thinning away of the choroidal pigment from their periphery inward to their centres, which appear white, and the absence of any accumulation of pigment outside them, except, perhaps, when they are very large.

Ætiology.—With regard to the causes of miliary tuberculosis of the lungs there is very little to be noted. Whatever may be said theoretically as to its relation to caseating tubercular glands, I have very rarely seen it directly traceable to any such affection which had been recognizable during life in the neck or elsewhere. Many of those who are attacked, whether children or adults, are robust and healthy looking, altogether devoid of those indications of a scrofulous diathesis to which I am, for my own part, disinclined ever to attach any importance. On the other hand, one must always be prepared for the supervention of miliary tuberculosis in cases of phthisis, as, of course, it brings the disease to an end far more quickly than might otherwise have been anticipated. It occurs at all ages. Among forty cases observed at Guy's Hospital between the years 1857 and 1873, one was in an infant aged nine months, three were in patients between one and ten years, seven between eleven and twenty, thirteen between twenty-one and thirty, six between thirty-one and forty, four between forty-one and fifty, six between fifty-one and sixty. But it is to be observed that I am excluding from this list most of the cases in which the symptoms clinically were those of tubercular meningitis; if they were taken in, the proportion of children would, of course, be far higher. Burkart gives very similar figures. Among my cases there were almost exactly twice as many males as females; among Burkart's the proportion was as sixteen to two. It is worthy of

notice that in two of my cases the patients were admitted into the hospital for what appeared to be acute pleuro-pneumonia. One, a young man, aged twenty-two, at first complained of pain round the lower part of the right side of the chest, which seemed to be the seat of his disease. He appeared to be doing well, and was allowed to go out to the hospital grounds, when he was seized with an epileptic fit. He was then carefully examined again, and was found to have ascites. The fits continued, and he passed into a state of stupor. Crepitation became audible throughout both lungs, and at length he died, ten weeks after his admission. At the autopsy, the pleura was found to be thickened over the base of the right lung, and there was fluid. The right lower lobe was completely airless; it contained only a few scattered miliary tubercles, forming a marked contrast with all other parts of the lungs, which were full of them. The pericardium was adherent and both layers of it were enormously thickened; nothing is said about its containing tubercles. The liver and the kidneys all contained many tubercles, the liver also was cirrhotic. There appeared to be only a few tubercles in the pia mater. The other patient, a youth of eighteen, came in three months and a half before his death with an attack of pleuro-pneumonia, which (in his case also) was on the right side. Afterward it appeared, according to the clinical report, that phthisis developed itself. On post-mortem examination the lungs were found to be studded with miliary tubercles, especially the right one. There were no cavities anywhere, nor was the pulmonary tissue solidified round the tubercles. The substance of the right lung was unusually firm, a condition which was thought to be the result of the pleuro-pneumonia. The liver and the kidneys contained miliary tubercles. The small intestine showed characteristic tubercular ulcers running transversely; the mesenteric glands and those near the cæcum were caseous. I think it is doubtful whether the relation of the miliary tuberculosis to the pleuro-pneumonia in these cases was other than one of mere coincidence. In children the disease often follows measles, scarlet fever, and smallpox.

Prognosis.—The duration of the disease is commonly three to four weeks, reckoning from the first commencement of marked symptoms up to the time of the patient's death. But, on the other hand, there may be a protracted illness, lasting three or four, or even eight months. Clinically they generally resemble severe bronchitis more than other affection, and they are not seldom mistaken for it until an autopsy reveals their real nature. Indeed, as Burkart has especially pointed out, it is common for miliary tuberculosis to develop itself in lungs which are already emphysematous from old bronchitis. Twelve of his eighteen cases were examples of this, and their course was often very prolonged. In no fewer than six of these, in fact, the tubercles which were found post-mortem were already calcified or fibrous, with a lustre like that of mother-of-pearl, so that they might fairly be considered to be obsolete; and the causes of death were various, sometimes pleural effusion, sometimes dilatation of the right heart, sometimes Bright's disease; the presence of the tubercles seemed to be little more than an accident. I think that I also can recall instances in which I have observed a similar obsolescence of miliary tubercles scattered through the lungs. But I have not hitherto been sufficiently careful in determining what part they took in bringing the patient's illness to a fatal termination. It is worthy of notice that ten of Burkhart's cases occurred in men exposed to the dust of stone or coal in their work, for this fact bears out my belief as to the tubercular character of the "pneumokonioses" (*supra*, p. 997), unless, indeed, it could be urged that the bodies found in the lungs by Burkart were not really tubercles at all, but fibrous nodules formed round embedded particles of silica or carbon.

This question of the possible obsolescence of miliary tubercles in the

lungs is of great importance, in reference to the diagnosis from enteric fever of those cases which run a rapid course, and which are attended with pyrexia as a principal symptom. I have already, at page 218, cited a case of Senator's, which shows that it is sometimes impossible, in the present state of our knowledge, to distinguish the two affections. In this country, I think it has been usual to regard the fact that a patient recovers as conclusive proof that he was not suffering under miliary tuberculosis; although Dr. Bristowe says that the progress of the disease "may be occasionally arrested, but with more or less permanent damage to the tissue of the lung." Now, in cases of fatal tubercular meningitis, we often have opportunities of observing that the number of miliary tubercles scattered through the lungs may be altogether very small. And it is certainly difficult to understand why, if the brain affection happened in such a case to be absent, recovery should not take place. All that would be necessary would be that the tubercles should fail to become the starting points of fresh tissue infection. It may, of course, be a question whether such a scanty development of miliary tubercles would be attended with pyrexia or other clinical symptoms. But as I have already stated, I am disposed to think that the high temperature in this disease is in great part independent of the local lesions, and due to infection of the blood. Wunderlich, as far back as 1860, recorded, in the "*Archiv für Heilkunde*," cases which he regarded as examples of a cured miliary tuberculosis. But he based his diagnosis solely on the fact that the temperature chart failed to correspond with what he regarded as the necessary course of enteric fever. Very few observers, I think, will be found at the present time to endorse his opinions in this respect. The points of distinction between the thermometric readings in the one and in the other disease will appear from the descriptions which I have already given at p. 203, and at p. 1019, respectively. But I must add, in reference to Wunderlich's supposed cases of recovery from acute tuberculosis, that he attached great importance to the occurrence of a prolonged "amphibolic stage," with alternate exacerbations and remissions (sometimes amounting to complete apyrexia) as conclusive against the presence of enteric fever. I must confess, however, that, as it seems to me, the only way in which the occurrence of recovery from an attack of miliary tuberculosis could be proved by clinical evidence alone would be by the discovery of tubercles in the choroid of the eye. Another possibility is, of course, that the patient, after getting well, should die from some other cause, and that the tubercles should be found in a state of obsolescence. In regard to this, Dr. Bristowe speaks of the lung, after the arrest of the discrete tubercles, as becoming "seamed throughout with minute patches of cicatricial tissue, the fibres of which have something of a stellate arrangement, and within the limits of which the lung tissue presents, from the presence of concurrent emphysema, a coarsely spongy character; occasionally in the centres of the scars minute fibroid knots or concretions may be recognized." Finally, I must mention that Burkart records the case of a woman, aged twenty-eight, who died after a fortnight's illness, and in whom the lungs, the peritoneum, and the kidneys showed recent miliary tubercles in abundance, while in the intestine there were the typical lesions of enteric fever, some of the ulcers having sloughs still adherent, or partially detached. He also cites eight cases recorded by Birch-Hirschfeld, in which acute tuberculosis is said to have developed itself immediately after enteric fever.

With regard to the *treatment* of miliary tuberculosis of the lungs, all that can be said is that the strength of the patient should be maintained, that symptoms should be checked by appropriate drugs, and that iodide of potassium, or, perhaps, even bichloride of mercury, should be given, in the hope of favoring the obsolescence of the tubercles.

FUNCTIONAL DISEASES OF THE RESPIRATORY ORGANS.

ASTHMA. INFLUENZA. WHOOPING-COUGH.

Asthma—THE TERM—DESCRIPTION OF AN ATTACK—DIAGNOSIS AND RELATION TO STRUCTURAL LESIONS OF THE LUNG—ÆTIOLOGY—NATURE AND PHYSIOLOGY OF THE DISEASE—TREATMENT AND PROGNOSIS.

Influenza — HISTORY — SYMPTOMS — EVENTS — ÆTIOLOGY — DIAGNOSIS — TREATMENT.

Whooping-cough—NOMENCLATURE—SYMPTOMS AND COURSE—EVENTS—PROGNOSIS—PATHOLOGY—TREATMENT.

Asthma is one of those terms of which the scope and application have been greatly narrowed during the present century. Formerly it was used to mean what we now call dyspnœa; and even at the present day it is common to hear persons spoken of as "asthmatic," who are suffering merely from bronchitis and emphysema. Indeed, soon after the discovery of auscultation Rostan and some other French physicians were strongly disposed to deny the existence of any disease deserving to be distinguished by this name, and occurring in persons with healthy lungs. But that there is such a disease there can be no doubt; and now that it has been separated from other affections with which it used to be confounded, its characters are found to be exceedingly definite and well marked. As for its nature, it is commonly believed to depend upon spasm of the smallest air tubes. We shall have to consider presently whether this is an entirely satisfactory theory with regard to it.

Symptoms of an Attack.—Asthma is, in the first place, a paroxysmal affection. It sets in generally with remarkable suddenness; most frequently in the middle of the night, between 2 and 4 A.M.; but in some cases at other times, between 6 and 8 A.M., or in the afternoon. The forenoon is almost always the period in the day when the patient is freest from it. In the same case it commonly begins at about the same hour. When this is, as usual, between two and four in the morning, the patient, who may have gone to sleep in perfect health, wakes up with a sense of oppression of the chest, which soon passes on to the most extreme distress of breathing. But sometimes the seizure is preceded by symptoms which previous experience enables him to recognize as premonitory; among them are a peculiar drowsiness, flatulence, a slight degree of sneezing, a troublesome itching under the chin, the passing of a quantity of pale, limpid urine, like that which is secreted in hysteria and other nervous diseases. However this may be, the urgent dyspnœa which now attacks him compels him to sit up, and, perhaps, drives him to the window, which he throws wide open, in the hope of getting air more freely. Or he may be obliged to sit with his elbows planted upon a table, or to stand with his hands grasping the mantelpiece or some article of furniture above his head; such attitudes being adopted for the purpose of fixing the shoulders and so assisting the muscles of forced

respiration in their action. His face becomes livid or purple, his eyeballs start from their sockets, his hands and feet are cold, his skin is covered with a profuse sweat, and his expression indicates extreme anxiety. In fact, he may appear to be at the point of death.

Examination of the chest shows that the physical conditions are as follows: The breathing is not accelerated, but of normal frequency, or even slower than natural. Its rhythm is perverted, the inspiration being short, whereas the expiration is greatly prolonged. With the inspiration there may be some wheezing, but this is nothing in comparison with that which accompanies the expiration, and which is audible all over the room. The shape of the chest is such as corresponds with a very deep inspiration; the upper ribs are raised to the fullest possible extent and widely separated from one another; the diaphragm has descended toward the abdomen, so that the area of pulmonary percussion resonance extends considerably lower than natural. During inspiration the sterno-mastoidei and the scaleni are brought into action, but there is scarcely any advance in the degree of expansion; during expiration there is but little recession, although the rigid abdominal muscles can be seen and felt to be doing their utmost to expel air from the lungs. Percussion shows much less than the natural amount of difference between inspiration and expiration, as regards the position of the edges of the lungs in relation to the heart and the liver. The percussion note over the chest generally is hyper-resonant. On auscultation the vesicular murmur is found to be almost or quite inaudible; it may be replaced by sibilus, or by rhonchi of varying quality. With the expiration there is heard through the stethoscope the same loud wheezing sound which has been already mentioned as being heard at a distance.

So entirely occupied is the patient with the mere act of breathing that he can scarcely utter a word, or turn his head to one side, or even stop to cough; but after a time a slight cough comes on, leading to the expectoration of a few grayish-white pellets of mucus about as large as peas. Not infrequently the mucus is stained with blood, and sometimes there is considerable hæmoptysis. The occurrence of expectoration generally indicates that the symptoms are about to subside. The duration of a paroxysm of asthma is very variable; usually, it lasts from one to three hours. As it passes off, the patient falls asleep, and when he wakes in the morning his breathing may be quite easy and unattended with any discomfort. But sometimes the disease continues for several days in succession with scarcely any abatement, except that there is almost always some increase in its severity at night, and some lessening during the early part of the day. In such cases the patient's condition causes extreme alarm, although a fatal termination scarcely ever occurs. One instance in which the breathing actually ceased and life was maintained only by artificial respiration has been mentioned at p. 25. When asthma passes off in the usual way, it is apt to return during the following night. The paroxysms may, in fact, recur for several successive nights, and may then cease, leaving the patient entirely free for weeks or months together, but there are other cases in which the disease shows itself night after night for years. I knew a gentleman who, for the last twenty-five or thirty years of his life, was never able to lie down to sleep. When night came on, he dressed himself in a flannel suit and seated himself in a large chair, and in this he remained until the morning.

Diagnosis.—From the above description of asthma it will be apparent that the disease can rarely be mistaken for any other by a careful observer; certainly it ought never to be confounded with those tracheal or laryngeal affections (such as bilateral paralysis of the abductors of the cords) in which the

dyspnœa is mainly inspiratory. On the other hand, it is often only by the history that one can tell whether a patient is suffering from asthma or from bronchitis and emphysema. And according to Trousseau, it sometimes happens that a child (and even an adult) is seized with what appears to be a very acute and dangerous attack of broncho-pneumonia, with abundant moist sounds over the chest, and that the rapid subsidence of the symptoms in the course of a day or two, and the recurrence of like attacks on future occasions, ultimately justify the conclusion that the affection is really asthma. The relations of bronchitis and emphysema to asthma are somewhat complicated. On the one hand, it is not uncommon for patients who have chronic bronchitis to suffer from time to time from paroxysms of dyspnœa, which cannot be accounted for by any increase of the bronchial inflammation, and which seem referable only to a concomitant spasm of the air tubes, and if such spasm is regarded as constituting the essential condition in asthma, it may be fairly said that in these cases a *secondary* asthma is really present as a complication of the bronchitis. A "bronchitic asthma" was, in fact, formally recognized by Salter and other writers; one peculiarity of it is that it is constantly worse in the winter than in the summer, which is not generally the case when asthma occurs as a primary affection. But, on the other hand, if a person with perfectly healthy thoracic viscera becomes subject to frequently-recurring attacks of asthma, his lungs always sooner or later become emphysematous. We have seen that during the paroxysm of asthma the ribs are raised and widely separated, and that the diaphragm is placed at a lower level than natural. It necessarily follows that the lungs are in a state of over distention. Now, when the symptoms quickly pass off, as is usually the case, the chest walls return in a few hours to their normal position and the lungs to their normal size. But if similar attacks recur again and again at short intervals, the inevitable result is that the elasticity of the pulmonary tissue becomes impaired, and that the alveoli become permanently overstretched and emphysematous; ultimately the right side of the heart undergoes dilatation, dropsy sets in and ends fatally. Patients with confirmed asthma gradually acquire a peculiar configuration, which is very characteristic and has been well described by Salter. They are round backed, high shouldered, and stooping; the chest is obviously rigid and without pliancy, and from it the arms hang suspended, but inclined rather backward and bent at the elbows. They are thin almost to emaciation, with prominent veins, cold, thin hands, and a dusky complexion. The cheeks are hollow, the eyeballs turgid and watery, the mouth generally open, and the jaw rather hanging. The voice is feeble and somewhat hoarse and rough.

Ætiology.—With respect to the causes of asthma, two questions have to be asked: 1. What are the conditions which render certain persons susceptible of the disease, whereas other persons seem to be incapable of being affected by it? 2. What are the various exciting causes which, in such individuals, are found to bring on the paroxysms?

The answer to the first of these questions must at present be incomplete. Inheritance plays a certain part in the "asthmatic tendency," as Salter calls it. This writer gives many striking instances of the transmission of asthma from generation to generation; he also mentions cases in which several brothers and sisters in a family were asthmatic without the parents being so. In early life a good many cases appear to be directly traceable either to measles, to whooping-cough, or to an attack of bronchitis; at the time the child seems to recover perfectly, but it becomes for the future liable to asthma, from which it had previously been entirely free. This fact certainly looks as though the fundamental defect were at least sometimes seated in the texture of the lungs or of the bronchial tubes. On the other hand, there are points which suggest that the disease is really a neurosis. Thus

Salter relates the case of an epileptic patient whose fits, after having set in with their usual premonitory symptoms, were on several occasions replaced by asthmatic paroxysms. And another of his cases is that of a gentleman in whom a violent attack of asthma was twice suddenly excited by fear. Then, again, asthma appears to be sometimes closely related to gout. And it has been observed to alternate with cutaneous eruptions, becoming worse when the skin has got better, and *vice versâ*.

The age at which patients first become affected with asthma is very variable. It sets in during childhood much more often than used to be supposed. Salter found that in a fourth of his cases it had begun before the tenth year, and he saw two cases in infants of fourteen and twenty-eight days. More males than females are affected with it, in the proportion of two to one.

The *exciting causes* of the asthmatic paroxysm vary widely in different cases. Indeed, hardly any two patients agree in their statements as to the precise conditions which bring on their attacks. Particular kinds of weather, certain winds, cold air, the confined air of crowded rooms or railway carriages, act as exciting causes in some cases. Or the disease may be especially apt to follow the inhalation of dust, fluff, or smoke, even the smoke of an extinguished candle or of a lucifer match. Some patients are sure to be attacked if they come near to, or in contact with, certain kinds of animals,—cats, rabbits, dogs, horses, guinea pigs, or the wild beasts of a menagerie. Salter relates many remarkable cases of this kind, and what is especially noteworthy is, that years have often passed before the patient has discovered to what simple cause all his sufferings are really due. One man, the proprietor of an equestrian establishment, was continually asthmatic until he retired from business, and then became almost entirely free; but whenever he went back among the horses the disease returned, and so at last he found out that they were to him the special exciting cause. I have known a lady who was attacked with asthma whenever she was in the same room with a cat; the animal could not be hidden anywhere near her without her discovering it by the painful sense of constriction in the air passages which she quickly began to experience. The asthma produced by hay, or rather by the pollen of grasses, is one form of the disease known as hay fever. Some patients never have asthma unless they are exposed to the influence of the pollen; others are habitually asthmatic, this being only one of many causes capable of exciting the disease in them. Many persons are attacked if they inhale the powder of ipecacuanha diffused in the air, and odors of various kinds act as exciting causes in particular cases.

Diet plays an important part in setting up the paroxysms in almost all asthmatic patients. Heavy suppers and late dinners are very injurious; many persons are unable to eat any solid food for several hours before bedtime. Special articles of food, among which are cheese, nuts, coffee, bottled stout and wine, are apt to provoke the disease.

Another occasional cause of asthma is the presence of polypi in the nose. This was first pointed out by Voltolini; it has since been confirmed by Haenisch. Removal of the nasal growths frees the patient from the liability to the recurrence of the bronchial affection. Salter mentions one case in which a paroxysm was sure to occur if the rectum was allowed to remain loaded. Sometimes the attacks are clearly traceable to uterine irritation, as when they return with each catamenial period or come on only during pregnancy or parturition.

But of all the exciting causes of asthma, the most important in its influence is *locality*. And here, again, there are the strangest differences between different cases, so that it almost seems as if the disease were

regulated only by caprice. In certain places the patient is sure to be attacked; in other places he is as sure to escape. As a rule, the places which are most favorable in their effects upon asthmatic subjects are large, crowded, smoky towns, like London, Glasgow, and Manchester. The most extraordinary stories are related by Salter of the effects of London air upon the disease. Persons whose lives had been rendered miserable for years have become entirely free from the disease on taking up their residence in the metropolis. He thought that three-fourths or seven-eighths of all cases of asthma might be cured in this way. It is to be noted that the influence of locality extends to neutralizing the ill effects of some other exciting causes of the paroxysms; the patient may be able in London to eat what he pleases and at whatever hours; whereas in the country the strictest dieting may be required to keep off the disease. On the other hand, there are a few cases in which the air of the seaside or of a bracing, hilly district is found to be the best.

Pathology.—Various theories have been framed to account for the paroxysms of asthma, but I am not sure whether even now the pathology of the disease can be said to be fully established. The *expiratory* character of the dyspnœa, exactly like that which accompanies capillary bronchitis, seems to clearly show that it must depend upon a morbid affection of the very small bronchial tubes within the lungs. During the act of expiration these tubes are as much exposed to pressure as the pulmonary alveoli themselves, and it is not difficult to understand how, when from any cause they are partially obstructed, they may admit air into the lungs in inspiration, and yet, by a kind of valvular action, refuse to allow it to pass out in expiration. The question is what is the nature of the obstruction in asthma. Now, the most obvious suggestion certainly is that it results from spasm of the muscular fibres in the walls of the tubes. That these fibres are capable of contracting, so as to narrow to some extent the calibre of the tubes, has been established by physiologists. The suddenness of the onset of the asthmatic paroxysm, the equally sudden way in which it sometimes subsides under the influence of a violent mental shock or emotion, the marked effect of such remedies as chloral, belladonna, and stramonium in bringing it to an end, the close relation which appears to exist between asthma and certain neuroses, may all be mentioned as tending to confirm the view that it is essentially spasmodic in its nature. On the other hand, it is by no means clear that spasm can account for such considerable narrowing of the tubes as must be present in asthma; nor that it is possible for spasm to be kept up for so great a length of time as that which is sometimes occupied by a prolonged paroxysm of the disease. The alternative hypothesis is that the mucous membrane of the tubes becomes very rapidly swollen by what German writers term a "fluxionary hyperæmia," or (as Weber has put it) by "a dilatation of its blood vessels through the influence of the vaso-motor nerves." The fact that the catarrhal form of hay fever is attended with an obvious swelling of the mucous membrane of the nose is a strong point in favor of this view; for it is surely very unlikely that the asthmatic form of the same disease should be altogether different in its pathology. And Störk is said to have actually observed with the laryngeal mirror that during an asthmatic attack the whole length of the trachea and part of the right bronchus were deeply congested. It is, however, not improbable that the smallest tubes may be affected with both hyperæmia and spasm.

In 1871, Leyden discovered in the sputa certain pointed octahedral crystals, identical with those found in the blood and viscera in cases of leuchæmia, which are commonly known as Charcot's crystals, having been first described by him. Leyden's idea was that these crystals might, perhaps, constitute the starting point of the asthmatic paroxysm, by

irritating the peripheral ends of the branches of the vagi in the bronchial mucous membrane, and so setting up a reflex spasm of the muscular fibres beneath. But this is not at all probable, for they have also been observed in the sputa of patients suffering, not from asthma, but from other bronchial affections.

Treatment.—This falls under two heads: we have, first, to prevent the recurrence of the asthma, and, secondly, to relieve the attacks when they develop themselves, and, if possible, to cut them short.

In endeavoring to prevent the recurrence of attacks of asthma, by far the most important thing is to study carefully its exciting causes in the individual patient, and, as far as possible, to remove him from their influence. A medicine which is in some cases very serviceable, although the theory of its nature is obscure, is the iodide of potassium in doses of about eight grains three times a day.

For the paroxysms of asthma different modes of treatment are useful, some in one case, some in another. Many patients are at once relieved when they are made faint and sick by an emetic dose of ipecacuanha or by smoking tobacco. The latter is said to be the best remedy for hay asthma; unfortunately, those who smoke habitually are incapable of deriving benefit from it. In some cases nothing does so much good as strong hot coffee taken on an empty stomach, or hot whisky and water, or gin, or brandy. In other cases the inhalation of chloroform gives very rapid, but generally only temporary relief. Smoking the leaves of stramonium (or of one of the other species of Datura, the *D. ferox* or *D. tatula*) is often very effectual. Or the patient may be given stramonium as a tincture or an extract, or the ethereal tincture of lobelia in full doses, or tincture of belladonna, or chloral. Some patients derive the greatest possible benefit from the fumes of nitre paper, burned so as to fill the room with white smoke. In other cases nothing does so much good as the inhalation of a green powder which is sold as a secret remedy in the United States, and which (it is said) may be imitated by mixing together nitre, powdered stramonium, and powdered aniseed. For details in the management of cases of asthma, I would strongly advise my readers to consult the late Dr. Hyde Salter's work, based as it is upon a vast experience of the disease, as well as upon his own sufferings from it. It contains on almost every page practical hints of the greatest value.

In regard to the *prognosis* in asthma, one very important fact is that when the disease occurs in childhood, it generally subsides about the age of puberty, leaving the patient free for the rest of his life. On the other hand, persons above the age of forty or forty-five seldom, if ever, get rid of a liability to it. The longer and the more frequent the paroxysms the more serious is the case. It is also essential to notice whether in the intervals between the attacks there is any shortness of breath, or cough with expectoration. For such symptoms indicate that the asthma is complicated with chronic bronchitis, or emphysema, or dilatation of the right side of the heart; and the presence of any permanent organic lesion of the thoracic organs adds enormously to the gravity of the disease.

Influenza.—There is a form of catarrh of the nose, throat, and air passages which, from the manner of its distribution as an epidemic, is evidently a specific disease. This is the *influenza*, as it is termed both in England and in Germany, the name being of Italian origin and having come into use about two centuries ago; in France it is known as *la grippe*, a word which is said to be derived from the Polish Crypka or Grypka (— *raucedo*). Its history is supposed to date back into remote antiquity; but, as might be expected, doubts exist as to the real nature of many of the older

epidemics, one of which is supposed to have attacked the army of Charlemagne, when returning from Italy, in the year 876. During the last four centuries the periods during which influenza has prevailed have been carefully recorded; the late Dr. Parkes (in Reynolds' "System") says that there were eleven in the sixteenth century, sixteen in the seventeenth, and eighteen in the eighteenth. Between 1800 and 1850, there were no fewer than ten, of which three were most important; one, in its spread over different countries, occupied the years 1830 to 1833, another occurred in 1837, and the third in 1847–1848. It seems to be doubtful whether there has been any reappearance of the disease during the second half of this century.

Course.—Most of the symptoms of influenza are very like those of a common feverish cold; but it is attended with greater pyrexia, and with more severe depression of the vital powers. The patient, perhaps, suddenly becomes chilly, and even shivers, and for some hours he may feel exceedingly ill, before any definite local affection appears. Sir Thomas Watson says that when he was first called to two cases on April 3d, 1833, the symptoms were just those which frequently mark the commencement of an attack of continued fever, and that he did not then know what was about to happen; but in the course of that and the following day all London was smitten with the disease. Since the introduction of the thermometer into clinical practice, no well-marked epidemic has occurred; but it is believed that the fever presents nocturnal exacerbations, and that it is sometimes very high; the pulse is rapid, and it is also remarkably weak and small, and sometimes intermittent. After a short interval, or even from the very first, the patient begins to sneeze, and a thin, acrid fluid runs from his nose, his eyes become red and watery, his fauces are reddened, he becomes more and more prostrate, so that he cannot keep up, and is altogether unable to perform his customary duties. He may suffer from giddiness or faintness; in some epidemics drowsiness is a very common symptom, in others many patients become delirious. Pains in the limbs or cramps in the calves may be complained of. There is always severe headache, and often an intense pain at the root of the nose which is attributed to extension of the catarrh to the frontal sinuses. At the same time, or a little later, he is attacked with an irritating cough, dry, or attended with scanty mucous expectoration. Sometimes there is great dyspnœa, with a sensation of distress in the precordial region, and a liability to severe suffocative attacks; the lips and face may even become livid. Loss of appetite is a marked symptom; the tongue is thickly furred; there may be nausea and even vomiting; sometimes diarrhœa is present; the skin may have an icteric tinge; the urine is scanty and high colored. In pregnant women abortion may occur, as the result (it is said) of the violent cough, or, if the menstrual functions have been suppressed from other causes, they sometimes now become re-established.

At the end of from three to five days the attack passes off. It it lasts much longer, the usual reason is either that pneumonia has developed itself, or that the catarrh of the air passages has passed into a regular bronchitis. The subsidence is sometimes gradual, sometimes by a critical sweating or diarrhœa; sometimes it seems to set in with epistaxis. The patient's convalescence is always slow, and he is long in regaining his strength. Relapses are said not to be infrequent, but Dr. Robert Williams says that few persons suffer more than one attack during the same season. On the other hand, to have had influenza in the course of an epidemic seems to confer no immunity against its recurrence on a subsequent occasion.

In some cases the disease assumes a rudimentary form, the patient merely complaining of a slight coryza, perhaps accompanied with severe sore throat and cough, and attended by a little malaise, headache, and disinclination for work.

Mortality.—It may appear paradoxical to say that influenza is seldom fatal, and yet that it always causes a great increase above the normal death rate of the towns in which it breaks out. But the explanation is that almost all the mortality from it is brought about indirectly, and that the numbers of those who fall ill with it are greater, beyond all comparison, than in the case of any other disease. In London, in 1847, it is said to have destroyed 5000 persons in six weeks; but then 250,000 persons are computed to have been attacked by it. In Paris, above one-fourth of the population suffered; in Geneva, not less than one-third. Those who die are chiefly old and debilitated subjects, who have previously labored under emphysema of the lungs, or who have feeble and dilated hearts. It is sometimes dangerous to very young children. Parkes states that patients with lesions of the valves of the heart, and some at least of those who have phthisis, pass through influenza without being the worse for it. But other writers have remarked that after its subsidence phthisis often takes a very rapid course. Dr. Farr pointed out, in 1847, that the mortality was much greater in those districts of England in which the death rate was generally high than it was in heathier places.

No special morbid changes are seen in the bodies of those who have succumbed during an attack. The lungs and air passages are congested, sometimes to an extreme degree, and there may be great œdema of the pulmonary tissue. Pneumonia is not infrequent, but this is regarded as a complication; it may be either catarrhal or croupous. Plastic exudation into the bronchial tubes has now and then been found.

Recovery from influenza is sometimes followed by parotitis; and it is also stated that when the patient had previously suffered from neuralgia this affection is apt to relapse after convalescence from the epidemic disease.

Ætiology.—As to the cause of influenza there have been many speculations, but as yet no positive conclusion has been arrived at. Certain observations which have been made as to the way in which it spreads are, however, of great importance, as suggesting some possible solution of the difficult question of its origin or as negativing others. No other disease diffuses itself equally widely over the earth's surface. Not only is it capable of existing in all inhabited regions, so far as is known, but in some epidemics it has ranged over every quarter of the globe, and has established itself in places presenting all kinds of soil and every variety of climatic conditions. It, therefore, cannot be attributed to any telluric emanation or miasm. The suggestion has sometimes been made that there may be a relation between it and ague, but it is not found to be especially severe in countries like Holland, which are infected with malaria; indeed, Holland is said to have escaped some epidemics which have traversed Europe.

Again, the progress of influenza from district to district occupies time. Many observers have thought that it commonly observes a definite direction, namely, from the east or the north-east toward the west or south-west. Thus, the epidemic which raged in London in 1833 is supposed by Hirsch to have been related to one which occurred in 1830 in China, and which reached Moscow later on in that year. In 1831 it spread over Russia, Poland, Germany, France, Sweden, Italy; it next appeared in the Isle of Man, and, lastly, in New Jersey, on the other side of the Atlantic. In 1832, it occurred chiefly in Spain and in some of the United States. In 1833, it broke out again in the north of Europe, and after extending over Russia and Germany, and passing to Denmark, it reached London, as I have already mentioned, in April. It was also observed at different parts of this year in France, Switzerland, the Tyrol, Italy, and Egypt. Such a manner of spreading is obviously inconsistent with any conceivable kind of electrical or mag-

netic influence. It is particularly worthy of notice that both in England and in France influenza seems to occur only as the result of extension from other countries. Some writers have even doubted whether it can arise in Europe, and have imagined that it has its home in some remote part of Asia, such as Chinese Tartary, and is always derived from thence.

On the other hand, this disease often suddenly breaks out at the same time in places far distant from one another, and at once attacks a large proportion of their inhabitants. And it has been said to appear on exactly corresponding dates on board ships which had been long at sea, and which had sailed from ports where influenza was not prevailing. Thus, in 1782, Admiral Kempenfeldt's squadron sailed from Spithead on May 2d to cruise between Brest and the Lizard. On the 29th, there having been no communication with any shore, the men who formed the crew of one of the ships were attacked with influenza, and soon afterward so many of the sailors on the other ships that by the second week in June the whole squadron had to return to port. In the meantime another fleet, under Lord Howe, had sailed, all in perfect health, for the Dutch coast. Toward the end of the month of May the disease appeared in several of his vessels, also, although there had been no intercourse with the land. So, again, on April 3d, 1833, the day on which Sir Thomas Watson saw his first two cases of influenza in London, a vessel called the "Stag" was coming up the Channel and arrived at two o'clock off Berry Head on the Devonshire coast, all on board being well. The breeze was blowing from the land, and in half an hour forty men were down with influenza; by six o'clock the number was increased to sixty, and by two o'clock on the following day to 120. The very same evening a regiment at Portsmouth was in a perfectly healthy state, but by the next morning so many of the soldiers were affected by the disease that the garrison duty could not be performed. Parkes, indeed, expresses some doubts as to whether these instances can be entirely relied upon. But, as he says, if they are not altogether without foundation, they effectually disprove all chemical theories as to the cause of influenza. Neither a vapor nor any kind of molecular matter wafted in the air could travel such distances without undergoing dispersion and destruction. Nor is it possible that the morbific agency can be any substance, such as ozone, which, naturally present in the air in small quantities, might conceivably become enormously increased in amount at the same time over a wide area. For the disease, instead of affecting the whole of a town or city, sometimes confines itself chiefly to certain districts, or even to particular streets; and it may leave some of the adjacent villages altogether free.

Thus, by a process of exclusion, we are brought to the conviction that, unless the cause of influenza is something of the nature of which we have no conception, it must be a living thing, which is capable of reproducing and multiplying itself when once it has been introduced into a particular district or country. It would seem, however, that there is one very important objection to such a view in the fact that the distribution of the disease has hitherto appeared to be in no way dependent upon any climatic or meteorological conditions. It may prevail at any season, in every climate, and during all kinds of weather. If it has sometimes broken out after a sudden thaw, or just when there has been a heavy fog, such occurrences are to be regarded as mere accidental coincidences; and it has often been epidemic in countries, such as Egypt, where the air is extremely dry as well as hot.

The difficulty involved in the foregoing statements is all the greater because it cannot be supposed that the organisms which give rise to influenza, if organisms there be, undergo multiplication and development anywhere except in the air itself. It is true that, according to Watson, there have been numerous instances in which the complaint has first broken out in those

particular houses of a town at which travelers had recently arrived from infected places. And there have also been examples of its having spared the inmates of prisons or convents, as though their isolation had served to protect them. But all that such cases can prove is that the morbific agency is capable of adhering to the human body, or to clothes, or luggage, so as to be conveyed from one place to another; its subsequent growth and development is doubtless altogether independent of this kind of assistance. Cullen, indeed, defined influenza as *catarrhus à contagio*, but if he meant that it is contagious in the sense in which we now use that term, it is tolerably certain that such a view is incorrect. The disease seems to occur as frequently among persons who are confined indoors as among those who go about; it often attacks bedridden people; it does not spread from one patient to another, nor to relatives or nurses of the sick. It is decidedly more common in women than in men; children suffer less than adults, and in some epidemics they seem very generally to escape.

Another point which negatives the idea that an attack of influenza is due to an organism which multiplies within the human body is the absence of a stage of incubation. Parkes cites several instances in which persons who have come from a distance to places infected with influenza have fallen ill on the following day or two days afterward. Such cases, indeed, are not in themselves conclusive, for they could be matched by exceptional cases of scarlet fever, in which disease the incubation usually lasts several days. But they go very far toward proving the point in question, when they are taken in conjunction with the fact that at the commencement of an epidemic of influenza an immense number of persons fall ill simultaneously, or nearly so. If the disease took several days to hatch, one may be quite sure that the preliminary morbid process would not thus come to an end in every patient at the same time. Parkes, indeed, alludes to cases "in which the incubation period must have been two or three weeks," but on looking up the reference which he gives to Dr. Robert Williams's work on morbid poisons, I find that the only instances given there are those already alluded to, in which the disease appeared on board ship when there had been no communication with the land.

Another circumstance bearing on this question is the fact that epidemics of influenza have almost always a definite duration of from four to six weeks, and that their subsidence is scarcely less sudden than their commencement. It is mentioned, as a solitary exception, that, in 1831, the disease prevailed in Paris for nine or ten months at a stretch. One cannot but see how easily this may be explained on the hypothesis that the pabulum which is required for the maintenance of the organisms rapidly becomes exhausted if such are supposed to exist.

The way to remove all doubt about the matter would, of course, be to inoculate upon a healthy person the blood of a patient suffering from influenza. In the horse this experiment has already been tried by Hertwig, but has failed. It is, however, a fair question whether the human disease is identical with the influenza to which horses are liable, and which, for example, raged in the United States in 1872, when it is said to have attacked about 16,000 horses in New York alone. During epidemics of influenza among human beings, horses, dogs, cats, and even birds are said to suffer. But when the equine epizoötic prevails, men seem entirely to escape.

Definition.—During the prevalence of an epidemic of influenza the only point to be mentioned in regard to its *diagnosis* is the risk of misinterpreting the early stages of other febrile complaints, such as enteric fever or the exanthemata. But is it possible for the disease to occur sporadically? and if so, how can it be distinguished from a non-specific catarrh? These questions are by no means easy to answer. Sir Thomas Watson, indeed, says that

"in the years immediately succeeding an epidemic it generally shows itself again, but in a milder and less general form." And in further explanation of this meaning he goes on to say that "many of the colds and bronchial disorders of the seasons which follow a period of genuine influenza are attended with much more languor, debility, muscular aching and distress, than belong to an ordinary attack of catarrh." Now, in the case of cholera (a disease which, from a certain point of view, is analogous to influenza) something precisely similar to this seems unquestionably to occur. But it seems to me that the statements just quoted are exceeding difficult of proof in regard to a complaint of which the symptoms are so little characteristic as are those of influenza. Parkes says that sporadic cases are not met with. We ought strongly to protest against the practice, so common among the higher classes in this country, of designating as influenza any catarrhal attack that happens to be painful or distressing. They think that in this way they give a sort of dignity to what would otherwise be a common complaint hardly worthy of attention; in reality they are debasing a word of which it is important to retain the special significance. A fair criterion may generally be found in the fact that these false influenzas occur during the damp and cold seasons of the year, whereas, as we have seen, the genuine disease prevails in all weathers.

A further question still remains as to the nature of certain localized forms of catarrh, affecting a greater or less part of the population of particular towns or districts. Thus, in 1864, influenza is said to have existed in Switzerland, during the spring of 1867 in Paris, and so recently as 1874 in Cape Breton. I agree with Parkes that strict proof that the disease in such "local epidemics" is really influenza ought to be required before they are admitted, but it is difficult to say how the proof is to be obtained.

Treatment.—I need say but little about the treatment of influenza. From a historical point of view it will always be interesting to know that bleeding and the administration of antimony were recognized by universal experience to be injurious in this disease at a time when they were regarded as almost essential to the cure of pneumonia and other inflammations. In one or two of the latter epidemics quinine was found useful even from the commencement of the attack; but the most usual practice is, after having given one purgative dose to prescribe salines during the first day or two, and afterward ammonia with senega or serpentary. Parkes gives many practical details as to the management of the disease, evidently based upon his own experience; and these would doubtless be found very serviceable if one should hereafter be called to deal with a new epidemic. One point on which he insists is that the common custom of feeding the patient with hot beef tea is a very bad one; it invariably, he says, increases the headache and the languor. As there is complete loss of appetite, and as the attack may be expected to come to an end in a few days, it may be sufficient, if he is young and healthy, to let him have such beverages as iced milk and soda water, barley water with lemon juice, or very weak, cold, white wine whey. To such persons stimulants should not be given during the early period of the disease. As it begins to pass off, a good supply of food should be allowed, and at this stage tonics are very necessary.

Whooping-Cough.—Like so many other epidemic diseases, whooping-cough, instead of being clearly traceable in the medical literature of past ages, can be certainly dated back only to a comparatively recent period—the earliest notice of it is said to have been by Schenck in the year 1600. In every country it has its popular name; in France it is called Coqueluche, in Germany Keuchhusten, in England Whooping-cough, or sometimes Chincough. When a Latin designation is desired, the one generally chosen

in this country is that of Pertussis, originally proposed by Sydenham; in Germany it is known by the less distinctive name of *Tussis convulsiva.*

Clinical Course.—The symptoms of whooping-cough when fully developed are so peculiar that it cannot be confounded with any other affection. But, on the other hand, during the early period of the disease no symptoms whatever are present by which its nature can be even suspected, unless, indeed, there have recently occurred in the same family or in the same neighborhood other cases from which it may have been derived. This "prodromal stage," as it is sometimes called, is characterized by the occurrence of an ordinary bronchial catarrh, with a more or less troublesome cough and some pyrexia. Sometimes there is also nasal catarrh, with running at the nose and sneezing. The child (for whooping-cough chiefly affects children) is pale, out of sorts, restless. A point on which Trousseau lays stress is that the cough is sometimes remarkably frequent, recurring fifteen, twenty, or thirty times in the minute; in such cases there is generally high fever. He speaks of having been able to diagnose the real nature of the disease at this period by the incessant repetition of the cough.

The duration of the prodromal stage is very uncertain. Sometimes in young children it lasts only a day or two, or it may perhaps be entirely absent. Sometimes it runs on for two, four, or even six weeks. In some cases it is believed to constitute the whole of the disease, the patient recovering without having any more distinctive symptoms; this possibility was, I believe, first suggested by Cullen. What ordinarily brings this stage to a conclusion is the development of the characteristic "whoop" with the cough. The change in question may occur either suddenly or very gradually, so that the boundary line between the two stages is often not to be fixed with any certainty. The cough may on some one or two occasions be attended with a sound which raises the suspicion of an experienced nurse or visitor, but several days may afterward pass before any confirmation of their suspicion is to be obtained. Or, on the other hand, in a case which is not really one of whooping-cough, doubts may arise; for some healthy children, especially if they happen to cough while crying, make a kind of crowing or whooping noise that is not very different.

But when whooping-cough is well marked, there can be no mistake about it. Pyrexia is now absent. There may be a good appetite, and except for the cough the child may appear to be well. The cough itself comes on in paroxysms, of which there may be only a few in the twenty-four hours, or as many as sixty or eighty, or even more. They are usually more frequently repeated in the night than during the day. Each begins with a series of short explosions succeeding one another rapidly, and, of course, consisting of as many expiratory efforts, but with no noticeable inspiratory movements between them; then, after the air in the lungs has been reduced to a very small amount, there occurs a long-drawn inspiration, attended with a loud whooping or crowing sound that gives the name to the disease. It, in its turn, may be followed by a repetition of the short explosions, and they by the whoop, and such a succession of attacks may continue over a period of several minutes. They end generally by the expectoration of a viscid mucoid fluid, and sometimes by the ejection of the contents of the stomach.

It may well be imagined that the paroxysms of whooping-cough, when severe, cause great distress. The child, when it feels one coming on, runs to its nurse or to its mother for support, or it clings to a chair or to a table so as to diminish as much as possible the shock which affects its whole frame. Patients old enough to take notice speak of experiencing a tickling sensation in the larynx, as if there were something there; or a feeling of compression about the throat, as though the air could not pass freely. Those who are younger betray the approach of an attack by rest-

lessness and by anxiety of face. Steffen (in Ziemssen's "Handbuch") says that the heart's action and the breathing become accelerated.

As the paroxysms continue, the child may become intensely cyanotic. The eyes may protrude, the face and the neck may become swollen, a cold sweat may break out. Steffen says that albumen sometimes appears in the urine. Hemorrhages are by no means infrequent; the expectoration may be stained bright red by blood which probably comes from the fauces or from the larynx, the nose may bleed, one or both of the conjunctivæ may become ecchymosed, the tears even may be mixed with blood, or the tympanic membrane may be ruptured, with escape of blood from the ear on one side or on both. Steffen says that a momentary stoppage of the heart is not uncommon. Sebregondi is cited by this writer as having observed a case, in a girl six years old, in which there was a temporary loss of sight during each attack. Sometimes spasmodic movements of the muscles of the face or of the eyeballs occur. Steffen speaks of a boy, nine years of age, in whom, when severe paroxysms occurred, he noticed an internal squint of the right eye, whereas the left eye looked straight forward, being held fixed by tonic spasm. General convulsions are not of very infrequent occurrence, and they often prove fatal.

After the subsidence of the paroxysm, the child may be out of breath for a time and may be glad to lie down; sometimes it complains of headache, which may continue all day long; it may be dull and apathetic or fretful. But in many cases it almost at once begins to play again and seems as gay and lively as though nothing had happened. When there has been vomiting it often asks for food and eats it eagerly.

Sequelæ.—Among the more remote effects of the violence of the cough is the formation of one or more shallow, whitish ulcers on the under surface of the tongue by the side of the frænum; at least, they are commonly supposed to be caused by the lower teeth, against which the tongue is forced outward during the paroxysm. In this country Dr. Thomas Morton, of Kilburn, first drew attention to these ulcers in a paper read before the Harveian Society in 1876; but on the Continent they had been previously described by Bouchard and others. Dr. Morton detected them in about 40 per cent. of his cases, generally between the third and the fifth week. He once saw an ulcer in an infant who had no teeth, but in that case I suppose that the tongue may possibly have been injured by the edge of the gums. The recognition of sublingual ulcers may sometimes be useful in diagnosis, if a clear history of the character of a child's cough is wanting.

Another result of whooping-cough is the production of pulmonary emphysema, and even the extravasation of air into the interlobular and subpleural areolar tissue, whence in some rare cases it reaches the mediastinum, and may ultimately diffuse itself into the subcutaneous connective tissue of the chest, throat, and limbs. Or a pulmonary alveolus may rupture so that pneumothorax results. Either of these affections may cause the disease to end fatally.

Prognosis.—It very seldom happens that a paroxysm of whooping-cough directly destroys life. A very young child, however, may die as the result of a complete closure of the glottis, or perhaps from syncope or from the rupture of an intracranial blood vessel. When the attacks are very violent and follow one another with extreme frequency, they sometimes give rise to a condition of apathy and stupor which is attributed to the effusion of serum upon the brain and its membranes and which may terminate in death.

Much more often fatal are the pulmonary complications of the disease. Of these the chief are bronchitis and broncho-pneumonia; it is said that they kill half or two-thirds of all the children who are attacked by them. As may easily be imagined, patients who before were weakly and

delicate are much more likely to succumb to whooping-cough than the strong and healthy. It is infinitely more dangerous among the poor than among the rich. There are, however, differences in different epidemics as regards its severity. It is far more serious in its consequences during the cold seasons of the year than in the summer. When it affects adult patients it is very distressing, but it is not dangerous, nor is its duration generally very long.

As a rule, even in children, the tendency of whooping-cough is, after a variable period, to subside and to terminate in recovery. The expectoration which ends the paroxysms becomes looser, more abundant, and more puriform. The violence of the cough lessens and the characteristic whoop disappears. Perhaps the length of time during which it continues to be heard may be altogether six weeks or two or three months. In a case related by Trousseau its duration was altogether only three days; the patient was a child three years old, an inmate of the Necker Hospital, where the disease was epidemic at the time. After the paroxysms have ceased, symptoms of ordinary bronchial catarrh may remain for a time. The child may be a long while in regaining its appetite and strength, especially if the season happens to be winter, so that there is a difficulty in getting it out of doors and into the fresh air. Sometimes, when the whoop has apparently passed off, a fresh attack of catarrh brings it back again for a few days in as marked a form as ever. Even after the lapse of a year it may be noticed that the cough arising out of a simple cold is attended with a somewhat similar sound.

In some cases, even when the paroxysmal stage of whooping-cough has completely passed off, the child nevertheless fails to recover its strength, and ultimately dies of marasmus. Not infrequently pulmonary phthisis develops itself as a kind of sequela. Permanent deafness and otorrhœa are said to be occasional results of the injury sustained by the tympanic membrane during the paroxysms.

Pathology.—There is still considerable uncertainty with regard to the nature of whooping-cough. That its proper place is among the infective diseases is suggested not only by its marked contagiousness, but also by the fact that those persons who have once had it are protected against future attacks. Steffen, indeed, says that its occurrence for the second time in the same individual, although not absolutely impossible, is infinitely more rare than that of scarlet fever, smallpox, or any other exanthem. It is said to be one peculiarity of the contagion of whooping-cough that it is far less apt than most other contagia to be transmitted to a distance in an active state. At the Evelina Hospital for Children the whooping-cough ward is allowed to be separated by a short passage only from other wards on the same floor, whereas the wards for the cases of measles and scarlet fever are isolated in a different building. Steffen remarks that he has never met with an instance of the conveyance of the contagion of whooping-cough by persons not themselves affected with the disease. However, one can hardly doubt that it might be spread by the use of handkerchiefs or towels contaminated by dried secretions from the air passages of patients; and I have read somewhere of an instance in which things sent to be washed on some island from a ship, on board which there were children affected with whooping-cough, conveyed it to the inhabitants of the island, where there were previously no cases of it. In vol. xi of the Clinical Society's "*Transactions*," there is recorded an observation, made by Dr. Bristowe, of a case in which a lady appeared clearly to have conveyed the contagion of the disease from Sydenham to London upon her dress. During a visit to the former place, a boy affected with whooping-cough was climbing on her knee and coughing and sneezing over her; she returned

home the same evening, and early next morning one of her children was found playing over her dress, which had been laid upon an ottoman. This girl took the disease and afterward gave it to two other children. A further point of interest is that the boy himself had only begun to have a constant troublesome cough on the very day on which the lady visited him; in fact, he was staying away from home in the hope that he might escape the disease, which was prevailing among his brothers and sisters. The case is also important as tending to show that the period of incubation in whooping-cough is about a fortnight; for the girl fell ill exactly thirteen days after she was exposed to the contagion, and the two other children sickened at about the same interval after the commencement of the disease in her. Dr. Squire, however, has stated the incubation of whooping-cough at about a week.

Climate does not appear to have much influence upon the prevalence of the disease, except that, perhaps, cold and damp countries are more favorable to it. And Hirsch has shown that it is not more apt to be epidemic at one season of the year than another. There are doubtless great differences among different individuals as regards their susceptibility to the contagion.

Female children are decidedly more liable to be attacked than males. The age at which whooping-cough is most common is between the first year and the eighth. Barthez and Rilliet recorded the case of an infant whose mother had had the disease for three weeks before its birth, and in whom severe paroxysms occurred on the second day. Sir Thomas Watson relates in his lectures how the grandchild of his bed maker at Cambridge whooped on the very day of birth, there having been another child affected with the disease in the same house for three weeks before. On the other hand, whooping-cough is sometimes observed in adults up to forty or fifty or even a still greater age.* Heberden is said to have met with one case in a woman aged seventy and another in a man aged eighty.

Steffen says that it is a mistake to suppose that delicate children are more apt to take the disease than those who are robust. An association is often traceable between epidemics of measles and those of whooping-cough, children falling ill with the latter disease soon after having passed through the former. Such cases are peculiarly apt to be accompanied with severe broncho-pneumonia and have a fatal termination, the exanthem having often already brought the lungs into a morbid condition. Sometimes, however, the relation between the two diseases is reversed, measles breaking out after whooping-cough has existed for some time. It is then sometimes noticed that the paroxysms become much less frequent and much less severe than before, and that they remain so as long as fever persists; but, indeed, the same thing may happen when pyrexia develops itself as a mere result of pulmonary or bronchial inflammation in cases of whooping-cough not otherwise complicated.

As I have already remarked, whooping-cough differs in one very important feature from infective diseases in general, namely, in not being attended with pyrexia at the time when its more characteristic symptoms are manifested. There is, however, a parallel instance in hydrophobia, and I think it is not impossible that Pasteur's discovery of the actual presence of the virus of hydrophobia in the nervous centres, and of its multiplication there, may hereafter be found to supply the key to the pathology of whooping-cough. One can easily imagine that the poison of this disease, having originally entered the air passages from without, and having set up a catarrh there, is during the prodromal stage conveyed to some part of the

* [One of the most eminent physicians in London suffered severely from an attack of whooping cough when sixty-five years of age.—ED.]

central nervous system and there sets up the peculiar spasmodic cough. So far as I can see, none of the theories which have been propounded by writers with a view to explaining this symptom really throw any clear light upon its nature. Some have regarded the disease as a pure neurosis, some have attributed it to pressure upon the vagus nerve by swollen tracheal or bronchial glands, some have maintained that it is a mere catarrh of the respiratory mucous membrane. Beare, on the ground that the paroxysms of whooping-cough resemble those that follow the entrance of a foreign body into the larynx, would have it that the fundamental lesion was an inflammation of the tract immediately above the vocal cords. Observers differ as to whether or not reddening of the laryngeal mucous membrane can be seen with the laryngoscope during life; according to Rossbach ("*Berl. Klin. Woch.*," 1880) no morbid change is discoverable either in the larynx or in the upper part of the trachea; according to Meyer-Hüni ("*Ztsch. f. Klin. Med.*," 1880) there is marked reddening, and even slight swelling, of most parts of the larynx (but not of the cords) as well as of the trachea.

Recent observations render it probable that the contagious principle of whooping-cough is an organism analogous to those which produce so many other infective diseases, and that it has already been seen with the microscope. As far back as 1870 Letzerich described and figured in "*Virchow's Archiv*" a whooping-cough fungus, consisting of thallus filaments as well as of spores. This he found abundantly in the sputa of patients affected with the disease, and he asserted that he had succeeded in cultivating it, and also in producing a like malady in rabbits by inoculating the trachea of these animals with the product of his cultivation experiments. In the "*Jahrbuch der Kinderheilkunde*" for 1876 Tschamer supported Letzerich's views, and also maintained that an identical fungus, commonly found adhering to the surfaces of oranges or apples, is capable of giving rise to whooping-cough when inhaled into the human air passages. More recently, however, Burger, in the "*Berlin. Klin. Wochenschrift*" for 1883, has cast doubt on the accuracy of Letzerich's observations, while at the same time he has asserted that the sputum in whooping-cough always contains large quantities of bacteria, which appear as rods of ellipsoid form, sometimes constricted in the centre, which are generally scattered quite irregularly, but occasionally arranged in chains. They are said to be easily brought into view by staining with fuchsin or methyl violet.

Treatment.—In whooping-cough it is of great importance that the patient should be kept in a spacious room, warm and equable in its temperature. Exposure to cold air has a marked tendency to bring on the paroxysms; and according to Hauke the presence of an excess of carbonic acid in the air has a like effect. In genial weather it is often right to allow a walk out of doors in the middle of the day. When the disease lingers in its course, nothing is so likely to bring it to an end as change of air, especially to the seaside. The food should be nourishing but simple. Talking, crying, and excitement of every kind should as much as possible be avoided.

As regards drugs, belladonna, hydrocyanic acid, chloral, bromide of potassium, bromide of ammonium, hemlock, appear each of them to diminish the frequency and severity of the paroxysms in some cases, and even to shorten the duration of the disease. But not one of these medicines can be depended on to produce a definite effect in all, or even in almost all, cases; they often fail altogether. Quinine is sometimes very serviceable; there are doubts as to whether it acts by diminishing the excitability of the nervous centres, or by checking the growth of the specific organisms on which the disease is believed to depend. The latter view has led to its administration by insufflation into the air passages.

Within the last few years it has become a common practice to treat whooping-cough by inhalations. The earliest attempts of this kind consisted in placing patients in the purifying chambers of gas works, where the air is loaded with tarry products, as well as with sulphuretted hydrogen and ammonia. According to Roger ("*Bull. de l'Acad.*," 1880) the evidence in favor of the value of this mode of treatment is by no means conclusive.

A plan more recently suggested is that of impregnating the air of the patient's chamber with turpentine, or with petroleum, or with carbolic acid. A solution of carbolic acid, for example, is diffused through the room by means of a spray apparatus, or by simply heating a vessel containing it. Children who are old enough may be made to directly inhale a weak carbolic spray for fifteen or twenty minutes two or three times a day. Thorner, in the "*Deutsches Archiv*" for 1878, reports very favorably of this practice; for about a week there was apparently little or no change; but at the end of that time the symptoms of the disease began rapidly to subside. I have heard of successful results attained in this country by similar means.

Inhalations of the vapor from a boiling solution of salicylic acid (of the strength of 2 per cent.) have also been recommended.

END OF VOL. I.